Anesth Prog 38:142–153 1991

Nitrous Oxide

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Since the popularization of nitrous oxide anesthesia in the nineteenth century, this drug has played an important role in both dental and medical practice. Although its specific manner of use has changed considerably in the past 150 years, it remains the most widely used anesthetic agent in North America. The introduction of anesthesia, in particular nitrous oxide, stands as a professional milestone in dentistry comparable to the organization of the American Dental Association, discovery of local anesthesia, and water fluoridation.[1,2] Early practitioners found it ideal for brief general anesthesia which permitted the painless extraction of teeth.[3] After the discovery of procaine, the dental profession gradually separated into those individuals principally interested in surgery (usually with general anesthesia) and those who used procaine and other local anesthetics for restorative dentistry. Nevertheless, by the late 1940s, many dental practitioners recognized that local anesthesia was insufficient in many instances and did nothing to relieve the anxiety and fear of dental treatment. Nitrous oxide subsequently went through a brief period of popularity as a sole analgesic agent. While general anesthesia could be used for these cases, nitrous oxide general anesthesia was only safe when used for brief procedures, and thus did not meet the technical requirements for the increasingly complex and time-consuming restorative and operative treatments. Consistency of pain control without unconsciousness was insufficient to justify continued popularity of this technique.[4]

Langa[5] and others[6,7] then gradually evolved a technique using nitrous oxide as a sedative/anxiolytic when administered in concentrations of less than 50%. Although initially described as "relative analgesia," the use of nitrous oxide is now incorporated into the general area of conscious sedation, which includes many forms of pharmacologic and nonpharmacologic intervention. Recent data indicate that approximately 50% of practicing dentists use nitrous oxide predominantly for conscious sedation.

In contrast, less than 5% still utilize office general anesthesia, although there is a consistent but small need for general anesthesia, as well as conscious sedation.[8]

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Within medical practice, nitrous oxide has continued to be an important adjunct drug. While no longer used in North America as a sole anesthetic agent, it is frequently combined with more potent inhalation or intravenous agents to provide a general anesthetic with a balance of potency, analgesia, rapid recovery, and a limited complication incidence. It has also occasionally been used for postoperative analgesia,[9] relief of ischemic cardiac pain,[10] and as an emergency analgesic in the ambulance system in Great Britain.[11] Similar to its use in dentistry, many diagnostic medical procedures can benefit from the use of nitrous oxide conscious sedation. Diagnostic colonoscopy is more comfortable and better tolerated with nitrous oxide sedation.[12]

CLINICAL CHARACTERISTICS AND DOSAGE

In 1967, Parbrook summarized data from many analgesia studies using nitrous oxide.[13] This data base included both experimental and clinical usage on humans and animals. As a result of this effort, he was able to produce an approximate dose-related response for most clinically observed characteristics of nitrous oxide, especially when it was used as a sole agent. Nitrous oxide effects were classified into four zones of analgesia. Zone I consisted of some analgesia with patients (or subjects) maintaining full verbal contact. In Zone II subjects became psychologically more detached and lightly sedated. Zone III consisted of marked inebriation, but often with some remaining verbal contact and, finally, Zone IV usually produced light general anesthesia (Figure 1).

While Parbrook's characterizations are still highly useful in estimating clinical responses from patients, one must be cognizant that considerable variation in patient response or sensitivity to nitrous oxide exists. Additionally, most of Parbrook's impressions were derived from carefully controlled conditions with tightly fitting face masks or endotracheal intubation, which is unlike the dental delivery system, where the nasal mask may leak if not carefully adjusted (Figure 2).[14] Thus, only an approximate relationship exists between Parbrook's dosage zones and the classic dental patient receiving nitrous oxide sedation. As with most drugs exhibiting CNS depression, when low concentrations of nitrous oxide are com-

ISSN 0003-3006/91/$3.50

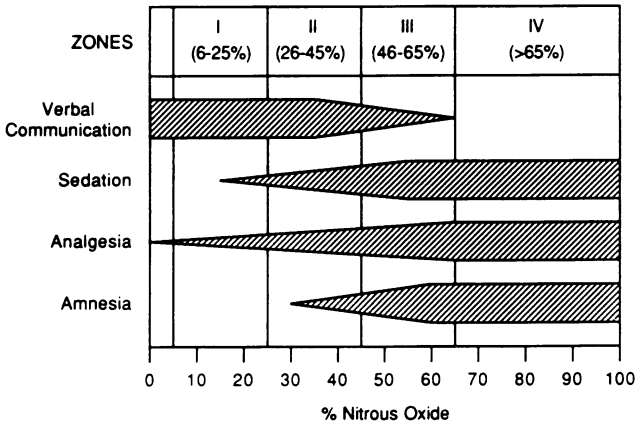


Figure 1. Parbrook's zones and subject responses to increasing concentrations of nitrous oxide. Adapted from Parbrook.[13]

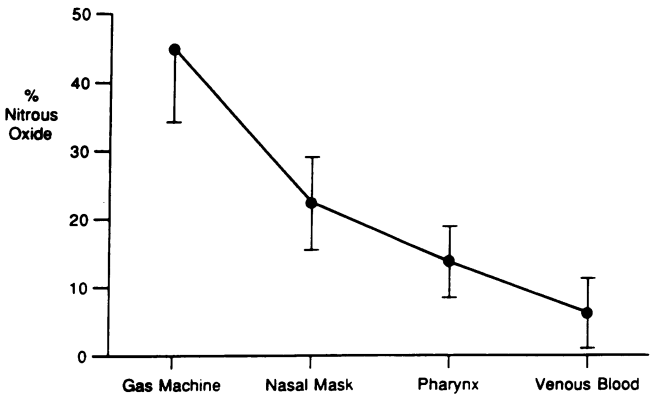


Figure 2. Nitrous oxide concentrations at various sampling sites versus initial concentration setting. Adapted from Sher et al.[14]

bined with other anesthetics, sedatives, or opioids, the effect obtained may result in unconsciousness.

