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Cochlear-microphonic and middle-ear pressure changes during nitrous oxide anesthesia in cats.

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Journal

The Journal of the Acoustical Society of America, 51(4)

ISSN

0001-4966

Authors

Starr, A
Schwartzkroin, P

Publication Date

1972-04-01

DOI

10.1121/1.1912985

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could be reconciled with current theories is to assume that the neural representation contains information about the phase as well as the magnitude of the stimuli

regardless of whether the stimuli are present at that moment. Whether phase information can be preserved neurally in this way is an as yet unanswered question.

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Received 9 June 1970

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Cochlear-Microphonic and Middle-Ear Pressure Changes during Nitrous Oxide Anesthesia in Cats

A. STARR* AND P. SCHWARTZKROIN†

Stanford University Medical Center, Department of Neurology, Stanford, California 94305

The amplitude of cochlear-microphonic responses in cats was attenuated up to 18 dB during both induction and recovery from nitrous oxide anesthesia, with smaller changes (up to 3 dB) occurring during the periods of maintained anesthesia. This response variability was clearly related to alterations of middle-ear pressure and could not be attributed to changes in middle-ear muscle or efferent olivocochlear bundle activities. Middle-ear pressure rose during inhalation of nitrous oxide with periodic abrupt returns to baseline levels during the period of maintained anesthesia; pressure became negative after withdrawal of nitrous oxide.

In the course of studying auditory responses in cats with chronically implanted round-window electrodes, we encountered a source of response amplitude variability attributable to the use of nitrous oxide inhalation anesthesia. Cochlear microphonic (CM) responses to tone signals (1-30 kHz) varied as much as 18 dB during both induction and recovery from nitrous oxide anesthesia and up to 3 dB during maintained anesthesia. Response variability was not the result of middle-ear muscle activity, since it persisted in cats paralyzed with gallamine triethiodide or d-tubo-

curarine, as well as in cats in which the tendons of the middle-ear muscles had been sectioned. Crushing the eighth nerve had no effect on the microphonic changes, indicating that the efferent olivocochlear bundle was also not involved in this phenomenon. Two lines of evidence suggested that nitrous oxide administration affected CM responses by influencing middle-ear pressure. First, nitrous oxide enters body cavities (such as the middle ear) before the normal major component of these cavities (nitrogen) has been resorbed (Matz *et al.*, 1967; Rasmussen, 1967). If the cavity has rigid

FIG. 1. Middle-ear pressure (top trace) and integrated CM response to a steady 3-kHz tone (bottom trace) during induction of nitrous oxide anesthesia.

FIG. 2. Integrated CM response to a tone signal swept from 1–30 kHz over 10 sec. The effect of introducing positive or negative pressure into the middle ear is shown in (a) and (b). In (c), for comparison, is the effect of attenuating the sound intensity in 6-dB steps.

walls, this differential rate of gas exchange is accompanied by an increase of pressure (i.e., an increase in $p\text{N}_2\text{O}$ without a corresponding decrease in $p\text{N}_2$). A number of studies have demonstrated just such a rise in pressure within the middle ear during nitrous oxide anesthesia (Matz *et al.*, 1967; Rasmussen, 1967; Thomsen *et al.*, 1965). Second, Wever *et al.* (1942, 1948) and more recently Møller (1965) have shown that the amplitude of CMs can be affected by producing a pressure difference across the tympanic membrane. The present study was undertaken to examine CM responses and middle-ear pressure changes during nitrous oxide anesthesia.

I. METHODS

Eight cats were used in the experiments. They were anesthetized with Nembutal, with Halothane 1% in room air, or with a mixture of nitrous oxide 50%/oxygen 49%/halothane 1%, and intubated. The bulla was exposed by a posterolateral incision and a small

hole drilled to visualize the round window. A 0.007-in. stainless steel wire, insulated except for a small ball (about 0.020-in. diam) at the tip, was placed on the round window and dental acrylic used to fix the electrode to the bulla and to seal the bulla closed. In six of the animals, a polyethylene tube (PE 190, i.d. 0.047 in.) was sealed into another small hole in the bulla for measurement of middle-ear pressure or for applying known amounts of pressure from a calibrated source. All wounds were infiltrated with a long-acting topical anesthesia (Procaine in oil), and the animal was then placed in a sound attenuating chamber and supported from a screw previously cemented to his skull. The animals were paralyzed with gallamine triethiodide (40 mg/h IV), artificially respired, and body temperature maintained between 37° and 39° C. Round-window activity was amplified (0.3–30 kHz) and integrated (time constant 50 msec), and the integrator output was displayed on both a dc penwriter and a digital volt meter. Middle-ear pressure was measured by an oil-filled blood-pressure transducer (Statham) and displayed on another channel of the dc penwriter. The sensitivity of our system was sufficient to detect pressure fluctuations as low as ± 5 mm H₂O. The pressure transducer was calibrated by directly applying known pressures to the device. Sound signals (single tonal frequencies or swept tones of 1–30 kHz) were presented free field via a $\frac{1}{2}$ -in. Brüel & Kjær microphone placed close to the external auditory canal. The intensity of the individual tones was adjusted to evoke 100 μV of cochlear microphonics. The intensity of the swept tone was adjusted so that the maximal response evoked was approximately 250 μV .

