

Carbon Monoxide Poisoning in Children Riding in the Back of Pickup Trucks

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Objective.—To describe the case characteristics of a series of children poisoned with carbon monoxide while traveling in the back of pickup trucks.

Design.—Pediatric cases referred for treatment of carbon monoxide poisoning with hyperbaric oxygen between 1986 and 1991 were reviewed. Those cases that occurred during travel in the back of pickup trucks were selected. Clinical follow-up by telephone interview ranged from 2 to 55 months.

Setting.—A private, urban, tertiary care center in Seattle, Wash.

Patients.—Twenty children ranging from 4 to 16 years of age.

Intervention.—All patients were treated with hyperbaric oxygen.

Main Outcome Measures.—Characteristics of the poisoning incident and clinical patient outcome.

Results.—Of 68 pediatric patients treated for accidental carbon monoxide poisoning, 20 cases occurred as children rode in the back of pickup trucks. In 17 of these, the children were riding under a rigid closed canopy on the rear of the truck, while three episodes occurred as children rode beneath a tarpaulin. Average carboxyhemoglobin level on emergency department presentation was $18.2\% \pm 2.4\%$ (mean \pm SEM; range, 1.6% to 37.0%). Loss of consciousness occurred in 15 of the 20 children. One child died of cerebral edema, one had permanent neurologic deficits, and 18 had no recognizable sequelae related to the episode. In all cases, the truck exhaust system had a previously known leak or a tail pipe that exited at the rear rather than at the side of the pickup truck.

Conclusions.—Carbon monoxide poisoning is a significant hazard for children who ride in the back of pickup trucks. If possible, this practice should be avoided.

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INJURIES commonly occur while children ride in the back of pickup trucks, but published reports typically are limited to musculoskeletal trauma.^{1,2} Over the past few years, we have treated several children for carbon monoxide poisoning sustained while they were passengers in the back of pickup trucks.

Carbon monoxide intoxication is common in the United States, with an es-

timated 10 000 persons seeking medical attention or missing at least 1 day of normal activity because of the syndrome each year.³ Approximately 3800 individuals die annually from carbon monoxide poisoning,³ making it the most common cause of death from poisoning. Internal combustion engines account for 75% of the carbon monoxide generated by human activities,⁴ with automobile engines being the most prolific. Emission standards for automobiles vary by state, but carbon monoxide levels as high as 8% (80 000 ppm) are permitted.⁵ Compare this with the maximal safe level for instantaneous carbon monoxide expo-

sure in the workplace of 200 ppm.⁶ Exposure to motor vehicle exhaust, even when substantially diluted, thus has significant potential to cause human injury. Unfortunately, such exposure may occur accidentally because individuals are unaware of the risk posed by certain activities.

Methods

Records of patients treated for severe carbon monoxide poisoning in the Hyperbaric Department of Virginia Mason Medical Center, Seattle, Wash, from 1986 to 1991 were reviewed retrospectively. Pediatric patients were defined as individuals younger than 18 years of age at the time of treatment. When identified, charts of pediatric patients were reviewed for the cause of carbon monoxide exposure. All poisonings that occurred while the individuals were passengers in the back of pickup trucks were selected for this report. Information was collected from emergency department records and interviews at the time of hyperbaric treatment (department records). All patients and/or their parents were subsequently interviewed by telephone to obtain missing data and to determine long-term outcome.

Carboxyhemoglobin levels reported represent those values measured during initial emergency department evaluation. Initial evaluation was sometimes performed at an emergency facility outside our institution. Prior to obtaining blood samples for carboxyhemoglobin determination, all patients had been removed from the source of carbon monoxide exposure, and many were treated with supplemental oxygen during emergency transfer from the accident site.