In clinical dental practice the average concentration needed for conscious sedation is 40%, but considerable variation has been demonstrated which represents differences in patient sensitivity to the agent, clinician expectation of sedation intensity, degree of procedural discomfort encountered, and mask fit.[15] Nevertheless, the agent has been highly efficacious when used as a sedative/analgesic, with acceptance rates of approximately 90% combined with a low incidence of side effects.[2,15]

Analgesia

Nitrous oxide demonstrates analgesic and antinociceptive properties. Early studies evaluating its potency in comparison to opioid analgesics reported that 30% nitrous oxide is equivalent to 10–15 mg morphine.[17,18] While useful for various pain control applications (Table 1), nitrous oxide does not produce complete analgesia in most cases, and its value to dentistry has to be understood within a context of controlling acute procedural pain rather than postinjury or postoperative pain seen in the medical model (other than general anesthesia). In this regard, lower (sedative) concentrations tend to be more effective against mild pain[10] rather than the more usual acute severe pain typical of a dental procedure without the benefit of local anesthesia. Figure 3 represents the increase in a measured pain threshold and the ability of human volunteers to tolerate severe tooth pulp pain when given low to moderate sedative doses of nitrous oxide and oxygen. While there is a significant enhancement of both pain tolerance and detection threshold as the dose of nitrous oxide is increased, pain is by no means obliterated by the use of concentrations under 50%. Indeed, even those changes can be significantly altered by providing the patient with positive verbal reinforcement, which enhances pain tolerance. Alternatively, if a patient is unaware of the benefit of nitrous oxide or has no particular expectations from the drug's use, pain threshold and tolerance may not improve or can even decrease.[16,20] Thus, at least the analgesic effects and possibly sedative/euphoric properties at lower doses appear to be highly dependent upon careful patient preparation, including the provision of detailed information about the agent and its benefits.

Table 1. Medical (Nonanesthetic) Use of Nitrous Oxide for Patient Analgesia

N_2O Concentration	*Clinical usage*	*Success*	*Reference*
35%	Acute MI pain	75% of patients with mild pain 25% of patients with severe pain	Thompson 1976[10]
50%	Acute MI pain	80% of patients had complete or partial control of pain	Kerr 1972[19]
50–70%*	Angiography pain control	92% of patients had adequate pain control	Braun 1985[22]
50%	Ambulance transport with various causes of pain	Up to 93% of patients had partial or complete pain control	Stewart 1983[23]

* Most patients were also premedicated with diazepam or meperidine.

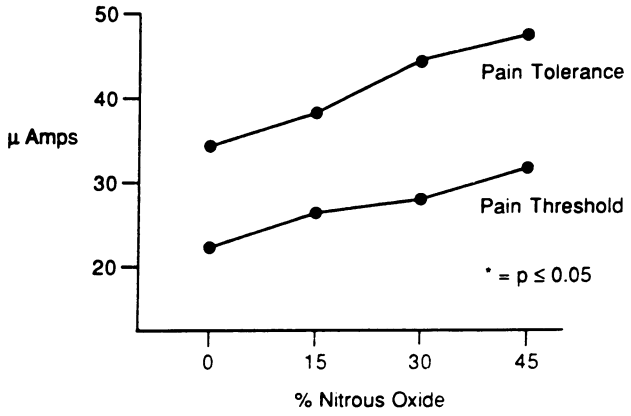


Figure 3. Nitrous oxide concentration versus pain threshold and pain tolerance during tooth pulp stimulation. Adapted from Dworkin et al.[21]

Nitrous oxide analgesia is also affected by altitude due to barometric pressure changes. Analgesic potency achieved at or near sea level is significantly decreased at higher altitudes where the partial pressure of the inspired gas mixture at a given concentration decreases proportionately. Studies done at 4,700 and 10,000 foot elevations revealed a 17% decrease of potency at the former altitude and a 29% decrease in the latter instance.[24] While this is not particularly important to most clinicians in North America, it does significantly affect those located in the higher altitudes (eg, Denver, Colorado).

Nitrous oxide analgesia and general anesthesia are now thought to be separately mediated at different CNS locations.[25,26] Recent work on the analgesic properties of the gas suggests that a major portion of its analgesic effect comes from interaction with opioid receptors. This may be a direct effect on the opioid receptor or perhaps an indirect activity which promotes release of methionine enkephalin[31] (not leucine enkephalin), and possibly an effect on beta-endorphin release.[32] The first indication of this relationship was reported in 1976 when nitrous oxide analgesia in mice was found to be reversed by injecting the opioid antagonist naloxone[27]; other work has supported this finding.[33] Prolonged morphine pretreatment (habituation) in animals essentially obliterates the analgesic effect of nitrous oxide, presumably due to the development of cross tolerance.[27] Similarly, abstinence syndrome will develop in mice when nitrous oxide is withdrawn, but can be decreased with morphine or again precipitated by naloxone.[28] Recent data have also documented acute tolerance development in human subjects.[29] Volunteers administered 60% to 80% nitrous oxide over 3 hours developed profound, but not complete, analgesia to cold water immersion (0° C) within 2 min of gas induction. This analgesia increased to a peak effect at approximately 30 min, and then slowly decreased to control values (little or no analgesia) at 150 min. The precise mechanism of this response is uncertain, but might be an acute decrease in opiate receptor density.[30] The clinical implication is that analgesic effects of nitrous oxide may not be effective after an hour of continuous usage. However, clinically most general anesthetics today utilize more than a single agent, and surgical or dental stimuli vary in intensity, unlike experimental conditions. Finally, most outpatient dental procedures are completed within an hour and well within the time frame of demonstrated analgesic effectiveness of nitrous oxide, even if used as a sole agent for supplemental analgesia and sedation.

