Research Article **ISSN 2639-944X**

Journal of Medical - Clinical Research & Reviews

Nitrous Oxide use in Labour, NO, it is not safe for Mother or Child

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Received: 12 Apr 2026; **Accepted:** 13 May 2026; **Published:** 24 May 2026

Citation: Gregory John Russell-Jones. Nitrous Oxide use in Labour, NO, it is not safe for Mother or Child. J Med - Clin Res & Rev. 2026; 10(5): 1-7.

ABSTRACT

Metabolic analysis is presented from a mother who had received the "safe" anaesthetic gas, nitrous oxide during labour and who gave birth to a child who was subsequently diagnosed with autism. The analysis showed evidence of functional vitamin B12 deficiency, a known consequence of the use of nitrous oxide. In particular, the functional B12 deficiency was associated more with methyl B12 deficiency, rather than adenosyl B12 deficiency. Attempts to fix the deficiency by high dose administration of a mix of Adenosyl/Methyl B12 were largely unsuccessful, thereby leaving the mother with potentially permanent nitrous oxide intoxication of the major enzyme involved in cycling vitamin B12, methionine synthase. The metabolic profile was very similar to that observed in the daughter with autism. The data strongly supports the notion, that contrary to the assurances of the medical profession that nitrous oxide was perfectly safe for mother and baby, that nitrous oxide use in labour is not safe for either mother or child, and the use of this Schedule 6 toxin should be totally avoided during labour.

Lay Summary

Studies of children with autism who were born to mothers who used nitrous oxide for pain relief during delivery, have shown that the nitrous oxide has inactivated the vitamin B12, and subsequently the presence of inactive vitamin B12 has led to the developmental delay. The current study describes a similar inactivation of vitamin B12 in one of the mothers of the children in the previous studies, and demonstrates that NO, nitrous oxide use during labour is NOT safe for either mother or children.

Keywords

Autism, Vitamin B12, Nitrous oxide, Labour.

Introduction

There are numerous reports on the post-surgical complications of the use of nitrous including peripheral neuropathy [1-4], metabolic encephalopathy [5], myeloneuropathy [6-10], neuropathy [11], pancytopenia [12], Myopathy [13], myelopathy [14-18], severe neuropsychiatric symptoms [19], combined degeneration of the spinal chord [20-37], neurotoxicity, neuronopathy [38], polyneuropathy [39], psychosis [40], dementia [41], ataxia [42], megaloblastic anemia [43], neurological impairment [44], neurologic decompensation [45], neurologic degeneration [46], spastic paraparesis [47].

Despite all of this, nitrous oxide is still used during pregnancy, and the mothers are still assured that the use of nitrous oxide is safe for both mother and baby.

Recently Russell-Jones [48,49] has shown a correlation between the use of nitrous oxide and the subsequent developmental delay and diagnosis of 13 children of 11 mothers receiving nitrous oxide during labour. In this report we describe the development of unresolving chronic fatigue syndrome, in combination with anxiety and depression in one of the mothers of one of the children. Organic Acid Test analysis of the urine from the mother showed the characteristic metabolic profile of nitrous oxide inactivation of methionine synthase, that was seen in the children with autism.

Methods

The methods and conditions of consent were as described previously [48,49,51]. For the analysis, urine was collected after

over-night fasting, frozen and then submitted for Organic Acids Test analysis. The data from the Urinary Organic Acids Testing (Oasis diagnostics) were used to compare the Adenosyl B12 (Adenosylcobalamin) deficiency marker, MMA (methylmalonic acid), with 4 methyl B12 deficiency markers HVA (homovanillic acid), VMA (vanillylmalonic acid), QA (quinolinic acid) and pyroglutamic acid. Data obtained from repeated analysis of samples taken over a period of 6 years, from the nitrous affected mother were compared to the control data previously compared in pooled nitrous oxide affected children with autism [48,49].

Patient Profile

The subject, a mother, was exposed to nitrous oxide for 1-2 hours during labour. The child born under nitrous was later diagnosed with autism. A second child born 2 years later, but without nitrous showed signed of regression at 2.5 years of age, but was treated with repeated dosing of iodine, selenium, molybdenum, riboflavin and high dose Adenosyl/Methylcobalamin, and the regression reversed to a large degree. Four years after the first child and two years after the second, the mother began to show signs of continual fatigue. Within a year the mother became so tired that she was bed-bound on week-ends, with extensive body aches, fatigue and depression. Her condition slowly worsened and she experienced many depressive episodes, with low mood, crying, CFS and aches and pains that slowly worsened to the extent that she contemplated suicide to end it all. Her symptoms largely resolved following similar iodine, selenium, molybdenum and Adenosyl/Methyl B12 treatment, however, her brain B12 markers, HVA and VMA showed little resolution.