Symptoms described in individual

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Table 1.—Case Characteristics in Group With Loss of Consciousness

Age, y	Sex	Duration of Exposure, min	COHb, %*	Other Symptoms or Signs	Outcome
5	M	10	19.6	Lethargy	Normal
7	F	10	21.1	Lethargy, headache, dizziness, nausea	Normal
10	M	45	15.9	Headache, ataxia, combativeness	Normal
4	M	60	11.2	None reported	Normal
8	M	60	13.1	Headache	Normal
13	M	45	36.8	Nausea, lethargy, headache	Normal
16	F	45	25.5	Headache, confusion	Normal
15	F	45	27.9	Headache, weakness, ataxia	Normal
13	F	45	31.5	Lethargy	Normal
5	M	15	22.0	Combativeness	Normal
11	F	120	15.9	Nausea	Normal
7	F	20	2.5	Nausea, headache	Normal
5	M	20	1.6	None reported	Normal
11	F	150	3.0	Lethargy, confusion, combativeness	Death due to cerebral edema
14	F	150	2.8	Lethargy, combativeness	Memory deficits

*COHb indicates carboxyhemoglobin.

Table 2.—Case Characteristics in Group Without Loss of Consciousness

Age, y	Sex	Duration of Exposure, min	COHb, %*	Other Symptoms or Signs	Outcome
8	F	10	15.1	Lethargy, dizziness, conjunctival irritation, blurred vision	Normal
6	F	10	15.6	Headache, lethargy, blurred vision	Normal
10	M	60	17.7	Lethargy, headache, disorientation	Normal
13	F	45	29.1	Headache, confusion, dizziness	Normal
8	M	15	37.0	Somnolence, nausea, dizziness	Normal

*COHb indicates carboxyhemoglobin.

cases are those recorded during initial evaluation and treatment, as well as those recalled by the patient during follow-up interviews. They typically were symptoms spontaneously volunteered by the patient and were not collected by use of a standard questionnaire.

Patients were treated with hyperbaric oxygen in a multiplace hyperbaric chamber. When more than one individual was poisoned in the same incident, the patients were treated simultaneously. Treatment consisted of hyperbaric oxygen administration at 2.8 atmospheres of absolute pressure for 46 to 92 minutes. In four cases, additional oxygen was also administered at 1.9 atmospheres of absolute pressure. Treatment duration was based on the severity of initial clinical presentation.

Results

Sixty-eight pediatric patients were treated for accidental carbon monoxide poisoning at our facility during the 6-year period examined. Of these, 20 cases occurred as children rode in the back of pickup trucks. In 17 episodes, children were riding under a rigid closed canopy on the rear of the pickup. In the remaining three episodes, children rode beneath a tarpaulin. Carbon monoxide exposures were frequently grouped, and

eight separate incidents were responsible for the 20 cases.

Loss of consciousness occurred at least transiently in 15 patients. Individual data for children with and without loss of consciousness are presented in Tables 1 and 2, respectively. Carboxyhemoglobin levels for all patients ranged from 1.6% to 37.0%, averaging $18.2\% \pm 2.4\%$ (mean \pm SEM). These levels did not differ significantly between the group with loss of consciousness (range, 1.6% to 36.8%; mean, $16.7\% \pm 2.9\%$) and the group without loss of consciousness (range, 15.1% to 37.0%; mean, $22.9\% \pm 4.3\%$). Patient ages in the two groups were also similar. In addition to loss of consciousness, symptoms included headache (10 patients), lethargy (nine), nausea (five), dizziness (four), combativeness (four), confusion (three), blurred vision (two), ataxia (two), conjunctival irritation (one), somnolence (one), disorientation (one), and weakness (one). These symptoms did not correlate with either carboxyhemoglobin level or history of loss of consciousness.

In one incident involving four children, two were dead when discovered. They were not referred for hyperbaric treatment and are not, therefore, included in this series. A third child in that incident died of cerebral edema

within 48 hours, despite hyperbaric oxygen treatment. The fourth sibling was also treated, but manifests residual neurologic injury, primarily in deficits in memory. Data regarding the latter two children are included in Table 1. The remaining 18 children in this series were described by their parents as normal at follow-up interviews ranging from 2 to 55 months after treatment. Parents noted no recognizable sequelae related to the episode, reporting that all were exhibiting normal social development and progressing normally in school. Neuropsychiatric testing was not performed on these children.