The recent characterization of opioid receptors into several subclasses (mu, kappa, sigma, delta), with distinct clinical responses attributed to each receptor, has stimulated research to identify the receptor(s) with which the agent specifically interacts. Initially, results suggested binding only at the mu and delta receptors and not at the kappa or sigma sites.[25] However, more recent data also indicate possible kappa receptor activity,[34,35] although additional study needs to be completed before a more precise understanding will emerge.

Cardiovascular Effects

In certain respects, the cardiovascular effects of nitrous oxide are unsettled. Its use as a sedative/analgesic in relatively normal patient populations is not usually accompanied by meaningful changes in either heart rate or blood pressure, and those that do occur tend to be masked (or induced) by the operating conditions encountered during clinical practice. This subtlety of effect and the drug's anesthetic weakness have led some practitioners to conclude that nitrous oxide's cardiovascular effects are compatible with or related to those produced by increased concentrations of oxygen.[36,37] Additionally, one carefully performed study on human volunteers failed to show any significant cardiovascular effect over a 2-hour time period when the subjects breathed 20% or 40% concentrations. Also, in that study, 60% actually produced transient increases of cardiac output, mean arterial pressure, heart rate, and stroke volume comparable with changes seen with classic anesthetic excitation.[38] Similarly, data obtained from patients receiving 50% nitrous oxide for analgesia/sedation during treatment of serious ischemic cardiac disease reportedly have not shown any deleterious changes in heart rate or blood pressure.[37,39] Finally, the gas does not appear to induce electrocardiographic evidence of myocardial ischemia when used with an opioid during general anesthesia for coronary artery surgery[40] or transesophageal echocardiography.[42]

However, a larger body of experimental and clinical data suggest that nitrous oxide does indeed possess sig-

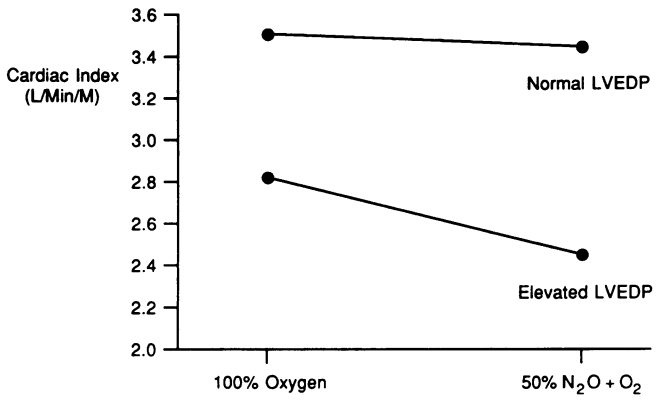


Figure 4. Myocardial depression in patients with elevated left ventricular end diastolic pressure (LVEDP > 15 mm Hg) versus those with normal pressure (LVEDP < 15 mm Hg). Adapted from Balasaraswathi et al.[49]

nificant cardiac depressant effects. Eisele's work on healthy volunteers subjected to 40% nitrous oxide (60% oxygen), when compared with a 40% nitrogen control clearly, demonstrated small but statistically significant decreases in heart rate, cardiac output, and ballisto-cardiographic effects compatible with a negative inotropic effect.[41] Blood pressure, however, remained unchanged, probably due to an increase of circulating norepinephrine and peripheral resistance.[38,41] In most circumstances, and particularly with healthy adults and children, this depression is clinically unimportant. Of concern is the agent's effect on the compromised patient, specifically the individual with cardiac decompensation. Recent animal studies have identified that nitrous oxide has a negative inotropic effect on cardiac muscle whether used by itself[43] or in addition to other anesthetic/analgesic agents, such as fentanyl,[44] halothane,[45,46] or isoflurane.[47] Clinical studies on patients undergoing cardiac catheterization,[48] coronary artery bypass surgery,[49,50] and infants with congenital cardiac disease[51] all demonstrate clear cut evidence of myocardial depression. Of particular importance in adults is the considerable variation seen among patients depending upon the severity of left ventricular depression.[50] Patients with mild or no myocardial depression are capable of substantial compensation from the effects of nitrous oxide, whereas those with severe myocardial decompensation are not (Figure 4). Although the cardiovascular effects of nitrous oxide reported in the literature have not always been predictable or in agreement, much of the difference seems to lie with the agent's weak effects at subanesthetic doses, differences in patient or animal populations used in each study, as well as the confounding effects of neurovascular compensation, disease, or interaction with other drugs. The most appropriate use of the agent in an elective ambulatory setting typical of dental practice is one of caution or restriction of use with patients exhibiting significant cardiac decompensation (congestive heart failure). However, selected patients with ischemic heart disease (angina) without decompensation may benefit from nitrous oxide's analgesic and sedative properties.

