The clinical manifestations of carbon monoxide (CO) poisoning may be mild, moderate or severe. The most serious complications are persistent or delayed neuropsychiatric sequelae, including dementia, amnestic syndromes, psychosis and parkinsonism. These symptoms may be preceded by a lucid period of 2-40 days after initial exposure. Treatment of CO poisoning-induced neuropsychiatric sequelae has not been well-defined. According to a review of the literature, hyperbaric oxygen (HBO) therapy may be effective for these complications. We report an 8-year-old boy who developed delayed neuropsychiatric sequelae 2 days after full recovery of consciousness from initial CO intoxication. His neuropsychiatric symptoms included consciousness disturbance, motor dysfunction, chorea, aphasia and agnosias. He received HBO therapy at 2.0 bar for 60 minutes once a day for 7 consecutive days. Three weeks later, he was functioning normally with no neuropsychiatric symptoms. A literature review concluded that HBO may be effective in treating neuropsychiatric sequelae. Moreover, immediate administration of HBO during acute CO intoxication may prevent these complications. [Chin Med J (Taipei) 2001; 64: 310-314]

**Case Report**

An eight-year-old boy was found unconscious in his father’s closed van, which had a blocked heating flue. According to his father, there was a duration of about 40 minutes before he was found. He received HBO therapy after initial exposure. Treatment of CO poisoning-induced neuropsychiatric sequelae has not been well-defined. According to a recent review of the literature, hyperbaric oxygen (HBO) therapy may be effective for these complications. We report an 8-year-old boy who developed delayed neuropsychiatric sequelae 2 days after full recovery of consciousness from acute CO intoxication. He received HBO therapy for 7 days. Improvement was found on the third day after completing the HBO therapy course. HBO therapy is worthy of clinical trial for neuropsychiatric sequelae of CO intoxication.
Three days after treatment, he could walk and sit. Rechecked COHb saturation was 0.2%. On day 5, he was discharged.

Unfortunately, stupor consciousness, chorea, apraxia and agnosia developed two days after discharge. The patient was sent for help to our OPD where delayed neurologic sequelae in duced by CO intoxication was impressed. Upon admission, neurological examinations showed generalized weakness and decreased DTR. He could not walk or sit, and was totally bed-ridden. Apathy was noted, and tests for aphasia (including word comprehension, word repetition, naming, reading comprehension, and writing) showed abnormal results. Cognitive function tests (such as orientation, attention, memory, and calculations) were also ab abnor mal. The electroencephalogram pattern revealed generalized low voltage indicative of diffuse cortical dysfunction. Tc-99m HMPAO brain SPECTs demonstrated hypoperfusion over the right temporal and bilateral parietal regions. Brain CT scan revealed mild decreased density over the bilateral globus pallidus region. Brain MRI showed hypersignal intensity on FLAIR and T2W1 over the bilateral globus pallidus region (Fig. 1A). These findings were compatible with post-CO intoxication change. A toxicologist was consulted and HBO therapy was suggested.

The patient underwent HBO therapy at 2.0 Barr for 60 minutes once a day for 7 consecutive days. He responded to the calling of his name on the 5th day of HBO therapy. Mental status improved on the 3rd day after completion of the HBO therapy course, including clear conscious ness, good motor behavior, happy facial expression and verbal expression. At the same time, speech, lan guage and cognitive functions recovered grad ually. He was able to recognize his parents, and had retained memory of his teachers’ names. In addition, he was able to perform simple calculations (e.g.: $1 + 1 = 2$), and put together meaningful sentences, but not to write. The function of motor system recovered on the 4th day after HBO therapy. He could sit with out support and walk on toes and heels. How ever, poor corticospinal atrophy (dysmetria and dystonia) and truncal ataxia were still noted.

Two weeks following admission, follow-up Tc-99m HMPAO brain scan revealed improved perfusion in the right temporal area. Intellectual quotient testing [Wechsler Intelligence Scale for Children-Revised (WISC-R)] revealed VIQ 90 and PIQ 45. The low PIQ score might be due to truncal ataxia.

At three weeks follow-up at our out-patient clinic, neurological examinations revealed intact cranial nerves, good motor system function and good memory. Finger-nose-finger test showed mild tremor, but no dysmetria. Writ ing ability had improved greatly. Two months later, follow-up brain MRI revealed bilateral globus pallidus necrosis with surrounding gliosis change, with decrease in size of focal hyperintensity area (Fig. 1B).

**Discussion**

Carbon monoxide (CO) is highly toxic to humans because it has an affinity for hemoglobin 200-250 times greater than that of oxygen. It crosses the blood-brain barrier from the lungs to the tissues. The clinical manifestations of CO intoxication can be classified into mild (headache, nausea, vomiting and dizziness), moderate (chest pain, blurred vision, weakness, ataxia and cognitive deficits) and severe (palpitations, confusion, seizures, coma, hypotension, pulmonary edema and arrhythmias). Neurologic symptoms may be caused by demyelination of the subcortical white matter.
The most serious complications of CO intoxication are delayed neurologic sequelae, such as dementia, amnestic syn drome, psycho sis, parkinsonism, cortical blindness, apraxia, agnosias, and peripheral neuropathy. Incontinence and chorea.

De layed neu r o psy chi at ric sequelae may be pre ceded by a lucid peri o d of 2-40 days after initial intoxication. The in cidence of neu ro psy chi at ric sequelae is 14-40% of dis charged pa tients. Up to 75% of pa tients recover within 12-18 months. Myers et al. found that 38.5% (82/213) patients received normobaric oxygen therapy in acute CO intoxication, but 12.1% (10/82) patients developed delayed neurologic sequelae. All ten patients under went HBO therapy which re sulted in dama mic im provement. At 6-12 months follow-up, all of them showed asymp tomatic.

The mech a nism of de layed neu r o psy chi at ric sequelae following CO intox i ca tion is un known. A study showed that brief peri ods of brain ischemia can re sult in vul ner able neu ron necrosis and de layed neuronal death due to pre vi ous cellu lar hy per ac tiv ity. Risk factors for these kinds of com pli cations in clude young age, old age, du ration of un con scious ness on acute in tox i ca tion, pre-existing cardio vas cu lar or pul mo nary dis ease, and preg nancy.

How does hyperbaric oxygen (HBO) work in treating CO intoxication? HBO en hances the de cay of CO. The half life of CO is 4-6 hours, 20 minutes in room air, 100% oxygen and HBO (2.5atm), respectively. In ad di tion to re ducing the half life of CO, HBO can re duce brain edema and pre vent lipid per oxidation af ter loss of con scious ness from CO intox i ca tion. A literature re view re vealed that 12-43% of pa tients treated with 100% oxygen have persistent neu ro logic symp toms, as com pared with 0-4% for those treated with HBO. Indica tions for HBO ther apy in clude syn cope, hemodynamic change, neu rologic impair ment, and persist ent neu rologic find ings af ter ox y gen ther apy. In the past, COHb level was cons idered an in di ca tor for HBO ther apy, but now it is not com monly used.

How HBO therapy im proves de layed neu r o psy chi at ric sequelae of CO intox i ca tion in the pres ence of recovered COHb level remains un known. Delayed neu ro logic sequelae are caused by ce re bral hypoxi-