In the six cases for which information was available, the pickup trucks were typically older model vehicles, ranging from 8 to 18 years of age. Children were riding under canopies in seven of the eight incidents. Canopy windows and doors were closed in all seven instances. In every case, vehicle exhaust systems exited from the side of the pickup and had a known leak (three vehicles), exited from the rear of the pickup and had a known leak (three vehicles), or exited from the rear of the pickup without a recognized leak (two vehicles). No instances occurred in pickups with a tail pipe that exited from the side of the pickup and had no recognized exhaust leak.

Comment

The cases discussed demonstrate the potential risk of carbon monoxide poisoning in children who travel as passengers in the back of pickup trucks. Carbon monoxide poisoning in school buses with faulty exhaust or ventilation systems has been reported⁷; however, association of this hazard with travel in the rear of pickup trucks has not previously been described. The rate of occurrence of this problem is unknown, but it is likely that additional cases of both similar and lesser severity occurred in the Seattle region during the same period. These cases may not have been referred to our facility for treatment because of either the lack of need for hyperbaric oxygen treatment or failure to recognize the syndrome. Typical symptoms experienced by the children in this study (such as dizziness, nausea, and headache) may be attributed by parents to motion sickness, viral illness, or other causes. In fact, many of the parents of these patients initially believed their children to be sleeping in the back of the pickup truck when they in fact were unconscious due to carbon monoxide intoxication.

Misdiagnosis of carbon monoxide poisoning by physicians is well described. The syndrome may not be suspected in

patients arriving in emergency departments with flu-like illnesses.⁸ If universal screening of patients in emergency departments is performed, 3% to 5% of all patients are found to have elevated carbon monoxide levels.⁹ Among patients ultimately found to have significant carbon monoxide poisoning, up to 30% may carry an erroneous initial diagnosis.¹⁰ Such reports underscore the strong possibility that other children have experienced carbon monoxide poisoning in incidents similar to those described, yet the syndrome may have gone undiagnosed or unreported.

Carbon monoxide intoxication can cause injury to hypoxia-sensitive tissues such as in the brain and the heart, resulting in permanent damage or death.⁴ Delayed neurologic deterioration following significant carbon monoxide exposure may also occur after a lucid interval ranging from 2 days to 6 weeks,¹¹ emphasizing the importance of long-term follow-up when attempting to study the effects of carbon monoxide poisoning.

The 20 children in this study were all treated with hyperbaric oxygen. The use of hyperbaric oxygen is generally recommended in cases of carbon monoxide poisoning when patients have (1) a carboxyhemoglobin level of 25% or greater, (2) anginal pain or ischemic changes on an electrocardiogram, or (3) any degree of neurological impairment, regardless of the carboxyhemoglobin level, including transient loss of consciousness.⁴ These are the criteria that were applied in determining the need for hyperbaric treatment in our series.

It is possible that patients may meet none of these criteria and still have significant carbon monoxide intoxication. The absolute indications for hyperbaric oxygen treatment remain controversial.

Neuropsychiatric testing has been proposed as a method for detecting individuals with subtle neurologic impairment¹²; however, appropriate testing for use in children has yet to be developed. Benefit from hyperbaric oxygen is thought to result from an enhanced rate of clearance of carbon monoxide from hemoglobin and tissue, as well as rapid restoration of tissue oxygen sufficiency by improvement in peripheral oxygen delivery.⁴ Oxygen at atmospheric pressure is appropriate for milder cases of carbon monoxide intoxication.

In the present series, specific configurations of the vehicle exhaust systems were associated with the injury in every instance. A leaking exhaust system or a tail pipe exiting beneath the rear bumper of the pickup truck was always present. We hypothesize that exhaust emitted at the rear of the moving pickup truck is drawn upward via the Venturi effect. In the case of canopy-related incidents, fumes presumably enter the bed of the vehicle through the space between the bed and tailgate, or between the tailgate and canopy door. We have seen no cases involving passengers in campers on the rear of pickup trucks, or in pickups with intact exhaust systems exiting the side of the vehicle.