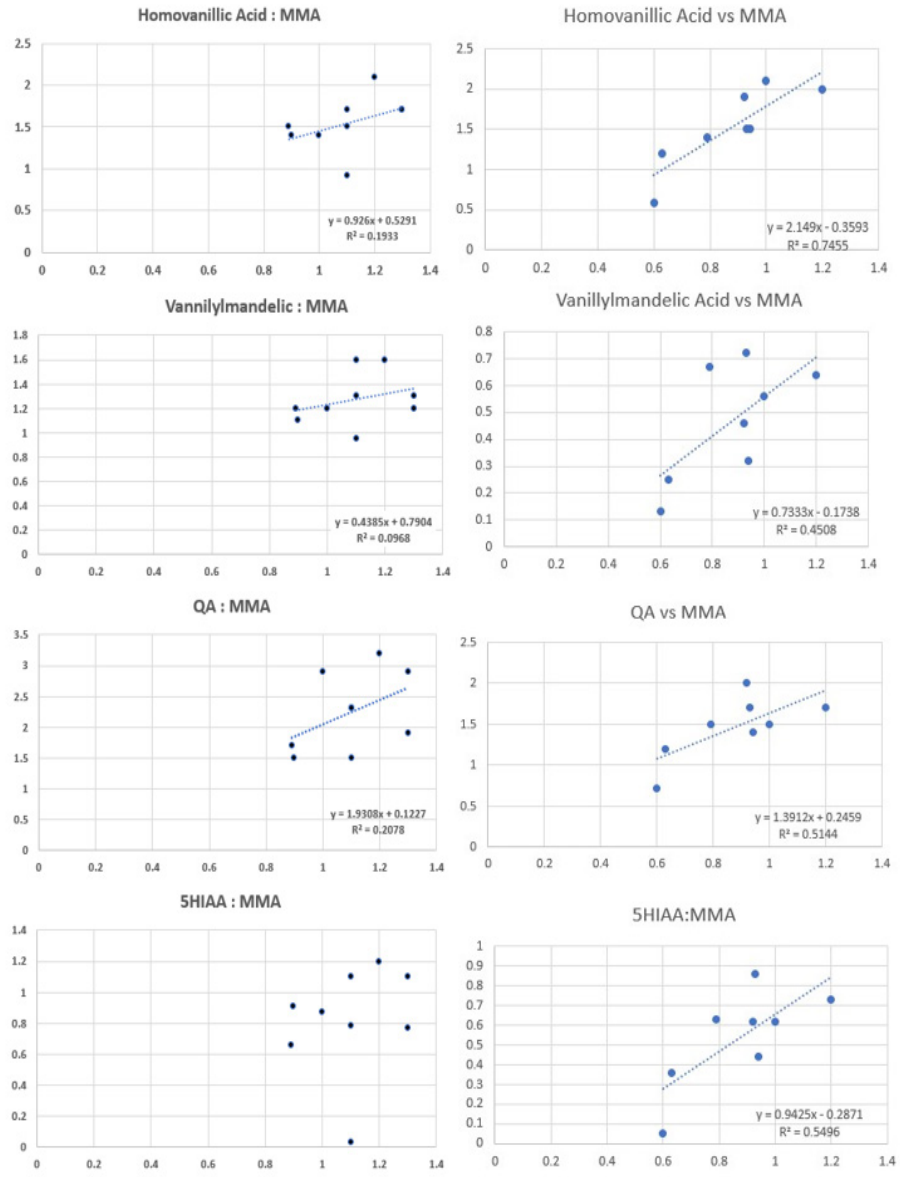

Figure 1: Comparison of AdenosylB12 deficiency marker, MMA, and methyl B12 deficiency markers HVA, VMA, QA, and 5HIAA, between the nitrous oxide affected mother (left panels), and a control, non-nitrous affected male (right panels).