Respiratory Effects

Nitrous oxide used by itself in subanesthetic doses does not cause respiratory depression per se. But the drug does have direct effects on respiratory function. Respiratory rate increases substantially with increasing inspired concentrations of the gas.[52] In this category, it is quite similar to more potent anesthetics, such as halothane, enflurane, or isoflurane. Also similar to those agents, nitrous oxide decreases tidal volume in direct proportion to the inspired concentration.[53] Unlike more potent agents, this decrease is easily compensated for by the increased respiratory rate. The net result is a modest increase in ventilation provided that no other agent such as an opioid, barbiturate, benzodiazepine, or potent inhalational agent is added. When other agents are employed, respiratory depression will vary with the type of agent and dosage used. Nitrous oxide will profoundly depress the ventilatory stimulation of both hypoxia[54,55] and hypercarbia.[56] In this regard, even at low inspired concentrations, nitrous oxide is capable of potentiating the apneic effects of other sedatives/anesthetics and is theoretically capable of producing complete apnea by itself when administered in a hyperbaric chamber at pressures greater than 1.5 atmospheres (Figure 5).[53] While sedative doses of nitrous oxide when used alone do not normally cause apnea,

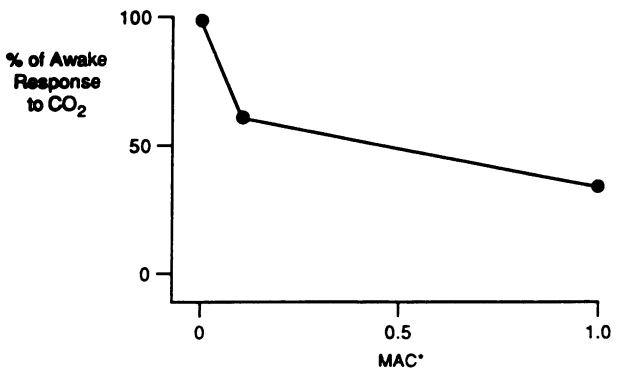


Figure 5. Depression of ventricular response of increased $PaCO_2$ versus dosage of inspired nitrous oxide. Adapted from Eger.[53]
*MAC = Minimum alveolar concentration of anesthetic required to prevent movement in 50% of patients subjected to noxious stimuli. 1 MAC N_2O = 104% or 1.04 atmospheres.[52]

except in selected patients with chronic obstructive pulmonary disease, combining the agent with either barbiturates or opioids has been shown to cause apnea.[57,58] Presumably a similar response would occur with intravenous benzodiazepines, such as diazepam or midazolam. Sedative concentrations of various oral or parenteral agents when combined with sedative amounts of nitrous oxide will not only deepen the CNS depression, but potentiate respiratory depressant effects not usually anticipated with sedation, thus resulting in a greater potential for major anesthetic complications.

CHRONIC EFFECTS OF NITROUS OXIDE

Effects on Vitamin B_{12} and Methionine Synthetase

Occasionally nitrous oxide has been used for prolonged time periods as a sedative and analgesic. In one such circumstance, Lassen[59] reported significant bone marrow depression which was absent in similarly treated patients not given nitrous oxide. More recent information suggests that early (initial) changes in hematopoiesis only requires 6 hours of continuous exposure to sedative concentrations of the gas.[60] Measurable granulocytopenia or thrombocytopenia requires 2 or more weeks of continuous exposure,[61,62] and related neurologic abnormalities are not observed until much later. Long term intermittent exposure to clinical concentrations of nitrous oxide produces neurologic symptoms reminiscent of multiple sclerosis, but no hematologic abnormalities. Layzer described 15 such cases; 13 of which were dentists abusing the drug recreationally for months.[63] Symptoms included ataxia, weakness, impotence, and loss of sphincter control similar to the polyneuropathy seen with chronic vitamin B_{12} deficiency.

Animal studies show that nitrous oxide inactivates vitamin B_{12} and thus adversely affects the synthesis of the critical amino acid methionine from its precursor homocysteine.[61,62,64] Interference with the methionine synthetase reaction relates to its critical role in deoxyribonucleic acid (DNA) synthesis, and thus production of sperm cells, hematopoiesis, and other cells with rapid turnover rates. Methionine synthetase activity can be measured, and when such testing is performed in the presence of nitrous oxide, initial inhibition can be seen in as little as 30 min.[65] Other workers have demonstrated up to a 63% reduction in methionine synthetase activity from 1 hour's exposure to 50% nitrous oxide.[66] Full recovery required approximately 3 days and was dose dependent. A similar response in man is much slower than the animal model. Normal sedative/analgesic usage does not produce any meaningful cell depression until after more than 6 hours continuous exposure.

Testing has been done to elucidate the effect of chronic trace exposure to nitrous oxide on methionine synthetase. Dentists chronically exposed to as much as 4,600 ppm (50 ppm is the current recommended standard) of nitrous oxide in the working environment failed to show any reproducible effect on methionine synthetase below 1,800 ppm.[67] It was noted that a combination of higher doses and prolonged exposure was needed for any abnormal findings to occur. Similarly, other practitioners could not detect any changes in vitamin B_{12} function when ambient concentrations of nitrous oxide were below 1,000 ppm.[68] As a result, 400 ppm has been suggested as a more reasonable standard than the current 50 ppm.[67]

Trace Contamination

The typical method of using nitrous oxide in a dental practice through the 1960s was by an open system. The patient breathed in nitrous oxide and then exhaled the gas through a one-way valve into the room. At the time, nitrous oxide was felt to be a relatively inert gas, and since there was no obvious adverse effect on the patient who received the gas, it was assumed that there was little possibility of adverse effects to the health care providers from inhaling the low concentrations in the ambient air.