Most pickup trucks are currently manufactured with tail pipes that exit the side of the vehicle, typically at the rear wheel well. When exhaust systems are worn or damaged, however, they may be replaced with systems that exit the rear of the vehicle. This practice should be avoided. Prompt attention to exhaust system maintenance would likely have prevented some of the cases reported. Exhaust leaks were known to exist by many drivers in this series prior to the incident, but they had not been repaired

because the risk to passengers in the rear of the pickup truck was not recognized.

Patients in this study were traveling legally in the back of pickup trucks. Washington State law addresses the issues of child restraints and safety belts (Revised Code of Washington, Sections 46.61.687 and 46.61.688), but allows passengers to ride in the rear of pickup trucks if all safety belts within the cab are in use. Nine states regulate travel in the rear of open pickups, but only New Jersey prohibits such travel entirely.¹³ Most state laws recognize the hazard of travel in the rear of open pickup trucks without specific regulation of such activity. Few address the issue of travel in an enclosed pickup truck bed, the activity associated with carbon monoxide intoxication in our patients.

One study that examined traumatic injury to passengers in the back of pickup trucks suggests that state legislatures should act to strictly regulate or totally restrict such travel.¹ Prohibiting this type of travel would presumably eliminate the risk of carbon monoxide poisoning, as no individuals simultaneously traveling in the cab of the trucks became ill. Short of enacting such legislation, meticulous attention should first be given to the condition and configuration of the pickup's exhaust system if passengers must travel in the rear. Second, passengers in the cab should monitor the status of riders in the rear of the pickup truck. Finally, programs directed at improving community awareness of the hazards associated with riding in the back of pickup trucks may help improve public safety.

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References

1. Angran PF, Winn DG, Castillo DN. Pediatric injuries in the back of pickup trucks. *JAMA*. 1990;264:712-716.
2. Tong T, Teaford PA. Falls from pickup trucks during childhood. *AJDC*. 1989;143:997-998.
3. Program Development Branch, Environmental Health Services Division, Center for Environmental Health, Centers for Disease Control. Carbon monoxide intoxication: a preventable environmental health hazard. *MMWR*. 1982; 31:529-531.
4. Piantadosi CA. Carbon monoxide intoxication. In: Vincent JL, ed. *Update in Intensive Care and Emergency Medicine*. New York, NY: Springer-Verlag NY Inc; 1990;10:460-71.
5. Guss DA, Neuman TS. Carbon monoxide poisoning: how to detect and what to do. *J Respir Dis*. 1990;11:773-786.
6. Occupational Safety and Health Administration, US Dept of Labor. *Air Contaminants: Permissible Exposure Limits*. Title 29 Code of Federal Regulations (part 1910.1000), 1989.
7. Johnson CJ, Moran J, Pekich R. Carbon monoxide in school buses. *Am J Public Health*. 1975;65:1327-1329.
8. Grace TW, Platt FW. Subacute carbon monoxide poisoning: another great imitator. *JAMA*. 1981;246:1698-1700.
9. Turnbull TL, Hart RG, Strange R, et al. Emergency department screening for unsuspected carbon monoxide poisoning. *Ann Emerg Med*. 1988;17:478-483.
10. Barret L, Danel V, Faure J. Carbon monoxide poisoning, a diagnosis frequently overlooked. *Clin Toxicol*. 1985;23:309-313.
11. Choi IS. Delayed neurologic sequelae in carbon monoxide intoxication. *Arch Neurol*. 1983;40:433-435.
12. Myers RAM, Messier LD, Jones DW, Cowley RA. New directions in the research and treatment of carbon monoxide exposure. *Am J Emerg Med*. 1983;2:226-230.
13. American Automobile Association. *Digest of Motor Laws*. 57th ed. Falls Church, Va: American Automobile Association; 1991.