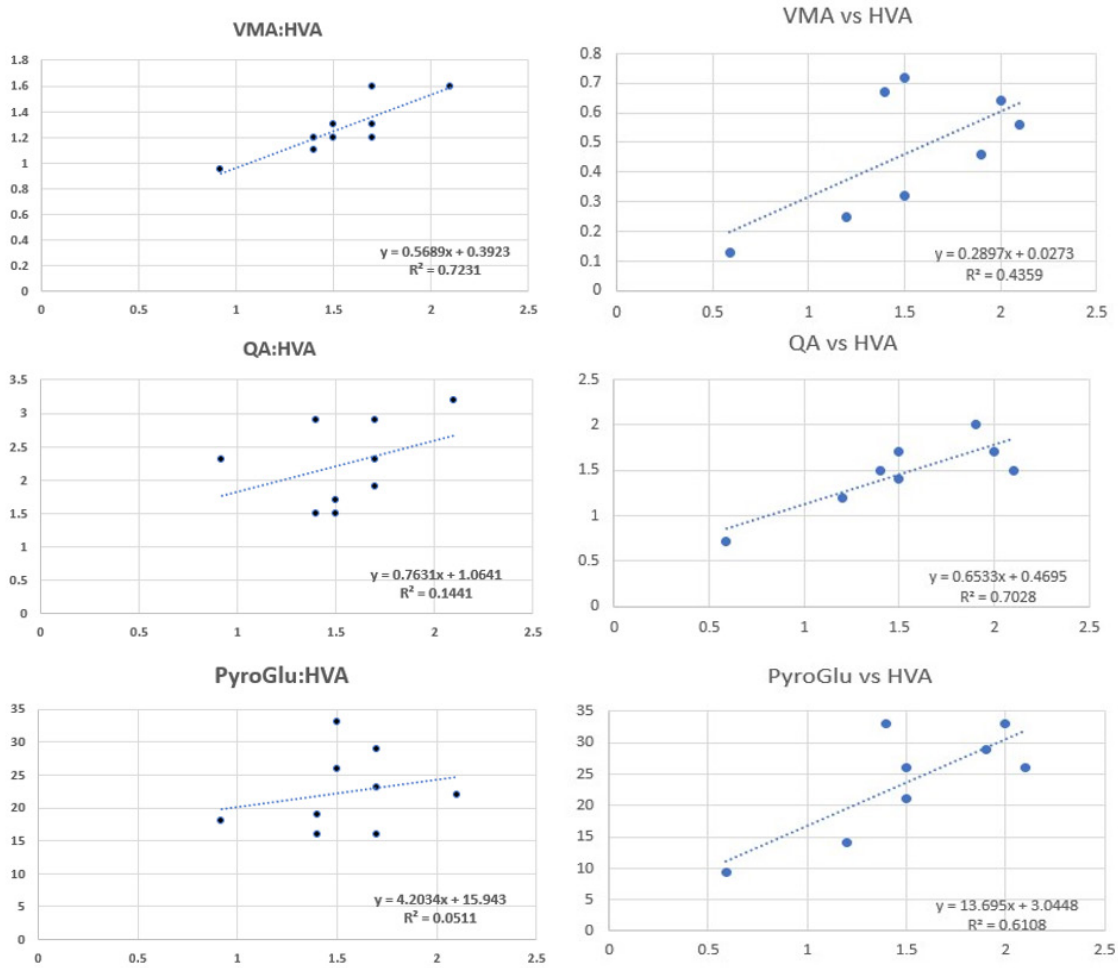

Figure 2: Comparison of the methylB12 deficiency marker, HVA, and other methyl B12 deficiency markers VMA, QA, and pyroglutamic acid, between the nitrous oxide affected mother (left panels), and a control, non-nitrous affected male (right panels).

Results

Initial analysis of urinary organic acids ascertained that the mother had functional B12 deficiency by many markers in OAT, including MMA, HVA, VMA, QA, and pyroglutamic acid.

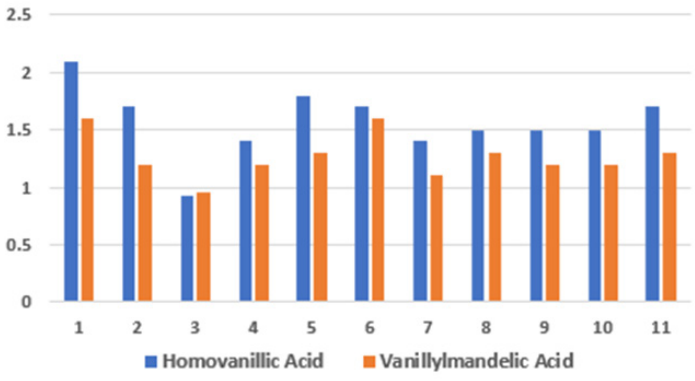

Figure 3: Comparison of the methylB12 deficiency marker, HVA and VMA over time.

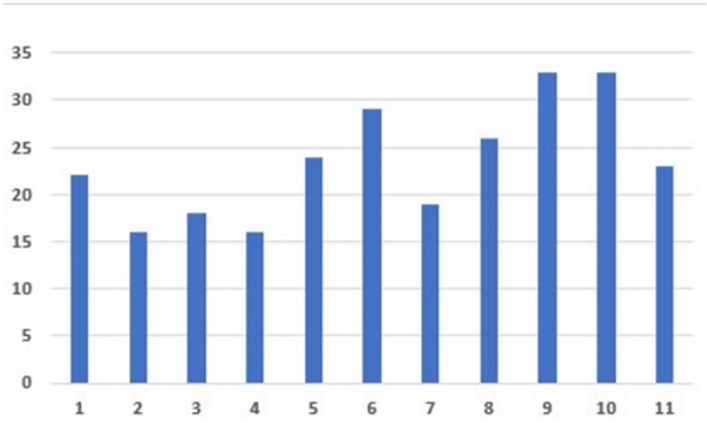

Figure 4: Comparison of the levels of pyroglutamic acid over time.

