

Caffeine Anaphylaxis: A Progressive Toxic Dementia

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Introduction

Cerebral allergy is an allergy to a substance which targets vulnerable brain tissue and alters brain function. Masked cerebral allergy can cause symptoms of mental illness.^{1,2,3} Symptoms can range from minimal reactions, such as disruptions in attention, lack of focus and comprehension, lack of organizational skills, and abrupt shifting of activities, to symptoms of a severe psychotic state, which include mood changes, irrational behavior, loss of insight, delusions, paranoia, and hallucinations.^{2,3} An allergic reaction can induce anxiety.^{4,5,6} Symptoms of allergic anxiety may be mistaken as hyperactivity, anxiety neurosis, or panic.

Caffeine Allergy Symptoms and ADHD

Caffeine allergy is a cerebral allergy. An allergic reaction to caffeine manifests as anaphylaxis.⁷ According to Dr. Charles Robert Richet, the 1913 recipient of the Nobel Prize in Physiology or Medicine for discovery and work in anaphylaxis, anaphylaxis always paralyzes higher cerebral function.⁸ During a state of caffeine anaphylaxis, the body enters the fight or flight mode, which may be mistaken as hyperactivity, anxiety, or panic disorder. Caffeine anaphylaxis causes cerebral vasculitis, and leads to the breakdown of the blood brain barrier. Poisoning brain cells, ongoing caffeine allergy induces a progressive toxic dementia.³ A buildup of caffeine can exceed tolerance level, and saturate the ability of metabolism.^{9,10} During ongoing caffeine anaphylaxis, rate of caffeine accumulation exceeds rate of elimination.

Toxic dementia induced by a stimulant or other toxin affects function of all brain areas.¹¹ Signs of toxic dementia include memory impairment, deterioration of so-

cial and intellectual behavior, and attention deficits.^{3,11,12,13}

Attention Deficit Hyperactivity Disorder (ADHD), which generally affects children, though of late is affecting adults, is indistinguishable from caffeine allergy. Claudia Miller, M.D. stresses that a chemical sensitivity, which includes caffeine as a chemical capable of inducing sensitivity, can induce attention deficits with hyperactivity.¹⁴ Deteriorating intellect, the first stage of caffeine induced allergic toxicity masquerades as ADHD. Inability to concentrate, lack of comprehension, hyperactivity, delusions, and disorganized thought processes are hallmark signs of caffeine allergy. An allergic reaction to caffeine results in poisoning of the prefrontal cortex. Damage to the underside area of the prefrontal cortex, above the eye sockets, renders a person absent minded and interferes with the ability to monitor personal activities.¹⁵ Complaints of lack of focus, failing memory, and other mental abnormalities, signify dementia.^{11,16} Unable to correlate the patient's symptoms with a textbook disorder, physicians assume ADHD.

Unlike Stephen Cherniske's awareness of instinct warning him that caffeine was affecting his behavior,¹⁷ a child does not know. A youngster can't feel the mild stimulant rush because the underdeveloped body has developed a tolerance. Similarly, a toxic adult loses natural insight which inhibits the ability to recognize caffeine-induced intellect and personality changes.^{3,18,19}

Caffeine causes faster speech and mobility in children.²⁰ With 80% of the world's population consuming caffeine, most persons have remained stimulated since childhood. Stimulated adults can't detect caffeine-induced changes in themselves or in children.

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Misjudging a child's natural state, adults assume children should speak and act at the same rate as stimulated adults. People forget that we are born relaxed. Acceleration of speech and action indicates mania,^{16,21} associated with bipolar affective disorder. Hypomania, a milder form of mania, accompanies the first stage of ongoing caffeine-anaphylaxis-induced fight or flight toxic dementia. Manic symptoms affect children. Psychiatrically hospitalized manic children display symptoms of ADHD.²²

Injury to the prefrontal cortex results in loss of verbal and social inhibition, interferes with focus and memory,^{23,24} and suppresses math skills.²⁴ In studies involving comprehension skills, as in mathematics and logical reasoning, caffeine has either exhibited no change, or has actually depleted performance.²⁵ Caffeine may jeopardize math skills and detailed projects requiring additional thought.^{25,26,27}

Caffeine anaphylaxis interferes with the ability to focus. Sitting still becomes a project. By raising the catecholamine level, caffeine produces additional dopamine, thus increasing activity. Agitation is associated with excess dopamine.²⁸

According to the American Psychiatric Association, which classifies caffeine as a substance, substance intoxication can present with disturbance in attention, perception, judgment, motor activity, and social functioning.²⁹ Caffeine toxicity can induce confusion, restlessness, agitation, irritability, and delirium.^{3,18,19,30,31} In addition, anaphylaxis can induce delirium.⁵

Symptoms of partial withdrawal can overlap traits of poisoning.³² During partial withdrawal, the body metabolizes some caffeine saturating cells. Clarity struggles to return.