However, in 1967, Vaisman reported the results of a 20-year survey on the health of Soviet anesthesiologists who were chronically exposed to anesthetic gases. An increased incidence of fatigue, irritability, nausea, and headache was noted as well as an unusually high incidence of spontaneous abortion.[69] A similarly high incidence of spontaneous abortion in exposed pregnant women was reported soon after by Askrog and Harvald[70] and Lencz and Nemes (as reported by Corbett[71]). With the chance observation earlier that nitrous oxide inhibited cell growth in human bone marrow,[59] it was postulated that the harmful effect of chronic exposure to nitrous oxide may be its effect on tissues with a high rate of cell proliferation. Subsequently, a number of studies were reported on the use of embryonic tissues, and increased fetal mortality was demonstrated in both the chicken and rat due to nitrous oxide exposure.[72–75] However, not all investigators agreed.[76–78] Growth retardation in the chick and rat embryo has been reported,[74,79] and newborn rats tend to decrease in weight after prenatal exposure.[80] Additionally, a variety of experimental animals have been reported to develop rib defects, hydronephrosis, cardiomegaly, and hydrocephalus.[79] Unfortunately, other studies were unable to document such malformations.[74,75,77,78,81] Additional data suggested that chronic exposure of male rats to 20% nitrous oxide produced decreased sperm counts and abnormal cell types capable of reverting back to normal within 3 days after termina-

tion of gas exposure.[82,83] These animals, when mated, uniformly produced abnormal litters.[83] Inconsistencies between studies may be explained by differences in animal species or experimental design, variations in exposure, and differing gas concentrations. However, they do leave considerable doubt as to the true toxicity of the agent.

In 1968, Bruce and his coworkers published a 20-year retrospective review of anesthesiologist mortality.[84] That study suggested that anesthesiologists were more likely to die of lymphoid malignancy and suicide than the general population. A prospective study by the same authors failed to confirm their previous findings.[85] Similarly, another paper reported that malignancy rates were higher in nurse anesthetists,[86] but this review was also criticized for a major methodologic deficiency. Because of concern over these inconsistent findings, the American Society of Anesthesiologists then sponsored a large-scale study of operating room personnel. The results of that epidemiologic survey confirmed that exposed women demonstrated an increased incidence of spontaneous abortion and congenital malformations, as well as hepatic disease.[87] Exposed males also reported a higher incidence of hepatic disease.

While all the previous human studies were strongly suggestive of environmentally induced abnormalities, none had the ability to specify which agent(s) was responsible or, for that matter, to exclude critical differences between experimental and control populations. Subsequently, the American Dental Association recognized that within dentistry was a large group of practitioners using a single agent (nitrous oxide) who otherwise were similar to those not using any inhalation agent at all. As a result, they commissioned a nationwide survey of dentists who used nitrous oxide and compared the data with those who did not. The information obtained from that effort confirmed that exposed women (dentists and assistants) did indeed have an increased rate of spontaneous abortions and spouses of exposed male dentists were likewise affected, presumably due to sperm abnormalities.[88,89] However, other findings such as increased rates of hepatic, renal, and neurologic diseases were more problematic with alternative explanations still being possible. Nevertheless, the American Dental Association recommended the use of gas scavenging to reduce the nitrous oxide exposure of office personnel to a level not thought capable of pathologic effects on humans.

Scavenging

Shortly after its Ad Hoc Committee's report on trace contamination, the American Dental Association published recommendations for the dental profession suggesting ways to minimize environmental exposure to nitrous oxide (Table 2). These recommendations have also been adopted by other countries.

Table 2. American Dental Association Recommendations to Reduce Environmental Exposure to Nitrous Oxide[95]

—Achieve levels of nitrous oxide below 50 ppm.
—Minimize patient conversation during use of nitrous oxide.
—Utilize monthly checks of nitrous oxide machines for leakage.
—Vent exhaust gases to the outside and not through the ventilation system.
—Use an airsweep.
—Monitor levels of nitrous oxide in the operatory on a regular basis.
—Utilize a scavenging system.

Without the use of gas scavenging devices, published data indicate that nitrous oxide contamination in selected dental operatories could be as high as 7,000 ppm.[90] Other studies revealed that scavenging devices specifically designed for dental use could under ideal conditions reduce environmental pollution to about 50 ppm.[91–94] As a result, this low level was adopted even though no specific scientific data suggested that this level was necessary or for that matter consistently achievable in a typical dental practice.

Early recordings of nitrous oxide levels in operatories suggested that when patients spoke, the amount of environmental contamination increased significantly.[93] Other recommendations are somewhat more difficult to adhere to, and indeed the use of an "airsweep" (fan) to circulate air in the operating area is universally ignored. Monitoring nitrous oxide trace levels on a regular basis has been problematic. "Grab" or "snatch" samples may not provide valid data,[95] and time-weighted sampling systems are usually recommended. The most convenient of these are dosimeter badges worn by office personnel containing a chemical absorbent for nitrous oxide.[96]

NITROUS OXIDE EQUIPMENT

Scavenging Systems

One of the earliest devices introduced to dentists was the Brown mask. In fact it is in part responsible for the 50 ppm standard in use today. It has frequently been described as a mask within a mask, or a double mask (Figures 6 and 7). In this system, nitrous oxide is delivered to the patient by a pair of tubes attached to an inner mask. The expired gases then pass through a one-way valve to the outer mask where another pair of tubes remove the gas by way of a suction system. The two masks are separated at the periphery so that suction applied to the outer mask draws air in from around the nosepiece. As a result, it is thought that a tight fit is not required for adequate

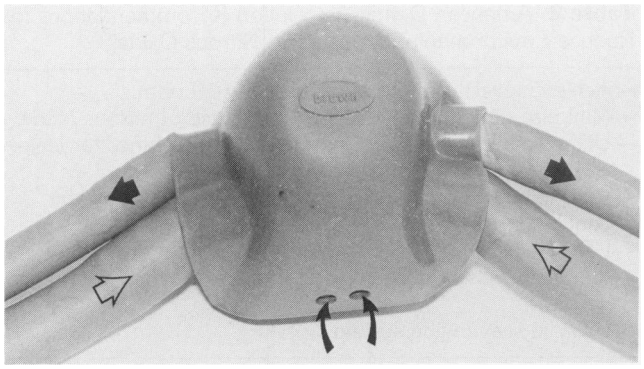


Figure 6. An exterior view of a Brown mask. Open arrows indicate gas intake and solid arrows waste gas removal. Smaller solid arrows denote suction intake holes.

