

Clinical Study on Carbon Monoxide Intoxication in Children

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Carbon monoxide intoxication has long been one of the most serious public health problems in Korea. This is mainly due to the wide use of anthracite coal briquettes as domestic fuel for cooking and under-the-floor heating. One hundred and seven cases of CO intoxicated children hospitalized at Yonsei Medical center from January 1970 to December 1986 have been investigated clinically. The sex ratio was 1.3:1 (male 60 cases, female 47 cases) with the peak incidence occurring in patients between 12 and 14 years of age (28%). The most common symptoms were vomiting, convulsions and headache; and the most frequent signs were altered mental state, increased deep tendon reflex and a positive Babinski sign. The outcome of patients was as follows: 4 cases (3.7%) expired, 77 cases (72.0%) recovered without neurologic sequelae and 26 cases (24.3%) survived with neurologic sequelae. The neurologic sequelae included persistent convulsions (7 cases), cortical blindness (3 cases), peripheral neuropathy (2 cases) and delayed neurologic sequelae (11 cases). Neurologic sequelae occurred most frequently in comatose patients (45.5%) and least often in mentally alert patients (6.1%), more frequently in patients exposed to CO gas for more than 8 hours than in those exposed for less than 8 hours, and in patients who did not receive hyperbaric oxygen therapy (29.4%) than in those who did (19.6%). Delayed neurologic sequelae were mental retardation (72.7%), epilepsy (36.4%), mutism (18.2%) etc. The lucid interval in 11 cases of delayed neurologic sequelae ranged from 2 to 20 days. The results of this study suggest that every patient exposed to CO gas should receive prompt and efficient oxygenation including hyperbaric oxygen therapy and that expeditious reduction of cerebral edema may be of value. The importance of providing follow-up facilities in anticipation of a relapse of the delayed neurologic sequelae has been established.

Key Words: Carbon monoxide intoxication, neurologic sequelae

In 1895, Haldane described the mechanism of carbon monoxide (CO) toxicity by demonstrating that CO gas reversibly interacts with hemoglobin as well as blocks the binding ability of oxygen to hemoglobin, eventually it causes tissue hypoxia, which damages many organs of the human body. The brain is the most sensitive to disruptions of aerobic metabolism and is the first and most severely affected organ, resulting in death or a high incidence of neurologic sequelae often even the victim recovers.

In Korea, due to the wide use of anthracite coal briquettes as domestic fuel for cooking and under-the-floor heating, about one million cases of CO intoxication occur annually, resulting in death in about

3000 cases (Song, 1985).

Although it has long been one of the most serious public health problems, a clinical study of CO intoxication in children has not been published. The authors hope to establish guidelines for the effective treatment and estimate prognosis of CO intoxication in children with this clinical study.

MATERIALS AND METHODS

Clinical data from 107 cases of CO intoxication in children under the age of 15 years who were admitted to the Department of Pediatrics at Severance Hospital, Yonsei University from Jan. 1, 1970 to Dec. 31, 1986 were reviewed and follow-ups were done up to 8 years for long term prognosis. The following data were analyzed; age and sex distribution, symptoms and signs on admission, outcome, neurologic complications as well as delayed neurologic sequelae and lucid intervals, the incidence of neurologic se-

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Table 1. Age and sex distribution

Age (year)	Male	Female	Total (%)
≤ 2	11	4	15 (14.0)
3 - 5	5	8	13 (12.2)
6 - 8	11	10	21 (19.6)
9 - 11	19	9	28 (26.2)
12 - 14	14	16	30 (28.0)
Total	60	47	107 (100.0)

quelaes as related to the mental state on admission, hyperbaric oxygen therapy given at different mental states and duration of exposure to CO gas.

RESULTS

Age and sex distribution

Among the 107 cases of CO intoxication, 60 cases (56.1%) were male and 47 cases (43.9%) were female, with a ratio of 1.3:1. The age distribution ranged from under 2 years through 14 years and the highest incidence of intoxication occurred between 12 and 14 years of age (28%), and the incidence decreased with age (Table 1).