Caffeine Withdrawal

Caffeine withdrawal can mimic depression.³³ As the noradrenaline level diminishes, symptoms of depression set in.^{21,34} Caffeine induced withdrawal depression can manifest as lack of focus, confusion,

hyperactivity, irritability, and lethargy. The glucose level, which rises with adrenaline,^{34,35} and remains elevated during the body's struggle to maintain homeostasis, drops during a state of caffeine withdrawal. A decrease in glucose encourages lack of motivation, which may also mimic depression.

As Allbutt and Dixon stressed in 1909 regarding caffeine, another "dose of the poison" provides minor relief, but continues to jeopardize organs.³⁶ A return to caffeine intake increases noradrenaline, heightening the fight or flight response. In turn, adrenaline, dopamine, and glucose increase, thus eliminating symptoms of depression. With continued substance exposure, toxins reaccumulate.³⁷

Caffeine allergy is a deceptive allergy. Ongoing caffeine anaphylaxis reduces allergic inflammation and maintains organ stimulation. Adrenaline, the drug of choice for anaphylaxis, is always present in a caffeine consumer. Catecholamines released during anaphylaxis serve as a protection against vasodilation.³⁸ Endogenous glucocorticoids (including cortisol) inhibit inflammation.³⁹ Theophylline, a caffeine metabolite, maintains open bronchial passages, allowing for easier breathing. Caffeine suppresses phosphodiesterase release.⁴⁰ A decrease in phosphodiesterase leads to an increase in cyclic AMP (cAMP). Excess amounts of cAMP inhibit histamine production.^{41,42} Phosphodiesterase inhibitors inhibit histamine release.⁴³

Caffeine, cAMP and Schizophrenia

Cyclic AMP is increased in patients diagnosed as schizophrenic, and many individuals diagnosed with affective disorders.^{44,45,46} Histamine is reduced in persons diagnosed with schizophrenia, a late stage of ongoing caffeine anaphylaxis. Although the histamine level is low in schizophrenics,^{47,48} schizophrenic patients exhibit a marked tolerance to histamine.⁴⁹ This suggests, in the case of caffeine anaphylaxis, that during the onset stage of schizophre-

nia, when anaphylaxis induced hyperactivity or anaphylaxis induced panic symptoms were mistaken as anxiety, panic, or ADHD, (before continued cerebral poisoning), histamine was increased, but the allergy went undetected. When a young person experiencing an excited state or panic episode arrives in an emergency room, doctors suspect a developing schizophrenia.¹⁶

Researchers theorize that prior to the onset of schizophrenia changes in a person's cognition may be subtle.⁵⁰ Attention and memory deficits accompany schizophrenia.^{50,51} Chlorpromazine (Thorazine), and other phenothiazine drugs exhibit an anti-histamine effect,^{47,52} similar to diphenhydramine (Benadryl). A person allergic to caffeine, taking a phenothiazine medication, will experience relief of the physical manifestations of ongoing caffeine anaphylaxis. In addition, phenothiazines reduce allergic induced abnormal psychological symptoms, including paranoia, delusions, and hallucinations, and generate a return of partial insight, focus, and comprehension.

Introducing a stimulant into a caffeine allergic individual's system further poisons the brain and continues to mask allergic symptoms of caffeine anaphylaxis. Continued stimulant use results in continued deterioration of cerebral function, including insight, intellect, judgment, and affect.