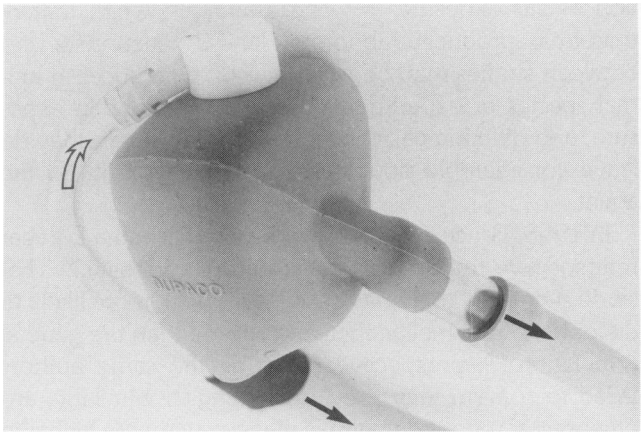


Figure 8. A Dupaco (Allen) scavenging mask with fresh gas inlet (open arrow) and waste gas removal (solid arrows).

function. This unit has been well accepted by both patient and dentist, the only drawback perhaps being the bulk of the double tube system.

A different approach is used by the Allen system (Dupaco). In this system, gases are delivered to the nosepiece through a single, small caliber tubing and removed by two larger tubes on either side of the nosepiece (Figure 8). Alternatively, the two tubes can be replaced by a single tube passing over the patient's forehead. Although the nosepiece is a simple design, the control of gases depends upon a sophisticated manifold suction apparatus for gas scavenging (Figure 9). The reservoir bag is on the exhalation side of the circuit. During exhalation fresh gas is delivered to the nosepiece via the small fresh gas tube. The large exhalation tubes then temporarily serve as fresh gas reservoirs after completion of exhalation and prior to inspiration.

The Matrx (formerly Fraser Harlake) system represents another fairly simple approach with gas being delivered to the nosepiece through a fresh gas tube (Figure 10). Exhaled gas then passes through a one-way valve into an umbrella shaped plastic structure which sits on top of the nosepiece. The waste gas is then removed by suction through a separate single exhalation tube. The umbrella-like structure is also open at the periphery so that it acts as a sweeper system, presumably collecting any gas which may escape from the periphery of the nosepiece or from the mouth of the patient. Again, some attention has been paid to controlling the amount of suction applied to the nosepiece for scavenging. Although a manometer is not supplied with the system, the suction is manually adjusted to an optimal level. Although the system is simple to use, a major complaint has been the noise made by the vacuum system resonating in the scavenging cone.

The Porter system is effective but more complex. Two large-diameter corrugated tubings attach to the nose-

Figure 7. An interior view of a Brown mask revealing the double mask construction and gas flow.

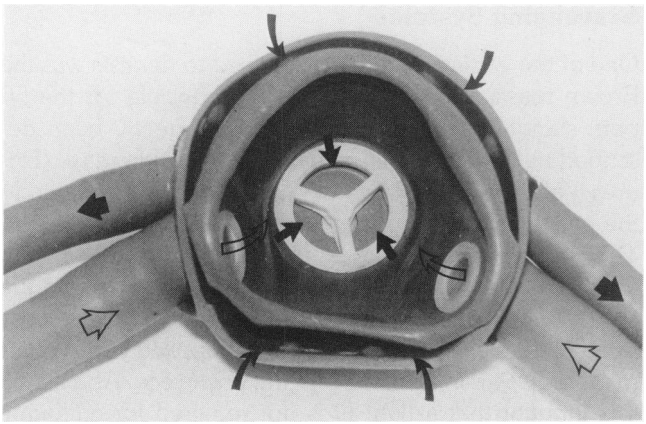
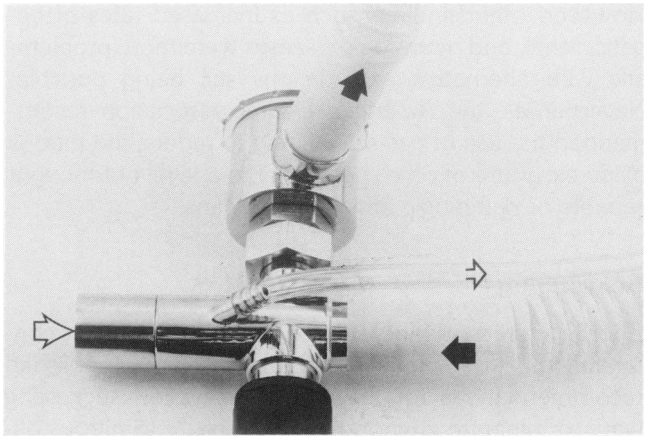


Figure 9. Dupaco manifold attachment demonstrating fresh gas outflow (open arrow) and waste gas removal (solid arrow).