Symptoms and signs on admission

The most common symptom on admission was vomiting (25.2%) followed by convulsions (14.0%), headache (13.1%), irritability (10.3%), speech disturbance (2.8%), blindness (2.8%) and dizziness (1.9%).

The most frequent sign was an altered mental state (85.0%) followed by an increased deep tendon reflex (46.7%), positive Babinski sign (44.9%), tachycardia (26.2%), neck stiffness (13.1%), ankle clonus (9.3%), cherry red colored lips (4.7%), nystagmus (2.8%), ataxia (0.9%), amnesia (0.9%), hemiplegia (0.9%) and tremor (0.9%) (Table 2).

Outcome of 107 intoxicated patients

The outcomes of patients was as follows:

4 cases (3.7%) expired, 77 cases (72.0%) recovered without complication and 26 cases (24.3%) survived with various neurologic sequelae. Neurologic sequelae included persistent convulsions (7 cases), cortical blindness (3 cases), peripheral neuropathy (2 cases), speech disturbance (1 case), hearing disturbance (1 case), hemiplegia (1 case) and delayed neurologic sequelae (11 cases) (Table 3).

Table 2. Symptoms and signs on admission

	No. of cases	%
Symptoms		
Vomiting	27	25.2
Convulsions	15	14.0
Headache	14	13.1
Irritability	11	10.3
Speech disturbance	3	2.8
Blindness	3	2.8
Dizziness	2	1.9
Signs		
Altered mental state	91	85.0
Increased deep tendon reflex	50	46.7
Positive Babinski sign	48	44.9
Tachycardia	28	26.2
Neck stiffness	14	13.1
Ankle clonus	10	9.3
Cherry red colored lip	5	4.7
Nystagmus	3	2.8
Ataxia	1	0.9
Amnesia	1	0.9
Hemiplegia	1	0.9
Tremor	1	0.9

Table 3. Outcome of 107 CO intoxicated patients

Outcome	No. of cases	%
Death	4	3.7
Survivors without N.S.*	77	72.0
Survivors with N.S.*	26	24.3
Delayed N.S.*	11	10.3
Persistent convulsions	7	6.6
Cortical blindness	3	2.8
Peripheral neuropathy	2	1.9
Speech disturbance	1	0.9
Hearing impairment	1	0.9
Hemiplegia	1	0.9

* N.S.: neurologic sequelae

Incidence of neurologic sequelae related to mental state on admission and hyperbaric oxygen therapy at different mental states

Neurologic sequelae occurred most frequently in comatose patients (45.5%), and least often in alert patients (7.1%); that is, the more impaired the mental state, the greater the neurologic sequelae.

In 11 cases of comatose mental state, neurologic sequelae occurred in 2 of the 7 cases (28.6%) treated

Table 4. Incidence of neurologic sequelae related to mental state on admission and hyperbaric oxygen therapy

Mental state on admission	No. of cases	H.B.O.*	No. of cases	No. of cases of N.S.** (%)
Alert	14	with	1	0 (0.0)
		without	13	1 (7.7)
Drowsy	11	with	2	0 (0.0)
		without	9	1 (11.1)
Stuporous	22	with	13	1 (7.7)
		without	9	3 (33.3)
Semicomatose	49	with	33	8 (24.2)
		without	16	7 (43.8)
Comatose	11	with	7	2 (28.6)
		without	4	3 (75.0)
Total	107	with	56	11 (19.6)
		without	51	15 (29.4)

*H.B.O.: hyperbaric oxygen therapy

**N.S.: neurologic sequelae

Table 5. Relation between duration of exposure to CO gas and neurologic sequelae

Duration (hour)	No. of cases	No. of cases with neurologic sequelae	(%)
<8	42	5	(11.9)*
≥8	56	18	(32.1)*
Unknown	9	3	(33.3)
Total	107	26	(24.3)

* p<0.05

with hyperbaric oxygen therapy and in 3 cases (75.0%) without hyperbaric oxygen therapy; in the semicomatose mental state, 24.2% and 43.8%; in the stuporous mental state, 7.7% and 33.3%. In patients in the drowsy and alert mental states, not treated by hyperbaric oxygen therapy, neurologic sequelae occurred in 1 case respectively. It is evident that neurologic sequelae were more frequent in patients who did not receive hyperbaric oxygen therapy (29.4%) than in those who did (19.6%) (Table 4).