Serum vitamin B12 of the mother was assessed and found to be high (1000 pmol/L), and had been mis-diagnosed as "normal" by the Pathology Laboratory. A common observation in paradoxical B12 deficiency. Similar paradoxical B12 deficiency [51] was previously observed in the children born under nitrous oxide

anaesthesia of the mother [48,49] and is common in children with ASD [51]. Comparison of the mother's methyl B12 deficiency markers HVA, VMA, QA and pyroglutamic acid with the Adenosyl B12 deficiency marker, MMA, revealed little to no correlation, which was in contrast to that observed with the normal subject (Figure 1). Comparison of HVA with other methylB12 deficiency markers, VMA, QA, and pyroglutamic showed a close association with HVA and VMA, however there was little to no association to QA and pyroglutamic acid (Figure 2). This lack of association is a diagnostic feature of nitrous oxide inactivation of methionine synthase and was similar to that observed previously in children born during nitrous oxide by mothers during labour [48,49]. Repeated high dose Adenosyl/methyl B12 treatment did not resolve the methyl B12 deficiency markers, HVA, VMA (Figure 3), or pyroglutamic acid (Figure 4), even after 7 years of treatment. An inability to improve methylation with repeated administration of methyl B12 is a feature of permanent inactivation of methionine synthase.

Discussion

Methionine synthase is arguably one of the most important enzymes in the body. This enzyme bridges the gap between the folate cycle and the methylation cycle and is essential for providing the methyl group that is subsequently used in over 200 methylation reactions in the body. Of particular note is the production of creatine, which is part of the energy transfer system, and whose importance is second only to the production of ATP. The function of the enzyme is critically dependent upon its co-factor, MethylCo(III)B12. The enzyme has two functions (i) methionine synthase methylates homocysteine to synthesize methionine (methylhomocysteine). During this reaction the methyl group of MethylCo(III)B12 is transferred to homocysteine to yield Co(I)B12 and methionine. (ii) Regeneration of the methyl group of MethylCo(III)B12 by the acceptance of the methyl group from 5-methyltetrahydrofolate (5MTHF), thus regenerating, MethylCo(III)B12 plus tetrahydrofolate (THF).

Reduced activity of methionine synthase can be determined by elevated homocysteine, the presence of which increases as the dose and time of exposure to nitrous oxide increases. Such increases are higher in paediatric patients than adults [54]. The activity of the enzyme, methionine synthase, can be totally and irreversibly destroyed by exposure to nitrous oxide, and it has been shown over 40 years ago that inactivation occurs in a time and dose dependent fashion [55] (Figure 5). During the inactivation Co(I) B12 is oxidized by nitrous oxide to form inactive NOCo(III)B12, which irreversibly inactivates the enzyme. Prolonged exposure at a high enough dose leads to death in every person exposed as has been shown by the recent increase in nitrous oxide associated deaths [56-62] and even the use of nitrous oxide as part of the death penalty [63] and as an aid in suicide [64]. NOCo(III)B12 is a cytotoxin, which has been used to kill cancer cells [65]

With the belief that somehow prolonged exposure of a mother and foetus to a Schedule 6, mind altering neurotoxin, would not affect functional vitamin B12 levels in the brains of either the mother or the foetus and that exposure of the mother to upwards of 20,000 times the recommended upper environmental safety limit would be safe, this mother, and the mother of many other nitrous oxide affected babies was assured that nitrous oxide was perfectly safe for her and her baby. This assurance was given, despite numerous accounts of the deleterious effects of chronic exposure of nursing and theatre staff to nitrous oxide which have included spontaneous abortions, miscarriages in female health personnel, and also congenital abnormalities in children, and which have been reported to cause an increase in uterine, cervical, and kidney cancers, liver diseases, adverse effects on the immune system, bone marrow, and psychomotor impairment in dental staff [66-76]. Potentially exhaled nitrous oxide from the mother, could be inhaled by theatre staff, and since nitrous oxide is psychoactive, producing euphoria, dissociation, and anxiolysis could lead to staff regularly recommending the use of the neurotoxin during labour.

