

BRIEF COMMUNICATION

Robert G. Meny,¹ M.D.; Hendrik J. Vreman,² Ph.D.; David K. Stevenson,² M.D.;
Fern R. Hauck,³ M.D., M.S.; Edmund R. Donoghue,⁴ M.D.; John E. Smialek,^{5,6} M.D.;
David R. Fowler,⁵ M.D.; and H. Ronald Zielke,¹ Ph.D.

Failure to Detect Elevated Levels of Carboxyhemoglobin in Infants Dying from SIDS*

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ABSTRACT: Carboxyhemoglobin (COHb) levels were determined in stored blood samples from 91 infants diagnosed to have died from the sudden infant death syndrome (SIDS) ($0.59 \pm 0.41\%$, excluding one outlying value of 10.83%); 48 age-matched controls ($0.53 \pm 0.38\%$); and three individuals who died from fire related causes ($41 \pm 20\%$). No statistical differences in COHb levels were detected between blood from SIDS and control infants ($p = 0.43$).

KEYWORDS: forensic science, sudden infant death syndrome, carbon monoxide, carboxyhemoglobin

The greater incidence of sudden infant death syndrome (SIDS) in the winter months and in poor families raises the question of whether faulty heating or cooking systems with excessive carbon monoxide (CO) emission might be contributory factors. It has been reported that the levels of carboxyhemoglobin (COHb) did not differ between infants who died in hospitals or at home in England from ascertainable causes and in infants ($n=27$) who died at home from unknown causes (1). The latter group would be expected to contain a significant proportion of infants who now would be diagnosed as SIDS cases. In another study of 87 infants, six infants had elevated COHb levels (range 7-49%), but none of these infants were in the group of 21 unexplained deaths (2). In a third study of 130 cases of which 103 were identified as having died of SIDS, COHb levels were measured in selected cases, but results were not

stated other than that COHb levels were not abnormal (3). In a fourth report it was stated that there was no evidence for CO poisoning in over 500 necropsies performed on infants diagnosed as "cot deaths" (4), but no actual COHb levels were published. Since these reports were published the criteria for the diagnosis of SIDS have become more stringent and uniform. The current study was undertaken in order to verify that the conclusions drawn from these earlier reports are still valid. This report is the first to report COHb levels in a large number of SIDS victims and controls using sensitive and accurate gas chromatographic analysis.

Experimental Subjects

Experimental subjects consisted of infants less than one year of age who came to autopsy either at the Office of the Medical Examiner of Cook County, Chicago, IL or at the Office of the Chief Medical Examiner, Baltimore, MD. The study population consisted of 91 infants diagnosed as SIDS, 48 age-matched controls and three individuals who died from fire related causes. Permission for retention of tissue at autopsy was obtained from next of kin prior to autopsy.

The cause of death was ascertained following the autopsy and review of the death scene investigation and medical history. Assignment of SIDS as cause of death adhered to NIH guidelines (5), namely, the sudden death of an infant under one year of age which remains unexplained after a thorough case investigation, including performance of a complete autopsy, examination of the death scene, and review of the clinical history. The attributed cause of death for the control group consisted of accidental deaths, asphyxia, complications due to prematurity, cardiac disorders, dehydration, hepatic necrosis, hypoxia, multiple congenital anomalies, seizure disorder, unexplained death, and viral infection. The average age of the SIDS group was 90 ± 58 days (range 19 days to 361 days) and of the control group 116 ± 71 days (range 3 days to 286 days).

Additional information was requested on two cases with elevated COHb levels. The cause of one non-SIDS case was listed as "unexplained death." The results from this case were not included in the calculation of average COHb level because of uncertain circumstance associated with the death, namely, recent bone fractures prior to the death of the infant. Attempts to locate the family at the time of compilation of the data to obtain further information regarding their heating system proved unsuccessful. Additional information was also obtained for the one SIDS case with elevated

¹Department of Pediatrics, University of Maryland, Baltimore, MD.

²Department of Pediatrics, Stanford University Medical Center, Stanford, CA.

³Department of Family Medicine, Loyola University Chicago Stritch School of Medicine, IL.

⁴Office of the Medical Examiner of Cook County, Chicago, IL.

⁵Office of the Chief Medical Examiner, State of Maryland, Baltimore, MD.

⁶Deceased, Chief Medical Examiner, State of Maryland, Baltimore, MD.