Relation between duration of exposure to CO gas and neurologic sequelae

In fact, it is difficult to estimate accurately the duration of exposure to CO gas, thus the authors counted as the duration of exposure, the time from going bed until the time the patient was discovered.

Table 6. Delayed neurologic sequelae of CO intoxication in 11 cases

Sequelae	No. of cases	%
Mental retardation	8	72.7
Epilepsy	4	36.4
Mutism	2	18.2
Urinary and fecal incontinence	2	18.2
Hemiplegia	2	18.2
Paraplegia	1	9.1
Monoplegia	1	9.1
Facial palsy	1	9.1
Psychosis	1	9.1

Table 7. Lucid interval in 11 patients with delayed neurologic sequelae

Interval (day)	No. of cases
1- 7	8
8-14	1
15-21	2

Neurologic sequelae occurred more frequently in patients exposed to CO gas for more than 8 hours (32.1%) than in those exposed for less than 8 hours (11.9%) by χ^2 -test ($p<0.05$): that is, the longer the exposed duration, the greater the neurologic sequelae (Table 5).

Delayed neurologic sequelae

Delayed neurologic sequelae in 11 cases included mental retardation (72.7%) followed by epilepsy (36.4%), mutism (18.2%), urinary and fecal incontinence (18.2%), hemiplegia (18.2%), paraplegia (9.1%), monoplegia (9.1%), facial palsy (9.1%) and psychosis (9.1%) (Table 6).

Lucid interval in patients with delayed neurologic sequelae

The lucid interval in 11 cases of delayed neurologic sequelae ranged from 2 to 20 days: in 8 cases from 1 to 7 days, in 1 case from 8 to 14 days and in 2 cases more than 2 weeks. Most of the delayed neurologic sequelae occurred with lucid interval less than 1 week (Table 7).

DISCUSSION

Carbon monoxide (CO) is an odorless and colorless

gas with a specific gravity of 0.97 relative to air and binds hemoglobin 230 to 270 times greater than does oxygen (O₂) in vivo to disrupt the O₂ transport system, resulting in anemic anoxia. The dissociation curve of oxyhemoglobin is shifted to the left in a carboxyhemoglobin (COHb) concentration above 45%, and so less O₂ is available to tissues at a particular O₂ tension. The affinity of CO for the iron of cytochrome oxidase and cytochrome P450 implies the possibility of an additional histotoxic element (Astrup 1972; Winter and Miller 1972; Zimmerman and Truxal 1981).

The incidence of CO intoxication in children has not been reported in other countries. In Korea, the incidence of CO intoxication in children younger than 15 years old is 7.8% in the total population, which constitutes a lower proportion as compared to 36% in the total population. This is probably due to the physiological and behavioral characteristics of the pediatric age group (Cho et al. 1981; Roh et al. 1984). In this study, the highest incidence was between 12 and 14 years (28%) and decreased as the age lessened. This is probably due to the fact that younger children usually share a bedroom with their parents and clinical symptoms are easily detected as well as in younger than older children.

The male to female ratio of CO intoxication including adults in Korea was from 1:1.4 to 1:1.96 (Kim et al. 1972; Choi 1982; Lee 1983; Park et al. 1984; Min 1986). In our series, the ratio was 1.3:1.

