Severe hypoxia following local anesthesia in a sedated patient: case report

Malka Ashkenazi, DMD  Beatrice P. Greenberg, DMD  Haim Sarnat, DMD, MS

One of the most serious adverse reactions to benzodiazepines is their potential respiratory depressant effect on the subcortical levels of the central nervous system. When used in the accepted oral therapeutic dosage required for healthy patients, this drug does not usually produce any clinically significant respiratory depression and does not potentiate the depressant effects of opiates. In addition, benzodiazepines produce virtually no changes in cardiovascular function. Ataxia and sedation develop only at doses above those required for anxiolytic effects. The reason for this is that the benzodiazepines depress the limbic system, which affects the emotions and behavior at much lower dosages than drugs that depress the reticular activating system and the cerebral cortex. This fact gives the benzodiazepines a very wide margin of safety between the therapeutic and toxic doses.1

The most frequently reported adverse reactions following oral administration of benzodiazepines for anxiety reduction include transient drowsiness, fatigue, and ataxia. Paradoxical reaction, though rare, may occur and is manifested by excitement, hallucinations, and rage. Discontinuation of drug administration will terminate these reactions.1

The aim of this report is to describe an unexpected adverse reaction to local anesthesia in a sedated patient during dental treatment in order to increase the awareness to the possible side effects associated with this situation.

Case report

A healthy, 12-year-old boy weighing 37 kg (25th percentile) was referred to the children's dental clinic for treatment because of lack of cooperation. His past medical history was unremarkable with no systemic diseases or any medication (ASA I), except for his dental problem. One week earlier, the child was treated in our department using 10 mg diazepam orally 1 h before treatment in conjunction with 40% N₂O/60% O₂ without negative consequences. A week later, 1 h before treatment, when his baseline vital signs were a pulse rate of 70 and 98% oxygen saturation, he received 10 mg diazepam (0.27 mg/kg) P.O. Inhalation of 45% N₂O/55% O₂ was started 5 min before treatment and the patient was connected to a pulse oximeter. The patient received a mandibular block injection (36 mg of lidocaine with 36 μg norepinephrine [0.002%]) following aspiration which was done as a routine procedure, and a rubber dam was placed. When cavity preparation (DO) on the left mandibular first molar commenced, the patient complained of pain. Therefore, a second cartridge (another 36 mg lidocaine and 36 μg norepinephrine) was added following aspiration, about 15 min after the first one. Immediately following the second injection, the patient complained of nausea and dizziness, and vomited. His pulse decreased from 70 to 47-52/min, and his oxygen saturation dropped from 97-98% to 75-87% intermittently. His pulse was very weak, and blood pressure could not be measured. His pupils were equal and responded to light, but he did not lose consciousness. Immediately, N₂O flow was stopped, 100% oxygen was delivered, and the patient was placed in the Trendelenburg position. When we noticed that his oxygen saturation had dropped because of the lowered rate and depth of his breaths, we were prepared to inject flumazenil to reverse the diazepam effect. The child objected to another injection. When he saw the syringe, the rate and depth of his breaths increased and immediately the saturation values returned to normal for a few minutes. During the entire episode, the patient was fully conscious with normal protective reflexes and his temperature was normal, but he was very pale. When oxygen saturation and breathing continued to be labile for 1 h, 25 min, emergency care was summoned. The patient's blood pressure was 120/80 mm Hg and his pulse had returned to 70, baseline values. The EKG done by the emergency care's staff showed sinus arrhythmia with bradycardia. The patient was transferred to a hospital for continued supervision. Three hours following the
beginning of this episode his pulse rose to 80 and his 
saturation and blood pressure spontaneously returned 
to normal ranges, and the patient was released.

For completion of dental care, the patient was re-
ferred to the hospital for general anesthesia, which was 
performed uneventfully a few weeks later. Blood tests 
(Complete blood count, PTand PTT), urine tests, 
and a physical examination performed a few days be-
fore general anesthesia were unremarkable and within 
normal ranges.