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TABLE 1—Carboxyhemoglobin levels in control cases and SIDS infants.

Diagnosis	Age (days)	Male (n)	Female (n)	African and American (n)	Caucasian (n)	Hispanic (n)	Oriental	COHb (% of Total Hb)
SIDS	90 ± 58	49	42	60	24	6	1	0.71 ± 1.15 (n = 91, p = 0.32) 0.59 ± 0.41 (n = 90, p = 0.43)*
Infant controls	116 ± 71	30	18	33	12	3	0	0.53 ± 0.38 (n = 48)
Fire-related deaths	175d, 289d, 48 yr.	3	0	2	1	0	0	41 ± 20 (n = 3)

Values are mean ± standard deviation. The *p* values were determined relative to the infant controls.

* Values with one outlier (10.8%) excluded.

COHb levels. No exogenous source of CO was evident. The infant died in March. Neither parent smoked, no kerosene heater was in the house, the fireplace was not in use at the time, an outside heat/AC pump was used to heat the house and an electric stove was used for cooking.

Three fire victims, including one adult, were included to demonstrate values of CO typically seen when there is known CO exposure and to show that storage of blood does not affect the level of COHb. Their ages were 25 weeks, 41 weeks, and 48 years.

Methods

Blood samples were obtained with a sterile syringe via cardiac puncture and frozen at -80°C in sealed polypropylene tubes. Eighty-six samples were collected in the course of the Chicago Infant Mortality Study and 57 samples were collected at the Office of the Chief Medical Examiner in Baltimore, MD by personnel of the Brain and Tissue Bank for Developmental Disorders at the University of Maryland. The time between collection and assay for the Chicago samples was 3.5 to 5 years. The time between collection and assay for the Baltimore samples was 4 to 5.5 years. None of the samples had been thawed more than once before they were sampled for this study. The samples were assigned a random number, sealed in a capillary tube (6), and shipped on wet ice for analysis. Three blood samples from two infants and one adult who died from fire related causes were obtained in Baltimore, MD. COHb was determined by gas chromatography and reduction gas detection with a Reduction Gas Analyzer, Model 2 (Trace Analytical, Inc, Menlo Park, CA) (7). Mean values of COHb for the SIDS and control infants were compared by using the Student's *t*-test.

Results

COHb levels from 91 infants diagnosed to have died from SIDS (0.59 ± 0.41%, excluding one outlying value of 10.83%) were not statistically different (*p*=0.43) from 48 age-matched controls (0.53 ± 0.38%) (Table 1). There was no statistical difference in COHb levels in control or SIDS infants collected in Chicago compared to Baltimore. Two cases, one SIDS and one control, had unexpected high levels of COHb. Both these cases were resampled, assigned new random numbers, and reassayed with similar results (10.8 vs. 8.2 % COHb and 3.7 vs. 2.6% COHb, respectively). The average COHb level for the SIDS population is presented both with and without inclusion of the 10.8% value. As expected, COHb levels in the three fire-related deaths was markedly elevated in the frozen blood samples (41 ± 20% COHb). There was no statistically sig-

nificant difference between COHb levels in SIDS infants and infants who died from other causes (excluding fire victims).

Discussion

These results support earlier findings that acute CO poisoning is not an immediate cause of death for SIDS infants. However, it does not address the hypothesis that intrauterine exposure of the fetus to CO due to smoking may impair the normal development of the fetus, making it more susceptible to stresses after birth that result in loss of life (8). The stability of COHb has not been established; however, Vreman et al. (9) have reported that it is stable at 4°C for at least two months. Furthermore, the COHb levels in blood of individuals with fire-related deaths were elevated when stored for a time equal to the study samples.

A previous report indicated a range of COHb levels from 0.5 to 6.7% in infants with known causes of death (1). Likewise, in another study six infants with known causes of death out of 87 cases studied had elevated COHb levels ranging from 7% to 49%, without an apparent explanation for these elevated levels (2). A follow up examination of the one SIDS case with elevated COHb in the current study did not indicate the presence of any factor contributing to elevated COHb. Although not likely, a mix up of samples cannot be fully ruled out. The data suggest that the other 90 SIDS victims did not die of acute CO poisoning.

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Additional information and reprint requests:
H. Ronald Zielke
University of Maryland
Pediatric Research, 10-035 BRB
655 W. Baltimore St.
Baltimore, MD 21201