Pathologic findings in the cadaver due to CO intoxication include a cherry-red colored mucosa, congestion and petechial or massive hemorrhage in almost all organs. In the brain, dilatation of the veins and capillaries supplying the parenchyma and severe petechiae and ecchymoses in the basal ganglia, especially in the globus pallidus are evident. It appears first in the form of pallor of the pallidum after a period of 24 hours in CO intoxication. The well circumscribed pallidal necrosis can be attributed to the compression of the anterior choroidal artery from increased intracranial pressure, may be unilateral or bilateral and occasionally extends to internal capsule and outer segment of the globus pallidus (Lindenberg 1955; Finck 1966; Garland and Pearce 1967).

The pathogenesis of the anoxic lesion due to CO intoxication involves three main factors; decreased oxygen tension, edema and circulatory disturbance. Edema develops secondary to the anoxic lesion in blood vessels and causes changes in neuronal and neuroglial cells, increased intracranial pressure and compression of many vessels. Circulatory disturbance is caused by hypotension due to the decreased output of the left ventricle, incompetent venous drainage due to the disturbed function of right ventricle and

small vascular obstruction due to the endothelial swelling and vasoconstriction (Lindenberg 1955; Brierly 1984).

It is generally considered that clinical symptoms do not appear in COHb concentration below 10%, while tightness across the forehead and dilatation of cutaneous blood vessels appear in 10 to 20% COHb, headache and throbbing in the temples, weakness, dizziness, dimness of vision, nausea, vomiting and collapse in 20 to 40% COHb, same as previous items with a greater possibility of collapse and syncope, increased respiration and pulse rate in 40 to 50% COHb, syncope, coma with intermittent convulsions and Cheyne-Stokes respiration in 50-60% COHb, coma and intermittent convulsions, depressed heart beat, respiration and possible death in 60-70% COHb, weak pulse and slow respiration, respiratory failure and death in 70-80% COHb. Transient central nervous symptoms are disorientation and speech disturbance, but traditional cherry-red color change was reported to 6 to 24% (Smith and Brandon 1973; Winter and Miller 1976).

In our cases, the most common symptom on admission was vomiting (25.2%) followed by convulsions (14.0%) and headache (13.1%) and the most frequent sign was an altered mental state (85.0%) followed by an increased deep tendon reflex and a Positive Babinski sign (44.9%). Because anthracite coal briquettes are the main domestic fuel for cooking and under-the-floor heating in Korea, it is easy to diagnose CO intoxication from history taking, clinical findings, environmental circumstances and statements by an eye-witness.

The partial pressure of oxygen will remain near normal in CO intoxication, though O₂ saturation will be reduced. Carboxyhemoglobin in the blood can be measured spectrophotometrically with a carboxymeter and the CO concentration during expiration can be measured in parts per million by a CO analyzer (Zimmerman and Truxal 1981; Yun 1985).

In relation to laboratory data, the white blood cell and red blood cell counts increase, protein and myoglobin appear in the urine and SGOT, SGPT, LDH and CPK levels increase in the blood, among which the increase in CPK is prominent (Aronow and Isbell 1973; Ginsberg et al. 1974).

Pneumonia and pulmonary edema could be found in chest roentgenography and the EKG changes such as ischemic ST segment depression, T wave inversion and rarely, ventricular arrhythmia and atrial fibrillation are found (Garland and Pearce 1967; Aronow and Isbell 1973; Ginsberg et al. 1974).

Electroencephalographic findings consist of slow waves of low voltage, frequently with a frontal

preponderance. In general the severer the EEG findings, the severer the neurologic sequelae which may occur and the improvements in EEG findings parallel the patient's clinical condition (Garland and Pearce 1967; Ginsberg *et al.* 1967; Ginsburg and Romano 1976).

With the development of the computerized tomography scan method, it has become possible to observe the pathologic lesions due to CO intoxication. The lesions appear as symmetrical low density areas in the basal ganglia, more dense than cerebrospinal fluid in the early stage and as well defined and more lucent lacuna, symmetrical and diffuse white matter low density areas, ventricular dilation and sulcal widening as late changes (Ikeda *et al.* 1978;

Kim *et al.* 1980). Brain CT scans were performed in 16 cases and 6 (37.5%) showed diffuse cerebral edema, 2 (12.5%) bilateral low density in the globus pallidus, 1 (6.3%) cortical atrophy and moderate ventricular dilatation and 7 (43.8%) were normal (Figures 1 and 2).