References

- Rippere V: Some varieties of food intolerance in psychiatric patients: an overview. *Nutr Health*, 1984; 3: 125-136.
- Sheinken D, Schachter M, Hutton R: *The Food Connection: How the Things You Eat Affect the Way You Feel-And What You Can Do About It*. New York. Bobbs-Merrill. 1979; 6-9.
- McManamy MC, Schube PG: Caffeine intoxication: report of a case the symptoms of which amounted to a psychosis. *N Eng J Med*, 1936; 215: 616-620.
- Bonner JR: Drug Allergy. In: Goldman L, Bennett CJ, editors. Cecil Textbook of Medicine. Philadelphia. W.B. Saunders. 2000;1463-1466.
- Kaplan AP: Anaphylaxis. In: Goldman L, Bennett CJ, editors. Cecil Textbook of Medicine. Philadelphia. W.B. Saunders. 2000;1450-1452.
- Walsh WE: The Complete Guide to Understanding and Relieving Your Food Allergies. New York. John Wiley & Sons. 2000;7-8.
- Przybilla B, Ring J, Burg G. Anaphylaxis following ingestion of coffee, chronic urticaria and analgesics idiosyncrasy. *Hautarzt*. 1983; 34: 73-76. Review. German.
- The Nobel Foundation. The Nobel Prize in Physiology or Medicine 1913. URL: <http://www.nobel.se/medicine/laureates/1913/press.html>. [Cited July 5, 2002].
- Carrillo JA, Benitz J: Clinically significant pharmacokinetic interactions between dietary caffeine and medications. *Clin Pharmacokinet*, 2000; 39: 127-153.
- Nehlig A: Does caffeine lead to psychological dependence? *Chemtech*, 1999; 29: 30-35.
- Nixon RA, Albert MS: Disorders of Cognition. In: *The Harvard Guide to Psychiatry*. Cambridge, Massachusetts. Belknap Press of Harvard University Press. 1999; 328-336.
- Allen TE, Park LC, Lieberman MC, et al: *A Primer on Mental Disorders: A Guide for Educators, Families, and Students*. Lanham, Maryland. Scarecrow Press. 2001; 22-24.
- Headlee R: *Psychiatry in Nursing*. New York. Rhinehart & Company. 1948; 73-75.
- Miller CS. Toxicant-induced loss of tolerance—an emerging theory or disease? *Environmental Health Perspectives*, 1997; 105: 445-453.
- Eliot L: What's Going on in There? *How the Brain and Mind Develop in the First Five Years of Life*. New York. Bantam. 1999; 294-295.
- Victor M, Ropper A: *Adam's and Victor's Principles of Neurology*. New York. McGraw-Hill. 2001; 1614-1627.
- Cherniske S: Caffeine Blues: *Wake Up to the Hidden Dangers of America's #1 Drug*. New York. Warner. 1998; 1-12.
- Shen WW, D'Souza TC: Cola-induced psychotic organic brain syndrome: a case report. *Rocky Mt Med J*, 1979; 76: 312-313.
- Crothers TD: *Morphinism and Narcomanias from other Drugs*. Philadelphia. W. B. Saunders. 1902; 308-311.
- Nehlig A, Daval J, Debry G: Caffeine and the central nervous system: mechanisms of action, biochemical, metabolic and psychostimulant effects. *Brain Res Rev*, 1992; 17: 139-170.
- Restak RM: *Receptors*. New York. Bantam Books. 1994; 80-84.
- Carlson GA, Kelly KL: Manic symptoms in psychiatrically hospitalized children—what do they mean? *J Affect Disord*, 1998; 51: 123-135.
- Eliot L: What's Going on in There? : How the

