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Nitrous oxide pollution hazards and their prevention in dentistry

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Keywords: Nitrous oxide; pollution

SUMMARY

Nitrous oxide pollution in dental surgeries and in operating theatres is now a proven health hazard for the dentist and staff. If sufficient is inhaled then reproductive function is altered. At present, research in rats has shown the threshold limit value (TLV) for intermittent exposure to be between 1 000 and 5 000 ppm. The TLV for man has yet to be firmly established, although health authority recommendations range between 25 and 100 ppm. Whatever the TLV, scavenging can reduce pollution and should be routinely used with an approximate ratio of fresh gas flow to suction, varying from system to system, but in the order of 1:6.

OPSOMMING

Distikstofoksiedbesoedeling in tandheelkundige spreekkamers en operasiesale is tans 'n erkende gesondheidsrisiko vir beide die tandarts en sy personeel. Genoegsame inaseming van die gas lei tot veranderinge in voortplantingsfunksies. Huidige navorsing op rotte toon dat die drempelwaarde ("threshold limit value, TLV") vir onderbroke blootstelling tussen 1 000 en 5 000 dele per miljoen lê. Vir die mens is die drempelwaarde (TLV) nog nie met sekerheid bepaal nie alhoewel aanbevelings van gesondheidsowerhede wissel tussen 25 en 100 dele per miljoen. Ongeag die TLV, kan lugreiniging besoedeling verminder en behoort as roetine toegepas te word in 'n verhouding van vloei van vars gas tot suiging van ongeveer 1:6. Dié verhouding kan wissel volgens die sisteem wat gebruik word.

The administration of nitrous oxide inhalation by Horace Wells in 1844 to produce anaesthesia for dental extraction was one of dentistry's great contributions to the relief of pain (Lee and Atkinson, 1973). Subsequent innovations have included the development of a portable nitrous oxide and oxygen apparatus by Boyle in 1917, which was followed in 1933 by Minnett's machine for the self administration of nitrous oxide and air in labour (Lee and Atkinson, 1973). Thereafter the use of nitrous oxide and oxygen mixtures fluctuated in popularity until the introduction, for both continuous flow and intermittent flow machines, of "fail-safe" apparatus which prevented hypoxia through the delivery of analgesic concentrations of nitrous oxide together with at least 25 percent oxygen (Roberts, 1983). The availability of such apparatus enabled clinicians such as Langa (1976) to popularise the relative analgesia (inhalation sedation) technique which is in common use today throughout the world including in South Africa.

STUDIES IN MAN

Initially concern over the safety of nitrous oxide dealt with the risk of death due to hypoxia. This concern was alleviated by the introduction of "fail-safe" apparatus which ensured an adequate minimum concentration of oxygen. A new problem then arose, following observations by physicians in Copenhagen. Lassen and his colleagues (1956) noticed that a granulocytopaenia and thrombocytopaenia developed in tetanus patients treated with prolonged inhalations of nitrous oxide. Later, similar leucopaenic effects were seen in rats exposed to concentrations of nitrous oxide similar to that inhaled by patients (Green and Eastwood, 1963; Parbrook, 1967). The mechanism of the cytotoxic effect of nitrous oxide is not completely clear but what is known is that nitrous oxide reacts chemically with vitamin B12 to decrease vitamin activity (Banks, Henderson and Pratt, 1968, Nunn and Sharer, 1982). Evidence to support a similar leucopaenic effect after inhalation of trace quantities of nitrous oxide has not been forthcoming.

Interest in the possible effects of longterm inhalation of trace quantities of anaesthetic gases by staff using these grew. Vaisman (1967) recorded a high frequency of minor health complaints among anaesthetists but what caught her attention was that 18 of 31 pregnancies in female anaesthetists terminated in spontaneous abortion. Several other contemporary studies (Askrog and Harvald 1970; Cohen, Bellville and Brown, 1971; Knill-Jones *et al*, 1975; Cohen, Brown and Bruce, 1974) con-

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firmed this trend among operating theatre staff which is now accepted (Vessey and Nunn 1980). These effects in man have not been associated with a particular anaesthetic agent, but with exposure to general anaesthetic gases in general.

Since dentists are also users of anaesthetic gases a large national survey was undertaken in the United States by Cohen *et al,* (1980). These investigators received completed comprehensive questionnaires from 22 555 dentists and 21 390 chairside assistants. Analysis of the results indicated an increase in liver, kidney and neurological disease among dentists heavily exposed to anaesthetics. Their wives had an increased spontaneous abortion rate. Similar findings were noted among female chairside assistants, who had also been heavily exposed to anaesthetics (more than 9 hours per week). A separate analysis indicated that exposure to nitrous oxide alone could account for the increased rate of adverse response. More recently Sweeney, Bingham and Amos (1985) have reported bone marrow toxicity in dentists exposed to nitrous oxide in their surgeries.