The immediate therapeutic goal is to reverse cerebral and myocardial hypoxia and the secondary concern is to accelerate CO elimination. In unassisted air breathing, it takes about 90 minutes for a 20% COHb concentration to be reduced to 10% COHb in the blood by dissociation of CO from COHb. If 100% O₂ inhalation is employed, it is more rapid (Lilienthal 1950; Winter and Miller 1976).

The treatment of CO intoxication has been essen-

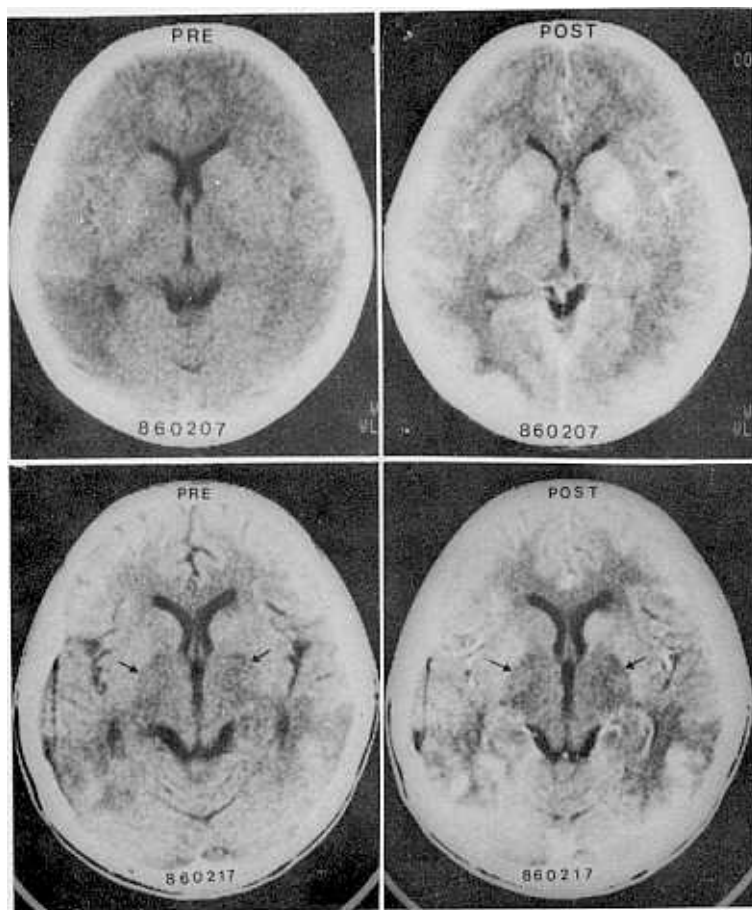


Fig. 1. Brain CT scan of 12 y-o girl with CO intoxication. (Above) Pre and post-contrast brain CT scan 10 days after intoxication. There are no definite abnormalities. (Below) Pre and post-contrast brain CT scan 3 weeks after intoxication. There are bilateral symmetrical low density areas in globus pallidus and gyral contrast enhancement associated with diffuse ischemic change.