Comparison of the adenosyl B12 deficiency marker, MMA, with several methyl B12 deficiency markers showed a characteristic metabolic foot-print of nitrous oxide poisoning, in that the

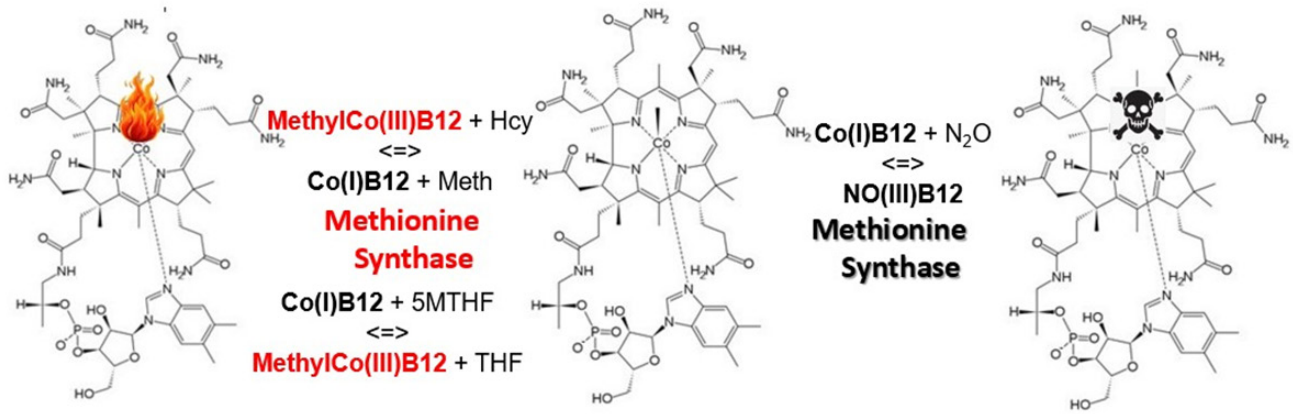

Figure 5: Activity of Methionine synthase in the methylation of homocysteine, and regeneration via reaction with THF. Inactivation via reaction with nitrous oxide.

adenosyl B12 deficiency markers did not correlate with the methyl deficiency markers (Figure 1). A similar pattern was observed in the 13 children with ASD born to 11 mothers who received nitrous oxide during labour [48,49]. The data presented is in direct-line with what the previous studies have shown [50] in which nitrous oxide has induced hypovitaminosis of vitamin B12. The observation that the mother suffers continuing bouts of depression, anxiety and fatigue are in-line with her diagnosis of chronic fatigue syndrome, a condition known to be associated with functional vitamin B12 deficiency, particularly as it pertains to reduced methylation and reduced production of creatine [51,52]. The bouts of depression and anxiety are supportive of the studies in which persons have sufficient brain injury following the use of nitrous oxide in dental procedures, that they were admitted to insane asylums [53,54] and the various reports of psychoses following nitrous oxide use [75,76]. Of particular concern is that the permanent inactivation of methyl B12 by nitrous oxide would compromise the possibility that the mother would be to ever give birth to a neurotypical child. This study, and many previous studies [48,49,54,77-80] belie the claim by the anaesthetic profession and the midwives' associations that the use of nitrous oxide in labour is safe for mother and child**.** Clearly it is not.

Summary

The current study describes a metabolic analysis of a mother who, under the assurance from the anaesthetic and midwives' associations, that nitrous oxide was safe, had used nitrous oxide for pain management during labour. Within two years of the birth of the child, the mother had described many symptoms of vitamin B12 deficiency, including depression, frequent attacks of anxiety and repeated bouts of fatigue. Sequential analysis of her Urinary Organic Acids revealed elevations in many markers of vitamin B12 deficiency, primarily of methyl B12 deficiency, which are commensurative with the known inactivation of methionine synthase by nitrous oxide.