There is evidence, therefore, that long-term exposure to anaesthetic gases, in particular nitrous oxide, is an occupational hazard to dentists and their surgery staff as far as the reproductive system is concerned. At present other untoward effects reported appear to be reversible but this has not yet been completely clarified.

STUDIES ON LABORATORY ANIMALS

Soon after attention had been drawn to a possible health hazard in man, experimental studies on laboratory animals such as mice, rats, guinea pigs and rabbits began using nitrous-oxide, halothane, isofluorane and diethyl ether. (Parbrook, 1967; Corbett *et al,* 1973; Stevens, Eger and White 1975; Ramazotto, Katz and Cupiola, 1975). These investigators showed various abnormalities of reproduction, growth, development and liver injury which varied according to the experimental conditions used. What is important is that effects noted in man were confirmed in animal experiments.

POLLUTION LEVELS

Parallel with epidemiological and experimental animal investigations into hazards of occupational exposure, methods of measuring anaesthetic gas pollution were tested based on an assumption of a causal relationship between the diseases noted and exposure to pollution. Nitrous oxide pollution was demonstrated in operating theatres by workers such as Linde and Bruce (1969), and Whitcher, Piziali and Sher (1975). Measurements were made in what was termed the breathing zone or the anaesthetist/surgeon zone and in the peripheral air — usually in the region of an airconditioning air vent.

Dental surgeries have also been shown to be polluted (Millard and Corbett, 1974; Whitcher, Zimmerman and Tonn, 1977; Cleaton-Jones *et al* 1978; Ross, Riekman and Carley, 1984), the levels in the breathing zone frequently being higher than in operating theatres. The pollution values recorded have varied considerably, for example in the USA Millard and Corbett (1974) showed levels ranging between 3 800 ppm and 6 800 ppm while in South Africa the mean pollution in 4 Johannesburg surgeries was 446 ppm.

It is difficult to compare pollution in operating theatres and dental surgeries since air conditioning systems, the number of patients treated, nitrous oxide flow rates, clinical techniques, equipment leakage, sampling methods and the presence and movement of people all influence pollution. The zone of highest risk is near the anaesthetic mask, but pollution has even been noted in a distant dental receptionist's area (Cleaton-Jones *et al* 1978).

EXPOSURE LEVELS

In spite of variations in study methodology it gradually became clear that there was a need to define a threshold limit value (TLV) for exposure. In the United States 2 levels were defined, <25 ppm in the operating theatre and <50 ppm in the dental surgery (NIOSH, 1977). A more recent recommendation has been the Swedish level of 100 ppm (National Board of Occupational Safety and Health, Sweden 1981). Other countries seem to have either accepted the United States levels or have not defined a TLV. This TLV pollution level is calculated as a time weighted mean using the following formula (Cohen 1980):

$$\text{Gas pollution (c)} = \frac{60 \times L \times 10}{n\,(L-r)V}$$

where L = rate at which non-recirculated anaesthetic gases are introduced in ℓ/min
n = number of room air changes per hour
r = fraction of air changes recirculated
V = volume of room in litres

A problem at the present time is that it is not certain what the TLV for man should be. The 25-100 ppm TLV are difficult to attain and may be too low. Halsey (1975) combined data from human and animal experiments, allowed a threefold safety factor and assumed less than 12 hours of individual exposure daily. His TLV suggestion for nitrous oxide was 300 ppm. Vieira (1981) and Vieira, Cleaton-Jones and Moyes (1983) attempted to investigate the TLV using laboratory rats. Cohorts were exposed to various pollution levels for varying periods. Using litter size as the determining variable it was noted that the TLV for reproductive defects is between 500 and 1 000 ppm after continuous exposure and between 1 000 and 5 000 ppm after intermittent inhalation. There is no information at present to show that these values can be extrapolated to man.

REDUCTION OF OCCUPATIONAL HAZARDS

General principles to prevent exposure to the occupational hazard of nitrous oxide pollution have been well described by Cohen (1980). These consist of the use of gas-tight anaesthetic equipment which must be maintained in that condition, regular monitoring of waste gases and the use of low-leakage anaesthetic techniques combined with efficient scavenging.

In the average dental surgery, relative analgesia apparatus is generally used. The tubing and connections on this can be tested using a paintbrush and soap solution or by placing tubing in a bucket of water while gases flow through the tubing. Any leaks indicated by the escape of bubbles should receive attention.

Scavenging however, is the most practical method, particularly if during administration of nitrous oxide, patients are encouraged not to mouth breathe in order to reduce exhaling of exhaust gases into the dentists' breathing zone, or if a rubber dam is used. Scavengers consist of apparatus in the nitrous oxide circuit that enable waste gases to be ducted away. There are many designs and all have been shown to work to a greater or lesser extent in the dental nasal circuit (Whitcher, Zimmerman and Tonn, 1977; Brown and Bell, 1984; Christensen, Vann and Linville, 1985; Whitcher, 1985, Sher *et al* 1986).

