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Research Article

ELECTROPHYSIOLOGICAL DISTURBANCES IN PATIENTS WITH PERIPHERAL NEUROPATHIES INDUCED BY NITROUS OXIDE ABUSE: STUDY CARRIED OUT AT THE PONTOISE HOSPITAL IN FRANCE

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Abstract

Background and Objective: Nitrous oxide abuse is common among young people. The objective are to report the electrophysiological characteristics of patients hospitalized for peripheral neuropathy due to nitrous oxide toxicity. **Methods**: We carried out a retrospective study with a descriptive aim from January 1, 2021 to April 30, 2023 at the Pontoise neurophysiological laboratory. We collected data on nerve conductions, needle detection and electroneuromyogram. The study of the conductance of small nerve fibers was carried out using SUDOSCAN. RESULTS: We recruited 15 patients. 12 electroneuromyogram completed. 9 anomalies on the ENMG. 8/9 had axonopathy. 6/8 had sensorimotor axonopathy. 2/8 had purely motor axonopathy. 9/15 SUDOSCAN carried out including 6 abnormal. 1 SUDOSCAN was abnormal with normal electroneuromyogram. **Conclusion and Implications for Translation :** Sensory-motor axonopathy is the dominant profile. The SUDOSCAN abnormality indicates associated damage to large and small fibers. This study shows the seriousness of nitrous oxide responsible for the neurological consequences objectified by the electrophysiological study

Keywords: Axonopathy ; electroneuromyogram ; nitrous oxide.

INTRODUCTION

Nitrous oxide (N2O) is a gas legally used in medicine, but also in the food industry. (1,2). In medicine, for example, it is used as an inhaled anesthetic and analgesic gas (3,4-5). A 2019 survey ranked N2O among the top 10 recreational drugs used worldwide (4). Access is easy thanks to the over-the-counter purchase of food balloons (4). Exposure to nitrous oxide (N2O) is associated with multiple complications, especially neurological ones, which are already known (3). N2O irreversibly inactivates vitamin B12, leading to deficiency (4). Vitamin B12 deficiency can lead to myelopathy and/or encephalopathy, but mainly to peripheral neuropathy (6). Involvement of the peripheral nervous system is evidenced clinically and the electroneuromyography may show altered sensory-motor nerve amplitudes and conductances. Given the extent of the marked increase in the number of cases of recreational nitrous oxide toxicity, and the severity of the neurological signs reported in the literature in recent years, the aim of this study is to establish the electrophysiological profile of patients exposed to nitrous oxide.

METHODS

Study Setting and Participants : The study setting was the neurophysiological exploration laboratory located within the Neurology Department of the Centre Hospitalier René Dubos in Pontoise (France). We conducted a retrospective descriptive study covering the period from January 1, 2021 to April 30, 2023.The study population consisted of patients presenting with peripheral neuropathies associated or not with myelopathies and consulting the emergency department or the neurology consultation service in a context of laughing gas abuse.

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Patients hospitalized for peripheral neurological and spinal cord disorders without laughing gas abuse were not included in our study.

Study Variables: The data used were collected from patients hospitalization records using a standardized questionnaire. The parameters measured by EMNG and SUDOSCAN were :

- Study of motor amplitudes, motor conduction velocities, distal latencies of external popliteal sciatica (EPS), internal popliteal sciatica (IPS), medial, and ulnar.
- Study of the amplitudes and sensory conduction of the musculocutaneous and sural nerves in the lower limbs, and of the median and ulnar nerves in the upper limbs.
- Study of needle detection of the gastrocnemius, anterior hamstrings and vastus lateralis in the lower limbs, and of the 1st interosseous (IO) in the upper limbs.
- Study of small nerve fiber conductance.

Data Sources/Measurements: Qualitative data were expressed as percentages. Quantitative data were expressed as mean +/- standard deviation. Data entry and results analysis were performed using Excel software. The confidentiality of the data collected was guaranteed by the anonymous nature of the data collection, based essentially on file numbers.