References

1. Neveu J, Perelman S, Suisse G, et al. Severe hyperhomocysteinemia and peripheral neuropathy as side effects of nitrous oxide in two patients with sickle cell disease. Arch Pediatr. 2019; 26: 419-421.
2. Egan W, Steinberg E, Rose J. Vitamin B_{12} deficiency-induced neuropathy secondary to prolonged recreational use of nitrous oxide. Am J Emerg Med. 2018; 36: 1717.e1-1717.e2.
3. Kaski D, Kumar P, Murphy E, et al. Iatrogenic B12-deficient peripheral neuropathy following nitrous oxide administration for functional tonic leg spasm A case report. Clin Neurol Neurosurg. 2017; 160: 108-110.
4. Richardson PG. Peripheral neuropathy following nitrous oxide abuse. Emerg Med Australas. 2010; 22: 88-90.
5. Vive MGD, Anguelova GV, Duim S, et al. Metabolic encephalopathy caused by nitrous oxide ('laughing gas') induced hyperammonaemia. BMJ Case Rep. 2019; 25: e232163.
6. Edigin E, Ajiboye O, Nathani A. Nitrous Oxide-induced B12 Deficiency Presenting with Myeloneuropathy. Cureus. 2019; 11: e5331.
7. Friedlander G, Davies T. The Last Laugh Reversible myeloneuropathy induced by chronic nitrous oxide use. Acute Med. 2018; 17: 232-235.
8. Alt RS, Morrissey RP, Gang MA, et al. Severe myeloneuropathy from acute high-dose nitrous oxide (N2O) abuse. J Emerg Med. 2011; 41: 378-380.
9. Sesso RM, Iunes Y, Melo AC. Myeloneuropathy following nitrous oxide anesthaesia in a patient with macrocytic anaemia. Neuroradiology. 1999; 41: 588-590.
10. Nestor PJ, Stark RJ. Vitamin B12 myeloneuropathy precipitated by nitrous oxide anaesthesia. Med J Aust. 1996; 165: 174.
11. Gullestrup A, Jensen RB, Bøgevig S, et al. Acute neuropathy and liver injury following the abuse of nitrous oxide. Ugeskr Laeger. 2019; 181: V12180890.
12. Norris F, Mallia P. Lesson of the month 2 A case of nitrous oxide-induced pancytopenia. Clin Med. 2019; 19: 129-130.
13. Williamson J, Huda S, Damodaran D. Nitrous oxide myelopathy with functional vitamin B (12) deficiency. BMJ Case Rep. 2019; 12: e227439.
14. Dong X, Ba F, Wang R, et al. Imaging appearance of myelopathy secondary to nitrous oxide abuse a case report and review of the literature. Int J Neurosci. 2019; 129: 225-229.
15. Mancke F, Kaklauskaitė G, Kollmer J, et al. Psychiatric comorbidities in a young man with subacute myelopathy induced by abusive nitrous oxide consumption a case report. Subst Abuse Rehabil. 2016; 7: 155-159.
16. Probasco JC, Felling RJ, Carson JT, et al. Teaching NeuroImages myelopathy due to B_{12} deficiency in long-term colchicine treatment and nitrous oxide misuse. Neurology. 2011; 77: e51.
17. Hathout L, El-Saden S. Nitrous oxide-induced B12 deficiency myelopathy Perspectives on the clinical biochemistry of vitamin B12. J Neurol Sci. 2011; 301: 1-8.
18. Pema PJ, Horak HA, Wyatt RH. Myelopathy caused by nitrous oxide toxicity. AJNR Am J Neuroradiol. 1998; 19: 894-896.
19. Lundin MS, Cherian J, Andrew MN, Tikaria R. One month of nitrous oxide abuse causing acute vitamin B (12) deficiency with severe neuropsychiatric symptoms. BMJ Case Rep. 2019; 12.
20. Lan SY, Kuo CY, Chou CC, et al. PCHAN Study Group. Recreational nitrous oxide abuse related subacute combined degeneration of the spinal cord in adolescents - A case series and literature review. Brain Dev. 2019; 41: 428-435.
21. Patel KK, Mejia Munne JC, Gunness VRN, et al. Subacute combined degeneration of the spinal cord following nitrous oxide anesthesia A systematic review of cases. Clin Neurol Neurosurg. 2018; 173: 163-168.
22. Anderson D, Beecher G, van Dijk R, et al. Subacute Combined

Degeneration from Nitrous Oxide Abuse in a Patient with Pernicious Anemia. Can J Neurol Sci. 2018; 45: 334-335.