- Brain and Mind Develop in the First Five Years of Life. New York. Bantam. 1999; 402-405.
24. Carter R: *Mapping the Mind*. Berkeley, CA. University of California Press. 1998; 180-207.
 25. Braun S: Buzz: *The Science and Lore of Alcohol and Caffeine*. New York. Penguin. 1997; 132-137.
 26. Serafin W: Drugs used in the treatment of asthma. In: Hardman JG, Limbird LE, editors. *Goodman and Gilman's The Pharmacological Basis of Therapeutics*. 9th ed. New York. McGraw Hill. 1996; 659-682.
 27. NTP Chemical Repository: Material Safety Data Sheet: Caffeine. Radian Corporation, 1991. URL: http://157.98.13.224/NTP_Reports/NTP_Chem_H&S/NTP_Chem5/Radian58-08-2.txt [Cited Oct. 12, 2001].
 28. Carter R: *Mapping the Mind*. Berkeley, CA. University of California Press. 1998; 54-79.
 29. American Psychiatric Association: *Diagnostic and Statistical Manual of Mental Disorders Fourth Edition*. Washington. APA. 1994; 176-181.
 30. Fisher Scientific Corporation: *Material Safety Data Sheet: Caffeine*. New Jersey: MDL Information Systems, 1997; 1-7.
 31. Turkington C: *Poisons and Antidotes*, 2nd ed. New York. Facts on File. 1994; 94-96.
 32. Strain EC, Griffiths RR: Caffeine use disorders. In: Tasman A, Kay J, Lieberman JA, editors. *Psychiatry V. I*. Philadelphia. W.B. Saunders Co. 1997; 779-794.
 33. Hirsch K: Central nervous system pharmacology of the dietary methylxanthines. In: Spiller GA, editor. *The Methylxanthines Beverages and Foods: Chemistry, Consumption, and Health Effects*. New York. Alan R. Liss Inc. 1984; 285.
 34. Ackerman S: *Discovering The Brain*. Washington. National Academy Press. 1992; 70-71.
 35. Davidson J, Henry JB: *Todd-Sanford Clinical Diagnosis by Laboratory Methods*. Philadelphia. W. B. Saunders. 1969; 601-602.
 36. Allbutt, Clifford T. *A System of Medicine. VII. Part I*. London. MacMillan, 1909; 986-988.
 37. Van Winkle E: The toxic mind: the biology of mental illness and violence. *Medical Hypothesis*, 2000; 55: 356-368.
 38. Schmidt-Traub S, Bamler KJ. The psychoimmunological association of panic disorder and allergic reaction. *Br J Clin Psychol*, 1997; 36 (Pt 1):51-62.
 39. Claman HN: Glucocorticosteroids I: anti-inflammatory mechanisms. *Hosp Pract*, 1983; 18: 123-126.
 40. Davidson J, Henry JB: *Todd-Sanford Clinical Diagnosis by Laboratory Methods*. Philadelphia. W. B. Saunders. 1969; 698-699.
 41. Dykewicz MS: Anaphylaxis and inflammation. *Clin Allergy Immunol*, 2001; 16: 401-409.
 42. Ernst ME, Graber MA: Methylxanthine use in anaphylaxis: what does the evidence tell us? *Ann Pharmacother*; 1999; 33: 1001-1004.
 43. Raderer I, Haen E, Schudt C, et al. Inhibition of histamine liberation in allergic rhinoconjunctivitis in relation to the season. *Wien Med Wochenschr* 1995; 145: 456-458. Review. German.
 44. Nishino N, Kitamura N, Hashimoto T, et al: Increase in [3H] cAMP binding sites and decrease in Gi alpha and Go alpha immunoreactivities in left temporal cortices from patients with schizophrenia. *Brain Res*, 1993; 615: 41-49.
 45. Erban L, Prokes J, Richtrova E: Plasma level of cyclic AMP and mental disease. *Act Nerv Super(Praha)*, 1980; 22: 269-273.
 46. Biederman J, Rimon R, Ebstein R, et al: Cyclic AMP in the CSF of patients with schizophrenia. *Br J Psychiatry*, 1977; 130: 64-67.
 47. Malek-Ahmadi P, Fried FE: Biochemical correlates of schizophrenia. *Compr Psychiatry*, 1976; 17: 499-509.
 48. Hoffer A, Osmond H: *The Hallucinogens*. New York. Academic Press. 1967; 320-321.
 49. Lea AJ: Adrenochrome as the cause of schizophrenia: investigation of some deductions from this hypothesis. *J Mental Sci*, 1955; 101: 538-547.
 50. Goldberg TE, Hyde TM, Kleinman JE, et al: Course of schizophrenia: neuropsychological evidence for a static encephalopathy. *Schizoph Bull*, 1993; 19: 797-804.
 51. Zuffante P, Leonard CM, Kuldau JM, et al: Working memory deficits in schizophrenia are not necessarily specific or associated with MRI-based estimates of area 46 volumes. *Psychiat Res*, 2001; 108: 187-209.
 52. Sifton DW, editor: *Physicians' Desk Reference. 48th ed*. Montvale, New Jersey. Medical Economics Data Production Company. 1994; 2286-2287.