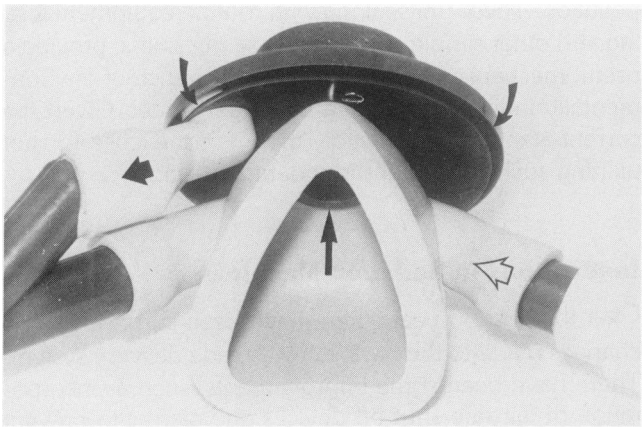
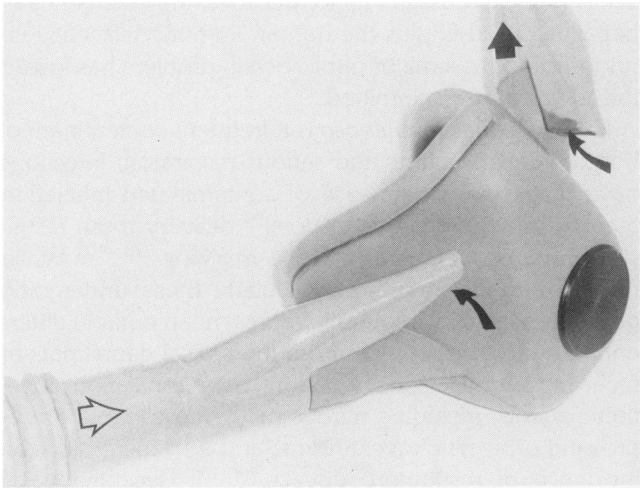


Figure 10. Internal view of Matrx nosepiece demonstrating fresh gas inlet (open arrow) and waste gas outflow through the plastic scavenging cone (solid arrows).

piece, one bringing gas to the nosepiece and the other taking expired gas away (Figure 11). Suction is only applied to the exhaust tube when the patient exhales. Sweeper flanges on either side of the nosepiece are attached to small tubes through which suction continuously passes. These are embedded within the large corrugated tubes. The mechanism for both vacuum systems is controlled by an enclosed diaphragm valve. A flow meter with vacuum control is also part of this cylinder.

The McKesson scavenging system appears different from the other systems, but in operation is similar to the original Brown mask (Figure 12). Fresh gases are delivered to an inner mask through a single tube. Exhaled and

Figure 11. External view of a Porter nasal hood. Fresh gas inflow shown with an open arrow and waste gas removal with solid arrow.



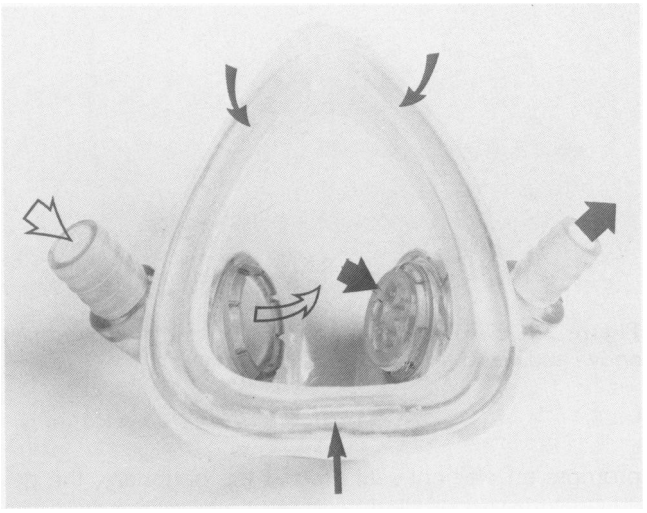
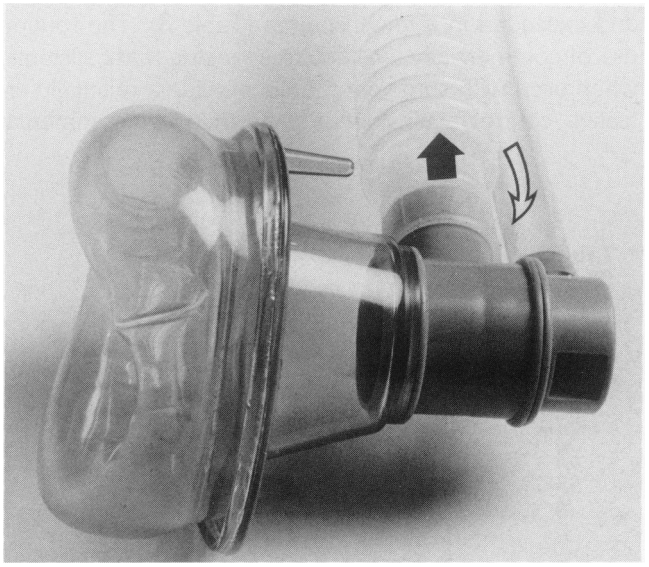


Figure 12. McKesson nosepiece demonstrating a modified mask within a mask design. Open arrows denote fresh gas flow and solid arrows waste gas removal.

excess gas passes through a one-way valve into an outer mask from which the vacuum system collects them to an outlet. While the periphery of the two masks is not open like the Brown Mask, the soft plastic material is perforated, thus permitting the perinasal area around the nosepiece to be scavenged. Although similar to the Brown mask, this device is less bulky.

A scavenging mask system, designed for disposable (single) use, is marketed under the name Comfort Cushion. A small tube delivers fresh gas to the mask and a single larger tube removes excess gas (Figure 13). To

Figure 13. Comfort Cushion nasal mask with arrows indicating gas flow and air filled plastic cushion.