Discussion

We present a report of a 12-year-old boy who re-
ceived diazepam (0.27 mg/kg) and 45% \( \text{N}_2\text{O} / 55\% \text{O}_2 \) 
for anxiety relief to facilitate dental treatment. After 
injection of 72 mg of lidocaine for local anesthesia, the 
patient developed bradycardia, his oxygen saturation 
fell to 75% (\( \text{PO}_{2} = 40 \text{mm Hg} \))\(^3\), and his pulse was la-
bile 47-52/min. The reason for this reaction is not 
completely clear. One possibility is that the combina-
tion of the sedative agents—diazepam and \( \text{N}_2\text{O}/\text{O}_2 \) 
in conjunction with two cartridges of local anesthetic—
caused this adverse reaction.\(^4\) The timing of the adverse 
reaction, immediately after the second injection, might 
suggest inadvertent injection into a blood vessel. This 
could have caused transient overdose although the to-
tal amount injected (72 mg) was far from the 
recommended limit of 4.4 mg/kg.\(^3,4\) which would have 
been 163 mg for this patient.\(^3,4\) The length of the re-
action supports this possibility, as the recovery period 
lasted about 3 h, which is the amount of time required 
to metabolize lidocaine (half-life is 1 1/2 h).\(^4\) The most 
likely explanation, therefore, is that overdose of 
lidocaine was caused by IV injection, and together with 
the diazepam administration in therapeutic dose, 
caused this severe hypoxemia.

The role of \( \text{N}_2\text{O}/\text{O}_2 \) in inducing this respiratory 
depression is probably negligible, as \( \text{N}_2\text{O} \) at therapeu-
tic levels does not exert any respiratory depression of 
the central nervous system. In addition, because \( \text{N}_2\text{O} \) 
is not metabolized in the body, the gas is rapidly and 
virtually completely eliminated from the body within 
a brief period of time (3-5 min).\(^1\) In our case, the pa-
tient recovered 3 h after the injection, although \( \text{N}_2\text{O} \) 
was stopped immediately after respiratory depression 
was observed. The only possible role of \( \text{N}_2\text{O} \) in this 
episode could be intensifying the changes induced by 
diazepam in respiratory rate and depth, which is more 
likely to result from its sedative relief of anxiety than 
its having a direct effect on the respiratory system.

Another possibility is that the patient developed a 
vaso-vagal reaction during the second injection, trig-
gered by the edges of the rubber dam which was not 
removed completely or by the fear of the second injec-
tion.\(^5\) This reaction is characterized by nausea or 
vomiting, pallor, perspiration, yawning, epigastric dis-
tress, hyperpnea, weakness, confusion, and pupillary 
dilation. There is initially tachycardia and decreased 
blood pressure. This is followed by bradycardia, pu-
pillary constriction, and syncope. Removing the 
offending stimulus will restore consciousness, with re-
covering within a few minutes.\(^5,6\) We observed bradypnea 
with no tachypnea in the beginning of the episode and 
no perspiration or pupillary dilation, however, the 
recovery period was 2-3 h. These findings seem to at 
least partially negate this possibility.

The emergency care in this case was called after 1 
h, 25 min. We did not call sooner, because it seemed 
that, during the period of observation, when the 
patient's labile pulse and \( \text{O}_2 \) saturation readings were 
noted, that he would stabilize and recover. Therefore, 
our decision to call for emergency care was repeatedly 
delayed. In retrospect, this decision should have been 
made much earlier.

Dr. Ashkenazi is senior physician. Dr. Greenberg is a postgradu-
ate student, and Dr. Sarnat is associate professor and head, 
all in the department of pediatric dentistry, The Maurice and 
Gabriela Goldschleger school of dental medicine, Tel-Aviv Uni-
versity, Israel.

References

1. Malamed SF, Quinn CL: Sedation: A Guide to Patient Man-
2. Anderson JA, Vann WF, Jr: Respiratory monitoring during 
pediatric sedation: pulse oximetry and capnography. Pediatr 
3. Goodson JM, Moore PA: Life-threating reactions after pe-
dodontic sedation: an assessment of narcotic, local anesthetic, 
and antiemetic drug interaction. J Am Dent Assoc 107:239-
45, 1983.
St. Louis pp 49.
Internal Medicine, 7th ed, McGraw-Hill—New York, pp 
73, 1972.