The scavengers comply with the guidelines laid down for the acceptance of nitrous oxide scavenging equipment by the Council on Dental Materials and Devices of the American Dental Association (1977). These require that the scavenging equipment must be adaptable to most existing sedation, anaesthesia and exhaust systems. They should be constructed so as not to significantly interfere with normal breathing systems and delivery of selected gas concentrations and be capable of attaining the lowest reasonably attainable level of nitrous oxide in the working and breathing zone of the dentist. They should be effective regardless of the heating and air conditioning system in use and they should be constructed to permit safe and efficient disposal of waste gases.

The commercially available nasal scavengers all appear to fulfil the American Dental Association criteria but it is difficult to know how to choose between the various scavenger designs from the viewpoint of efficiency. A similar problem existed with conventional anaesthetic scavengers but this was solved when Wetterhahn *et al* (1986) developed a system for comparing scavengers in the laboratory. Recently Askew, Moyes and Cleaton-Jones, (1987) modified the Wetterhahn system to enable the testing of nasal scavengers and this system offers promise. In time comparison between scavengers' efficiencies will be possible.

What has emerged from clinical studies and the recent work of Askew *et al* (1987) is that the suction flow rate and its relationship to fresh gas flow is critical. In operating theatres low pressure high flow suction, specifically designed for scavenging to outside air, is available and is effective. Dental surgeries generally have good high pressure suction installed which may be coupled to the scavenger systems. Askew *et al* (1987) have shown that a ratio of about 1:6 of fresh gas inflow to suction is needed to provide good scavenging in the comfort cushion mask (Mission Dental, USA). However, fresh gas flow/suction ratios need to be defined for each circuit and system. Thus for a 4ℓ/min inflow of nitrous oxide, at least 24ℓ/min of suction is required to minimise pollution. It is easy to measure suction in the dental surgery by means of a rotameter connected to the suction at the upper end of the rotameter. It must be remembered that waste gases must be exhausted to the atmosphere. Recirculation air-conditioners play no role in the reduction of pollution and may, through gas mixing, increase pollution in the peripheral zone of a surgery.

Any attachment to a breathing circuit has the propensity to cause malfunction in the anaesthetic breathing circuits (O'Conner, Daniels and Pfitzner, 1982). The increased complexity may result in mistakes in assembly. Obstruction of the scavenging pathway may cause a rise in airway pressure or undue negative pressure may similarly be applied. The use of negative pressure near the expiratory valve of a ventilator may cause the valve to obstruct or possibly trip prematurely. Many of the scavenging systems 'hood' the expiratory valve and this may remove sounds which are useful adjuncts to measure ventilatory efficiency. Inappropriate levels of inhalational agents may be sensed by smell but this modality is removed during scavenging.

CONCLUSION

This short article has shown that pollution with nitrous oxide is an occupational health hazard, that pollution occurs in the dental surgery and that scavenging can reduce pollution to a varying degree. Scavenging should always be done.

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Footnote
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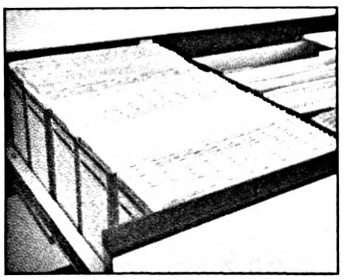

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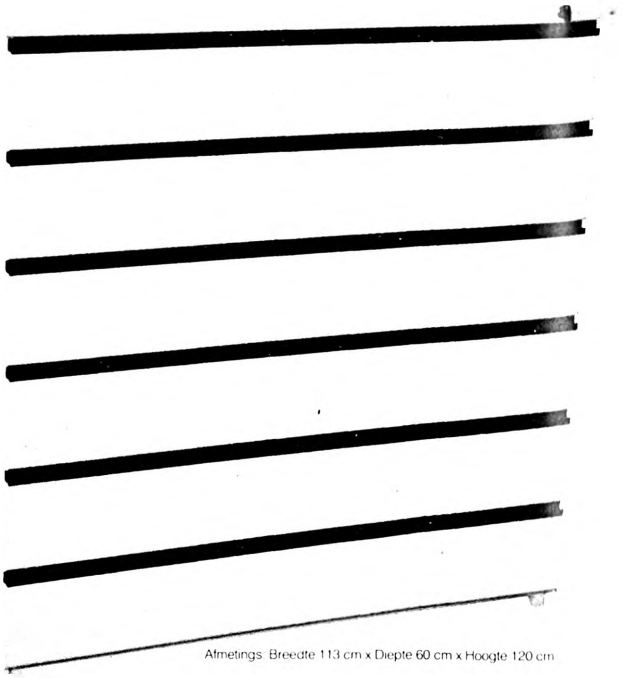

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