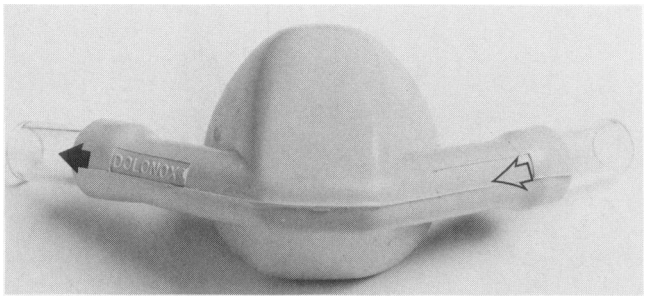


Figure 14. Dolonox nasal hood with fresh gas flow (open arrow) and waste gas removal (solid arrow).

promote an efficient seal around the periphery, the design includes an air filled "cushion," hence, the product's name. An unusual feature with this unit is the use of a neck strap to maintain the nosepiece in place.

The Dolonox scavenging unit is a relatively simple system. It consists of a nosepiece with fresh gas being supplied through a tube on one side and waste gas exiting through another tube on the opposite side (Figure 14). The exhaled gas side of the circuit terminates at a T-attachment to which an open collecting tube and a one-way valve with suction are attached. During exhalation, waste gas passes through the valve into the open collecting tube where it is evacuated by the suction. It is essential that this collecting tube be no less than 4 feet long to prevent waste gas from escaping into the environment.

Comparative evaluations have been done on all these systems under actual dental practice conditions.[92] By permitting a sufficient familiarization time with each unit prior to making any measurements and using a supervising technician during the actual data collection, reproducible results have been obtained. These suggest that with care, the 50 ppm standard can be approximated by most units and exceeded in a few instances (Table 3). The routine use of commercially available scavenging mask systems, when used with some care, does provide a relatively effective control against waste anesthetic environmental pollution. These units, along with routine equipment testing and other simple common sense measures, provide a useful mechanism to prevent the occurrence of environmentally induced disease in office personnel. Given the current standard of practice, there is no excuse for not utilizing such equipment in a dental practice.

Table 3. Comparison of Nasal Masks Tested for Scavenging Adequacy

Tests (n)	*Mask type*	*Mean ppm N_2O*
35	Brown	43.4
29	Porter	48.2
24	Parkell	54.4
23	Dupaco	61.2
33	Fraser Harlake	62.7
6	Comfort Cushion	34.8
6	McKesson	22.3
6	Dolonox	30.5

Innovations in Sedation Machines

Over the past 50 years there have been surprisingly few changes made to the basic nitrous oxide delivery system. There have been some improvements, such as incorporation of fail-safe shut off devices and alarms to prevent nitrous oxide delivery without oxygen, and multicylinder manifolds permitting transfer of gas sources without dismantling regulators. However, in the last 20 years, one of the most useful innovations has been the introduction of the mixing valve which allows a single dial to control the concentration of nitrous oxide delivered to the patient and a separate control for total gas flow. The advantages of the so-called percentage mixing units include less confusing adjustment of total gas flow and a very simple method of determining nitrous oxide dosage. A very recent approach to nitrous oxide machine design has been the use of electronic touch controls and digital readouts. Thus, touching a portion of the machine's front panel alters the concentration of nitrous oxide while another adjusts gas flow from 1 to 9.9 L/min. Although expensive and requiring availability of electrical current not needed with other units, an attractive feature of this machine is its ability to be decontaminated easily by wiping the control face with chemical disinfectants.

Accessory Sterilization

One of the historical difficulties with nitrous oxide delivery systems has been decontaminating or sterilizing them. While rarely discussed in prior decades, the emergence of hepatitis B and C plus the human immunodeficiency virus infections as a major public health problem has forced this issue to be reexamined.

Pseudomonas aeruginosa is a frequent contaminant of hospital environments and serious respiratory infections have resulted from the use of contaminated inhalation therapy equipment,[98] ventilators,[99] delivery room resuscitators,[100] as well as anesthesia machines.[101–105] While hospitalized patients and particularly those undergoing general anesthesia frequently represent an entirely different immunologic problem from the typical dental patient receiving nitrous oxide sedation, most inhalation anesthetic agents, including nitrous oxide are capable of depressing protective mechanisms, and thus increasing the incidence of respiratory disease.[106–108] Recently, nasal masks specifically used for nitrous oxide sedation have

been shown to be contaminated with a variety of human pathogens capable of transmission from one patient to another.[109]

A number of methods have been used to clean, disinfect, or sterilize masks, hoses, and reservoir bags from nitrous oxide sedation machines. These vary from simple soap and water washing, which only diminishes bacterial or viral contamination, to the use of alcohol,[110] glutaraldehydes (eg, Cidex), iodophors, and autoclaving, all of which have some disadvantages. Recently, microwave sterilization has been experimentally used on dental instruments[111] and nasal masks.[112] Note that because of the nonuniform distribution of microwaves, a special three-dimensional rotating microwave oven is requiring for effective sterilization, and the usual kitchen microwave oven is not suitable.[111]

Under the Spaulding classification of inanimate surfaces, nitrous oxide accessories are considered semi-critical in terms of infection risk.[113] Items in this group generally come in contact with intact mucous membranes and are therefore resistant to infection by common bacterial spores. Sterilization is recommended as standard of care, a disinfection procedure including washing in warm soapy water, a 10-min immersion in a glutaraldehyde solution, and thorough rinsing should be employed so that accessories are free of vegetative bacteria and viruses.

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