23. Antonucci MU. Subacute Combined Degeneration from Recreational Nitrous Oxide Inhalation. J Emerg Med. 2018; 54: e105-e107.
24. Keddie S, Adams A, Kelso ARC, et al. No laughing matter: subacute degeneration of the spinal cord due to nitrous oxide inhalation. J Neurol. 2018; 265: 1089-1095.
25. Yuan JL, Wang SK, Jiang T, et al. Nitrous oxide induced subacute combined degeneration with longitudinally extensive myelopathy with inverted V-sign on spinal MRI a case report and literature review. BMC Neurol. 2017; 17: 222.
26. Buizert A, Sharma R, Koppen H. When the Laughing Stops Subacute Combined Spinal Cord Degeneration Caused by Laughing Gas Use. J Addict Med. 2017; 11: 235-236.
27. Chen HJ, Huang CS. Nitrous Oxide-induced Subacute Combined Degeneration Presenting with Dystonia and Pseudoathetosis A Case Report. Acta Neurol Taiwan. 2016; 25: 50-55.
28. Pugliese RS, Slagle EJ, Oettinger GR, et al. Subacute combined degeneration of the spinal cord in a patient abusing nitrous oxide and self-medicating with cyanocobalamin. Am J Health Syst Pharm. 2015; 72: 952-957.
29. Chaugny C, Simon J, Collin-Masson H, et al. Vitamin B12 deficiency due to nitrous oxide use: unrecognized cause of combined spinal cord degeneration. Rev Med Interne. 2014; 35: 328-332.
30. Cheng HM, Park JH, Hernstadt D. Subacute combined degeneration of the spinal cord following recreational nitrous oxide use. BMJ Case Rep. 2013; 2013: bcr2012008509.
31. Wijesekera NT, Davagnanam I, Miszkiel K. Subacute combined cord degeneration a rare complication of nitrous oxide misuse. A case report. Neuroradiol J. 2009; 22: 194-197.
32. Renard D, Dutray A, Remy A, et al. Subacute combined degeneration of the spinal cord caused by nitrous oxide anaesthesia. Neurol Sci. 2009; 30: 75-76.
33. Wu MS, Hsu YD, Lin JC, et al. Spinal myoclonus in subacute combined degeneration caused by nitrous oxide intoxication. Acta Neurol Taiwan. 2007; 16: 102-105.
34. Ahn SC, Brown AW. Cobalamin deficiency and subacute combined degeneration after nitrous oxide anesthesia a case report. Arch Phys Med Rehabil. 2005; 86: 150-153.
35. Ilniczky S, Jelencsik I, Kenéz J, et al. MR findings in subacute combined degeneration of the spinal cord caused by nitrous oxide anaesthesia two cases. Eur J Neurol. 2002; 9: 101-104.
36. Beltramello A, Puppini G, Cerini R, et al. Subacute combined degeneration of the spinal cord after nitrous oxide anaesthesia role of magnetic resonance imaging. J Neurol Neurosurg Psychiatry. 1998; 64: 563-564.
37. Rösener M, Dichgans J. Severe combined degeneration of the spinal cord after nitrous oxide anaesthesia in a vegetarian. J Neurol Neurosurg Psychiatry. 1996; 60: 354.
38. Morris N, Lynch K, Greenberg SA. Severe motor neuropathy or neuronopathy due to nitrous oxide toxicity after correction of vitamin B12 deficiency. Muscle Nerve. 2015; 51: 614-616.
39. Alarcia R, Ara JR, Serrano M, et al. Severe polyneuropathy after using nitrous oxide as an anesthetic. A preventable disease. Rev Neurol. 1999; 29: 36-38.
40. Sethi NK, Mullin P, Torgovnick J, et al. Nitrous oxide "whippit" abuse presenting with cobalamin responsive psychosis. J Med Toxicol. 2006; 2: 71-74.
41. El Otmani H, El Moutawakil B, Moutaouakil F, et al. Démence postopératoire toxicité du protoxyde d'azote Postoperative dementia toxicity of nitrous oxide. Encephale. 2007; 33: 95-97.
42. Miller MA, Martinez V, McCarthy R, et al. Nitrous oxide "whippit" abuse presenting as clinical B12 deficiency and ataxia. Am J Emerg Med. 2004; 22: 124.
43. Barbosa L, Leal I, Timóteo AT, et al. Acute megaloblastic anemia caused by inhalation of nitrous oxide in a patient with multiple autoimmune pathology. Acta Med Port. 2000; 13: 309-312.
44. McNeely JK, Buczulinski B, Rosner DR. Severe neurological impairment in an infant after nitrous oxide anesthesia. Anesthesiology. 2000; 93: 1549-1550.
45. Felmet K, Robins B, Tilford D, et al. Acute neurologic decompensation in an infant with cobalamin deficiency exposed to nitrous oxide. J Pediatr. 2000; 137: 427-428.
46. Flippo TS, Holder WD Jr. Neurologic degeneration associated with nitrous oxide anesthesia in patients with vitamin B12 deficiency. Arch Surg. 1993; 128: 1391-1395.
47. Lee H, Milgrom P, Huebner CE, et al. Ethics Rounds Death After Pediatric Dental Anesthesia An Avoidable Tragedy. Pediatrics. 2017; 140: e20172370.
48. Russell-Jones GJ. Vitamin B12 Nitrous Oxide Use During Labor and Autism. Med Clin Case Rep. 2026a; 6: 1-7.
49. Russell-Jones GJ. Developmental Delay in Children Due to Inactivation of Methionine Synthase by Nitrous Oxide During Labour. J Med Clin Res Reviews. 2026b; 10: 1-7.
50. Harker Robert DM, Martinez B, Tabaac BJ. B12 Deficiency and Clinical Presentation in the Setting of Nitric Oxide Use. Case Rep Neurol Med. 2021; 2021: 5590948.
51. Russell-Jones, G. Functional Vitamin B12 deficiency in Chronic Fatigue Syndrome. Int J Psychiatry. 2022a; 7: 153-158.
52. Russell-Jones G. Functional Vitamin B12 Deficiency in Autism. J Psychia. 2022b; 25: 495.
53. Edwards ML, Bause GS. From Dental to Mental Institutions Did Hypoxic Anesthetics by "Dental Associations" Add More Brain-Injured Patients to America's Insane Asylums. J Anesth Hist. 2017; 3: 136-137.
54. Edwards ML, Bause GS. From Dental to Mental Institutions An American Dentist and a British Psychiatrist Highlight Insanity Following Nitrous-Oxide Administration. J Anesth Hist. 2018; 4: 133-134.

55. Nagele P, Tallchief D, Blood J, et al. Nitrous oxide anesthesia and plasma homocysteine in adolescents. Anesth Analg. 2011; 113: 843-848.