RESULTS

We enrolled 15 patients. Electroneuromyograms (ENMG) were performed in 12 patients. 9/12 patients had an abnormal ENMG. 8/9 had an axonopathy profile. 1/9 had a demyelinating profile. 6/8 had sensitivomotor axonal involvement. 2/8 had purely motor axonal involvement (Table I). Of the 15 patients, only 9 underwent SUDOSCAN. 6/9 abnormal SUDOSCANs. 1/6 had an abnormal SUDOSCAN but normal ENMG (Table I).

Table 1. SUDOSCAN features and ENMG conclusion

Patients	1	2	3	4	5
Skin conductance	Not realized	Normal	Slightly reduced hands and feet	Not realized	Significant reduction in hand size
ENMG conclusion	Abnormal sensitivomotor axonopathy with active denervation in the lower and upper limbs	Normal	Abnormal sensitivomotor axonopathy with denervation in the lower limbs	Examination within normal limits	Abnormal motor axonopathy with active motor denervation in the lower limbs
Patients	6	7	8	9	10
Skin conductance	Normal	Slight decrease in feet	Not realized	Not realized	Significant reduction in hands and feet
ENMG conclusion	Abnormal sensitivomotor axonopathy in lower limbs	Abnormal motor axonopathy in the lower limbs	Not realized	Not realized	Abnormal sensitivomotor axonopathy with active denervation of the lower limbs
Patients	11	12	13	14	15
Skin conductance	Not realized	Decrease in hands and feet	Slightly reduced hands	Not realized	Normal
ENMG conclusion	Abnormal motor myelinopathy of the lower limbs	Abnormal sensitivomotor axonopathy with denervation	Normal	Not realized	Abnormal sensitivomotor axonopathy in lower limbs

DISCUSSION

Twelve out of 15 patients underwent ENMG in this study, as 3 were discharged against medical advice. 9/12 patients had an abnormality on ENMG (Table1). 8/9 had axonopathy. Of the 8 patients with axonopathy, 6 had sensitivomotor axonal involvement and 2 had purely motor axonal involvement. Studies show that most neuropathies associated with vitamin B12 deficiency or insufficiency are characterized primarily by axonal pathology (2). Lee et al have reported a pattern of predominantly motor axonal-dependent polyneuropathy (6). In our series, 1/9 had demyelinating involvement with prolongation of distal latencies, lengthening of F waves, slowing of motor conduction velocities (without conduction blocks) with preserved motor amplitudes, as reported in the literature (2). In their study of 20 patients using N2O, Li et al reported a lengthening of distal latencies and a slowing of motor and sensitive conduction velocities, more marked in the lower limbs than in the upper limbs (a more demyelinating profile) (2). In the latter study, the authors pointed out that patients exposed to nitrous oxide abuse could have: either an initial demyelinating profile with secondary axonal damage, or a profile of axonopathy that first evolves into myelin damage (2). Furthermore, in the meta-analysis reported by Oussalah et al (7), electroneuromyograms showed demyelinating polyneuropathies in the majority of cases (23%). Among the 12 patients who underwent ENMG in our study, we found needle detection abnormalities in 9 patients, 3 of whom had resting activities. This allowed us to conclude that the motor neuropathies were of moderate to severe intensity, underlining the severity of neuropathies associated with the abuse of laughing gas. In our study, SUDOSCAN revealed altered sweat conductance in 6/9 patients. This finding is an argument in favor of a probable involvement of the small fibers in association with an involvement of the large fibers. 1 patient/6 had a SUDOSCAN abnormality with normal EMNG. Is this early damage prior to large-fibre damage?

Limitations

This study had a very limited sample size, making it difficult to interpret the results, which were non-significant

Conclusion and implications for translation

Our study highlights the importance of the impact of nitrous oxide abuse on peripheral nerve dysfunction, based mainly on nerve conductance studies, ENMG detection and SUDOSCAN. Sensitivomotor axonal damage is the dominant pattern in our series. It is important to look for small-fiber involvement using SUDOSACAN. Our main aim was to establish an electrophysiological profile of neurological disorders in the context of nitrous oxide intoxication, in order to alert the competent authorities and the scientific community to the real danger of this recreational trend, which is becoming increasingly common in young people.

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Conflicts of Interest: The authors declare no competing interests.

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