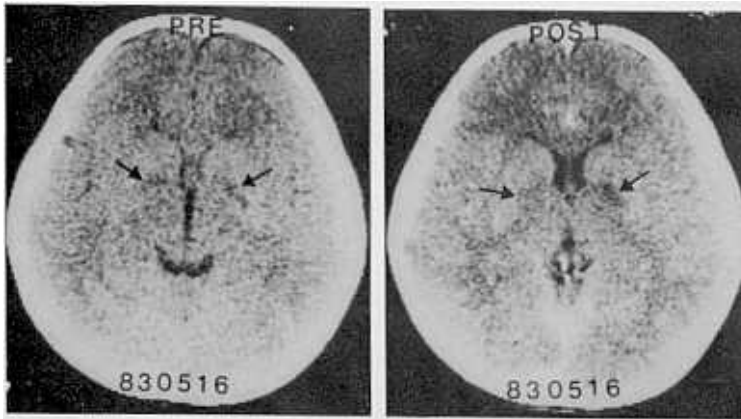


Fig. 2. Brain CT scan 3 days after CO intoxication in 8 y-o boy.
(Left) Precontrast (Right) Postcontrast. Bilateral symmetrical low density areas are seen in globus pallidus.

tially unchanged since Halane's work in 1895. Physically dissolved O_2 can contribute to tissue oxygenation, especially under hyperbaric condition. When 100% O_2 at 1 atm. is breathed, 2 vol.% of O_2 that is 33% of O_2 content of the normal arteriovenous O_2 difference is physically dissolved in the blood. At 2.5% atm., breathing 100% O_2 , 5.6 vol.% oxygen is dissolved in the blood. Thus, in addition to the reduction in the half life of COHb with O_2 therapy, O_2 delivery to severely hypoxic tissues can be improved by physically dissolved O_2 under hyperbaric conditions (Jackson and Menges 1980).

Boerema (1961) designed a hyperbaric oxygen tank and applied it clinically. By this method Smith (1965) successfully treated 32 patients of CO intoxication. Winter and Miller (1976) recommend that 100% O_2 be administered at 2 or 2.5 atm. for one hour or till the COHb level fall below 20%. If symptoms do not regress after these regimens, hypoxic damage or cerebral edema or both should be suspected. Meyers *et al.* (1981) recommend that hyperbaric oxygen for two to three hours, or till the COHb level is less than 10%. If the patient remains unconscious, a second treatment with hyperbaric oxygen is required six to seven hours later.

In Korea, there have been several reports on hyperbaric oxygen therapy in CO intoxication (Chi *et al.* 1967; Yun and Cho 1977; Park 1985). Because CO intoxication usually occurs during the night, the patient may be exposed for a long period. Therefore it is safe to treat the patient who is unconscious or has a clouded mentality with hyperbaric oxygen therapy.

Severe hypoxia can lead to cerebral edema and increased intracranial pressure, with a resultant

decrease in perfusion, thereby creating a vicious cycle. Appropriate intervention with corticosteroids and diuretics may be indicated (Winter and Miller 1976).

Prognosis depends on the duration of exposure to CO gas, COHb concentration in the blood and appropriate therapy (Shillito *et al.* 1936). Abbott (1972) reported on a young patient with a prolonged coma and decerebrate rigidity, but who eventually recovered neurologic and intellectual functions almost completely.

The level of consciousness on admission was reported to correlate well with the development of gross neuropsychiatric sequelae (Richardson *et al.* 1959; Smith and Branden 1973). In our cases, the more impaired the mental state, the greater the neurologic sequelae occurred, but one case who was alert on admission developed neurologic sequelae as well. Therefore, it is important to observe closely the patient who has been exposed to CO for a long period.

The longer the exposure to CO gas, the greater the neurologic sequelae (Lee, 1978; Park *et al.* 1984; Min 1986). In our cases, neurologic sequelae occurred in 32.1% of the patients exposed to CO gas for more than 8 hours and in 11.9% of those exposed to CO gas for less than 8 hours ($p < 0.05$). These results were similar to those of Lee (1978).

Persistent neuropsychiatric deficits that result from acute CO intoxication are well described but poorly appreciated in adults, and poorly described and even less well appreciated in children (Lacey 1981). Shillito *et al.* (1936) reported the incidence of neurologic sequelae after CO intoxication as 8%, whereas others reported it as 10 to 30% (Richardson *et al.* 1959; Garland and Pearce 1967; Smith and Brandon 1973).