56. Landon MJ, Creagh-Barry P, McArthur S, et al. Influence of vitamin B12 status on the inactivation of methionine synthase by nitrous oxide. Br J Anaesth. 1992; 69: 81-86.

57. Liu H, Liang M, Yang Y, et al. A fatal case of accidental asphyxia following nitrous oxide inhalation and alcohol consumption. Forensic Sci Med Pathol. 2025; 21: 826-831.

58. Cioffi A, Cecannecchia C, Bosco MA, et al. Lethal nitrous oxide (N_2O) intoxication during surgery the contribution of immunohistochemistry in identifying the cause of death a case report. J Med Case Rep. 2023; 17: 424.

59. Glasper EA. Nitrous Oxide Deaths Among Children and Young People is No Laughing Matter. Compr Child Adolesc Nurs. 2023; 46: 259-261.

60. Schwark T, Schaul M, Schneider S, et al. Two Cases of Fatal Inhalation of Easily Available "Recreational" Substances. Am J Forensic Med Pathol. 2022; 43: 186-190.

61. Gillman MA. Polydrug abuse associated with nitrous oxide causes death. Oxf Med Case Reports. 2016; 2016: 117.

62. Hirvioja J, Joutsa J, Wahlsten P, et al. Recurrent paraparesis and death of a patient with 'whippet' abuse. Oxf Med Case Reports. 2016; 2016: 414-413.

63. Potocka-Banas B, Majdanik S, Dutkiewicz G, et al. Death caused by addictive inhalation of nitrous oxide. Hum Exp Toxicol. 2011; 30: 1875-1877.

64. Bairett L, Wang E. Use of nitrous oxide for execution is a concerning development in ongoing medicalisation of the death penalty. BMJ. 2024; 384: q593.

65. Byard RW, Heath K. How common are suicides using nitrous oxide. Med Sci Law. 2019; 59: 160-163.

66. Bauer JA, Frye G, Bahr A, et al. Anti-tumor effects of nitrosylcobalamin against spontaneous tumors in dogs. Invest New Drugs. 2010; 28: 694-702.

67. Eftimova B, Sholjakova M, Mirakovski D, et al. Health Effects Associated With Exposure to Anesthetic Gas Nitrous Oxide-N(2)O in Clinical Hospital Shtip Personel. Open Access Maced J Med Sci. 2017; 5: 800-804.

68. Fujinaga M, Maze M. Neurobiology of nitrous oxide-induced antinociceptive effects. Mol Neurobiol. 2002; 25: 167-189.

69. Jevtovic-Todorovic V, Wozniak DF, Benshoff ND, et al. A comparative evaluation of the neurotoxic properties of ketamine and nitrous oxide. Brain Res. 2001; 895: 264-267.

70. Jevtovic-Todorovic V, Beals J, Benshoff N, et al. Prolonged exposure to inhalational anesthetic nitrous oxide kills neurons in adult rat brain. Neuroscience. 2003; 122: 609-616.

71. Sanders RD, Weimann J, Maze M. Biologic effects of nitrous oxide a mechanistic and toxicologic review. Anesthesiology. 2008; 109: 707-722.

72. Parker NW, Behringer EC. Nitrous oxide a global toxicological effect to consider. Anesthesiology. 2009; 110: 1195.

73. Myles PS. Nitrous oxide deep in the zone of uncertainty. Anesthesiology. 2013; 119: 1-3.

74. Wiesner G, Hoerauf K, Schroegendorfer K, et al. High-level but not low-level occupational exposure to inhaled anesthetics is associated with genotoxicity in the micronucleus assay. Anesth Analg. 2001; 92: 118-122.

75. Zaffina S, Lembo M, Gilardi F, et al. Nitrous oxide occupational exposure in conscious sedation procedures in dental ambulatories a pilot retrospective observational study in an Italian pediatric hospital. BMC Anesthesiol. 2019; 19: 42.

76. Garakani A, Jaffe RJ, Savla D, et al. Neurologic psychiatric and other medical manifestations of nitrous oxide abuse A systematic review of the case literature. Am J Addict. 2016; 25: 358-369.

77. Bäckström B, Johansson B, Eriksson A. Death from Nitrous Oxide. J Forensic Sci. 2015; 60: 1662-1665.

78. Herff H, Paal P, von Goedecke A, et al. Fatal errors in nitrous oxide delivery. Anaesthesia. 2007; 62: 1202-1206.

79. Herff H, Paal P, Lindner KH, et al. Fatalities due to nitrous oxide. Complications from mistakes in nitrous oxide supply. Anaesthesist. 2008; 57: 1006-1010.

80. Flippo TS, Holder WD Jr. Neurologic degeneration associated with nitrous oxide anesthesia in patients with vitamin B12 deficiency. Arch Surg. 1993; 128: 1391-1395.