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Laboratory Report

Failure of Nitrous Oxide to Inhibit Transformation of Lymphocytes by Phytohemagglutinin

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Nitrous oxide, in an incubator concentration of 70 per cent, failed to inhibit nuclear enlargement of lymphocytes stimulated to transform by phytohemagglutinin (PHA). (Key words: Anesthetics, gases, nitrous oxide; Blood, lymphocytes; Immune response, phytohemagglutinin.)

SINCE THE REPORTS that halothane inhibited transformation of human lymphocytes by phytohemagglutinin (PHA),[1,2] we have studied this phenomenon in greater detail using that anesthetic agent.[3,4] It also was of interest to see whether nitrous oxide (N_2O) had a similar effect. Studies have now been done with N_2O, and show no effect of this agent on lymphocyte transformation.

Materials and Methods

Cultures were prepared, incubated, stimulated with PHA-P, and nuclear volume changes following PHA were determined as described previously.[3] CO_2 was delivered at a flow rate determined empirically to maintain a 5 per cent concentration of this gas in the air-containing incubator. The anesthetic-containing incubator was supplied with flows of gases to maintain the following concentrations: N_2O, 70 per cent; O_2, 25 per cent; CO_2, 5 per cent. N_2O and CO_2 were monitored by gas chromatography and O_2 by paramagnetic measurements. Due to large leaks in the anesthetic incubator, high flow rates were necessary and the "G" cylinder of N_2O was changed every 12 hours. The high flows initially caused cultures to evaporate significantly, and by trial and error a humidification system was developed to prevent this. From the same donor, cultures of 10^6 lymphocytes were exposed in triplicate to one of four conditions: air, no PHA; air with PHA; N_2O, no PHA; N_2O with PHA. Cells were harvested for nuclear volume determinations 3, 6, 24, 48 and 72 hours after PHA addition. Two complete experiments were done according to this design. Data were analyzed by the t test for non-paired data. Significance was assigned to $P < .05$.

Results

From 24 hours post-PHA to the 72-hour measurements, the mean nuclear sizes were significantly larger than control values, as found in the previous studies.[3] In sharp contrast to that study, done with halothane, the anesthetic (N_2O)-treated cultures were not different from air-exposed ones. Comparisons made between air- and N_2O-treated cells at the various time intervals showed no significant difference in the extents of nuclear enlargement caused by PHA.

Discussion

Nitrous oxide was given in concentrations used clinically and did not inhibit lymphocyte transformation. The earlier studies with halothane involved concentrations having considerably higher anesthetic potency than that of 70 per cent N_2O. Thus, the present study does not rule out a similar effect of equipotent concentrations of these agents, but it does suggest that clinical usage of N_2O will not be immunosuppressant.

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3. Bruce DL: Halothane effect on nuclear volume of PHA treated human lymphocytes. Reticuloendothel Soc J 15:497–502, 1974
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Perinatology

MIXED CORD COMPRESSION One hundred twenty-two consecutive patients were electronically monitored in labor. The purpose was to relate a variable acceleration–acceleration:variable deceleration ("mixed cord compression") pattern to umbilical cord compression by comparing the monitor patterns to abnormal cord positions, length, or presentation as noted at delivery. Standard, accepted definitions of fetal heart rate patterns were used in addition to this combined pattern. Thirty-four (27.9 per cent) babies had abnormal cord positions. Of the 34, 28 (82.4 per cent) of these cords were in patients with variable decelerations of the mixed acceleration:variable deceleration pattern; 19 of the 28 (67.8 per cent) showed the mixed pattern, and nine (32.3 per cent) were variable decelerations. Accelerations alone had the same incidence of abnormal cord positions as normal monitor records. A review of the literature on the dynamics of umbilical cord compression and speculation on the results, as they relate to cord compression, are included. *(Goldkrand JW, Speichinger JP: "Mixed Cord Compression," Fetal Heart Rate Pattern, and Its Relation to Abnormal Cord Position. Am J Obstet Gynecol 122: 144–150, 1975.)*

FHR MONITORING A new method of continuous fetal heart rate monitoring, employing for cardiotachometry the fetal electrocardiogram obtained from electrodes placed on the maternal abdomen, was evaluated over a period of 26 months at the Lying-in Division of the Boston Hospital for Women. A total of 2,460 hours of intrapartum monitoring was analyzed. This "noninvasive" method of fetal ECG-based monitoring was shown to be as accurate as the direct scalp electrode method and more reliable than indirect ultrasound. Useful fetal monitoring, from very early labor to the time of delivery, was possible in 91 per cent of 507 patients using maternal skin electrodes alone. Beat-to-beat variability determinations, possibly of significance in evaluating fetoplacental function in the antepartum period, were precise and without the artifactuality of ultrasonic or phonocardiographic methods. *(Leventhal JM, and others: A New Method of Fetal Heart Rate Monitoring. Obstet Gynecol 45: 494–500, 1975.)*

MATERNAL HYPERTHERMIA The role of hyperthermia in the absence of infection has been investigated in the pregnant baboon. Twenty-three near term animals were used. Catheters were placed in the maternal esophagus and fetal esophagus. Maternal temperature was raised to between 41 and 42 C by applying external heat. The temperature gradient between fetus and mother (ΔT_{F-M}) was 0.47 degree C under steady-state conditions with maternal temperature 38 C, and rose to 0.75 degree C at 42 C. Hyperthermia caused a twofold increase in uterine activity: metabolic acidosis developed in the mother and profound acidosis and hypoxia developed in the fetus. There was also a marked fall in blood pressure with increases in heart rates of both mother and fetus; late deceleration of the fetal heart rate occurred at a higher oxygen tension and pH_a than has been observed under normothermic conditions. *(Morishima HO, and others: Increased Uterine Activity and Fetal Deterioration during Maternal Hyperthermia. Am J Obstet Gynecol 121: 531–538, 1975.)*