II. RESULTS AND CONCLUSIONS

The relation between middle-ear pressure and CM responses was examined in four cats. The animals were prepared under Halothane anesthesia (1% in room air) and allowed to respire room air for 1 h before beginning data collection. Results were as follows.

(1) *Unanesthetized condition.* No significant deviation of middle-ear pressure from atmospheric levels was noted in the control periods. Cochlear-microphonic responses showed small amplitude fluctuations (± 1 dB) during this same period. No attempt was made to define the source of this response variability.

(2) *Nitrous oxide condition.* Within 2–5 min after introducing the nitrous oxide (N₂O 50%/oxygen 50%) middle-ear pressure began to increase at a slow steady rate (2–5 mm H₂O/min) (Fig. 1). Pressure rose to about 140 mm H₂O (range in the four cats was 80–140 mm H₂O) and then dropped sharply back towards 0 mm H₂O. This cycle of slow pressure rise and abrupt fall was repeated several times, usually with each succeeding pressure drop occurring at a lower pressure level. After 10–25 min of inhaling nitrous oxide, the

pressure swings ceased, and middle-ear pressure remained in equilibrium with atmospheric pressure. The episodes of sudden loss of middle-ear pressure can probably be attributed to transient opening of the Eustachian tube with venting of the middle ear into the nasopharynx. The failure of the Eustachian tube to close following one of these ventings most likely accounts for the stabilization of middle-ear pressure close to atmospheric levels.

Cochlear-microphonic amplitudes changed inversely with middle-ear pressure. Response amplitudes gradually decreased with the slow rise of pressure and then increased again towards control values upon venting of the middle ear. When middle-ear pressure stabilized at atmospheric levels CM also became stable at maximum amplitude.

(3) *Nitrous oxide discontinued.* Within 5 min after discontinuing nitrous oxide, middle-ear pressure gradually decreased below atmospheric levels, stabilizing at values close to -100 mm H₂O. A negative pressure was maintained for several hours. The amplitude of CMs gradually decreased during the negative pressure shift, finally leveling off when middle-ear pressure became constant. The development of negative middle-ear pressure is apparently the consequence of a rapid diffusion of N₂O out of the bulla accompanied by closure of the Eustachian tube. Equilibrium of middle-ear pressure with the atmosphere is thus dependent solely on gas exchange through the mucous membrane lining the cavity or through the tympanic membrane.

Control experiments confirmed that middle-ear pressure changes were indeed responsible for the variations in cochlear responses during nitrous oxide anesthesia. In two animals, CMs in responses to tone sweeps were

recorded while pressure in the middle ear was altered. Positive and negative pressure changes (about 100 mm H₂O) caused reduction of CM amplitude similar in magnitude to the changes we had observed in our chronic preparations (10–18 dB) (Fig. 2). Furthermore, pressure changes as low as ± 10 mm H₂O could noticeably affect the amplitude of CMs.

Two other cats were implanted bilaterally (under Halothane/N₂O anesthesia) with round window electrodes. One bulla was sealed with dental cement and the other left open, so that pressure in the middle ear could not build up. When N₂O was withdrawn (or administered again), CMs recorded from the sealed bulla changed in the expected fashion, but response amplitude remained stable on the open side.

Finally, we can say with certainty that it was the N₂O, not Halothane or oxygen, that brought about pressure changes in CM variability. Halothane administered alone had no such effects; oxygen and room air were used interchangeably without any noticeable difference.

Nitrous oxide should obviously be used with caution in experiments analyzing auditory responses. A recent report on the effect of nitrous oxide on decreasing scalp-recorded auditory responses in man probably reflects middle-ear pressure changes rather than any alteration in central auditory mechanism (Lader and Norris, 1968). Furthermore, middle-ear pressure fluctuations may be a source of response variability particularly in experiments in which animals are immobilized or anesthetized for long periods.

* Supported by Grant No. NB 05700, National Institute of Neurological Diseases and Stroke, and CDA No. NB 31242, NINDS.

† Recipient of an NSF Fellowship.

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