In Korea, Cho *et al.* (1974) reported it as 13.6% and Choi (1982) reported it as 5.3% among the total patients population and 23.9% among hospitalized patients but a discussion of neurologic sequelae in children was found only in a case report by Min *et al.* (1961). In our study, neurologic sequelae occurred in 26 children (23.2%) of 107 hospitalized children and this was similar to that of Choi's report in adult patients (1982).

Shillito *et al.* (1936) described increased reflexes, hemiplegia, muscular hypertonia, muscle atrophies and skin hyperesthesia as neurologic changes which showed steady improvements, leading often to complete recovery from within a few days to several years, depending on the degree of the lesions and disorientation, confusion, memory defects as psychotic changes. There are about 200 cases described in the literature who have sustained notable neuropsychiatric sequelae after surviving for at least 30 days after acute CO intoxication (Garland and Pearce 1967; Smith and Brandon 1973). The most frequently reported symptoms and signs in these patients, from 30-60%, involve the so-called higher cortical functions. In order of decreasing frequency, these symptoms are memory impairment, personality alterations, specific signs of parietal lobe dysfunction, such as visual agnosia, dyspraxia, dysnomia and dysgraphia. In about 10% of the cases, motor symptoms were hemiplegia, hypotonus, extrapyramidal rigidity and akinesia. Zimmerman and Truxal (1981) reported a 13 year old patient who manifested transient cortical blindness, chorea, persistent memory and motor defects and amnesia.

In this study, 7 cases had been convulsing up to 48 hours, 3 cases with cortical blindness improved gradually from the 20th hospital day and gained cognitive functions up to 100th hospital day, 2 cases with peripheral neuropathy improved gradually for the following 2 months and cases with hearing, speech disturbances and hemiplegia gradually improved.

The exact time of the onset of symptoms of neurologic sequelae in CO intoxication is variable. The two types are seen roughly: those cases with the onset of symptoms within one week and those cases with the onset of symptoms after a lucid interval from one to three weeks. The lucid interval before the appearance of neurologic sequelae is peculiar to CO intoxication compared to the other types of anoxic encephalopathy (Shillito *et al.* 1936; Plum *et al.* 1962; Blagbrough *et al.* 1973).

The incidence of delayed neurologic sequelae was 0.05% by Shillito *et al.* (1936), 2.8% by Lee (1983), 2.4% in total patients and 10.9% in admitted patients by Choi (1983). In our study, delayed neurologic sequelae

occurred in 10.3% of the admitted patients, which were mental retardation (72.7%) followed by epilepsy (36.4%), mutism (18.2%), urinary and fecal incontinence (18.2%), hemiplegia (18.2%), paraplegia (9.1%), monoplegia (9.1%), facial palsy and psychosis (9.1%). In 6 cases followed up from 1 month to 6 years, mental retardation and intermittent convulsions experienced in 3 cases and urinary and fecal incontinence, facial palsy and psychosis improved within 6 months from onset.

There are several hypotheses in the pathogenesis of delayed neurologic sequelae: direct toxic effect of CO gas, cerebral edema, anaphylaxis and disturbance of blood vessels (Richardson *et al.* 1959; Plum *et al.* 1962; Ginsberg *et al.* 1974).

The reported lucid intervals have varied, from several hours as reported by Garland and Pearce (1967), 1 to 21 days by Shillito *et al.* (1936), 3 to 47 days by Lee (1978) and 4 to 40 days by Choi (1983). In our study, the lucid intervals varied from 2 to 22 days and were most common within 1 week.

The mortality rate due to CO intoxication was 1.8% in admission cases by Choi (1983), 0.3 to 0.4% in estimated cases by Yun and Cho (1977) and Song (1985) and 3.7% in our study.

The results of this study suggest that every patient exposed to CO gas should receive prompt and efficient oxygenation including hyperbaric oxygen therapy, and that expeditious reduction of cerebral edema with hypertonic solutions and diuretics may be of value. The importance of providing follow-up facilities in anticipation of a relapse of the delayed neurologic sequelae has been established.

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