CARBON MONOXIDE INTOXICATION IN CIGAR SMOKERS

By W. HAMILL, M.D. and R. P. O'NEILL, M.D.

University of Kentucky, Department of Medicine, Lexicon, Kentucky.

Introduction

CARBON monoxide intoxication, most frequently resulting from exposures in industry, poorly vented homes and suicidal attempts, is a relatively common clinical entity. Although cigarettes are known to produce significant amounts of carbon monoxide\textsuperscript{1-4}, the relationship between smoking and carbon monoxide intoxication has received little clinical emphasis. Cigar smoke contains comparable or greater amounts of carbon monoxide; however, since the smoke usually is not inhaled, less carbon monoxide is absorbed. Many reports appear in the literature concerning the clinical\textsuperscript{1}\textsuperscript{4}, experimental\textsuperscript{14,18} and biochemical\textsuperscript{15,23} effects of carbon monoxide, yet polycythemia in man secondary to arterial oxygen desaturation from smoking has to our knowledge not been reported.

The following cases relate chronic carbon monoxide intoxication from cigar smoking with polycythemia and decreased arterial oxygen saturation.

Case Report

Case 1: R. P., a 47 year old white male, was admitted to the Ohio State University Hospital, in September, 1964, for evaluation of polycythemia with decreased arterial oxygen saturation. He had been a heavy cigarette smoker from age 14 until three years prior to admission, thereafter smoking only commercial cigars, progressing to 40 to 50 per day and inhaling deeply. He had dyspnea on mild exertion, but had no other symptoms of congestive heart failure. For several years he had a morning cough productive of small amounts of non-purulent sputum. During his work as a welder he had found that breathing 100 per cent oxygen for a short period would improve many of his symptoms. Since December, 1963, he noted staggering when walking (without lateralization), fine tremor of his hands after exertion or on attempting precise movements, and bifrontal throbbing headaches.

In June, 1964, he was admitted to his local hospital with a history of seven months of nervousness, anorexia, shortness of breath and staggering gait. His evaluation revealed: a moderate plethora, a blood pressure of 120/90 mm. Hg. and a fine rhythmic constant tremor of the hands. The remainder of the examinations was within normal limits with no other signs of cardiac, pulmonary or neurological disease. Laboratory data included a hemoglobin of 19.2 gm.%, a hematocrit of 57 per cent and a normal WBC, differential, urinalysis, BSP, serology, FBS, BUN, thyroxine turbidity, PBI, and spinal fluid dynamics and chemistries. Blood volume determinations (RISA) revealed a plasma volume of 2.65 litres, a RBC mass of 2.45 liters and a total blood volume of 5.1 litres normals: 3.1, 2.1 and 5.2 respectively). The arterial oxygen saturation was 70 per cent (capacity of 26.3 Vol. % calculated from the total hemoglobin, content of 18.4 Vol. % determined with the Natelson microgasometer\textsuperscript{24, 25}). The arterial pH was 7.39, the pCO\textsubscript{2}, 35.9 mm. Hg., the HCO\textsubscript{3}, 20.9 mEq/liter, and the carbon dioxide, 22.0 mEq/liter. Repeat studies five days later, during which time his tobacco consumption decreased, revealed an arterial oxygen saturation of 82 per cent (capacity of 24.0 Vol. % calculated from the total hemoglobin, content of 19.6 Vol. %). The electrocardiogram showed a complete right bundle branch block. Pulmonary function studies included a normal vital capacity and maximum expiratory flow rate. The chest x-ray was normal, as were the pulmonary arteriograms which were of good quality. His discharge medications were chloralothiazepoxide 10 mg. q.i.d., thiamine 25 mg. t.i.d. and multivitamin q.d., and he was advised to discontinue smoking. His subsequent tobacco consumption was reduced to 8 to 10 cigars per day. His symptoms improved progressively.

At the time of admission to Ohio State University Hospital, he had only moderate dyspnea on exertion with no tremor or ataxia. Physical examination revealed a well...
developed, muscular white male in no distress with a blood pressure of 130 80 mm.
Hg. in both arms supine, a pulse of 80/minute and regular and respiration of 20/
minute. The skin was warm with good turgor and slight facial plethora. Eyes, ears,
nose and throat were unremarkable. The chest was symmetrical with equal ex-
pansion but slightly prolonged expiration. Scattered rhonchi were present bilaterally
that cleared partially with coughing. The heart was not enlarged and the sounds were
of good quality in sinus rhythm. No murmurs or adventitious sounds were present.
The abdomen was soft with no hepatosplenomegalgy or masses. Equivocal softening
of the nail beds was noted suggestive of early clubbing. The remainder of the exam-
ination including the neurologic was within normal limits. The laboratory data
included a hemoglobin of 16.7 gm. per cent. a hematocrit of 50 per cent. normal
WBC, sedimentation rate, platelet count, bilirubin, uric acid, electrolytes, creatine
and alkaline phosphatase. The electrocardiogram showed complete right bundle
branch block unchanged from his previous tracing. The arterial oxygen saturation
was 96 per cent (capacity of 22.7 vol. %, content of 21.8 vol. %, determined spectro-
photometrically) with a pH of 33.3 mm. Hg. The blood gases were normal. Pulmonary
function studies revealed a normal maximum breathing capacity, vital capacity, maximum expiratory flow rate. These were felt to be com-
patible with very minimal obstructive lung disease. At cardiac catheterization, right
heart pressures, saturations and cardiac outputs were normal at rest and with exer-
cise. No evidence of a shunt was found with repeated indocyanine green injections.

Following the above studies, on request he increased his cigar consumption to
approximately 20 cigars per day. During this time his blood carboxyhemoglobin
became elevated to 13%, and 15% on two occasions. While at this level he de-
veloped bifrontal headaches and slight tremulousness. However, no neurologic
deficits could be demonstrated.

Case II: F.W., a 46 year old white male was referred to the Lovelace Clinic,
Albuquerque, New Mexico, for evaluation of polycythemia, headaches and dizziness
of one year's duration. For many years he had smoked two to three packs of cig-
arettles daily until three months prior to referral, thereafter smoking only cigars
(10 to 15 per day) and inhaling deeply as had been his custom with cigarettes. Three
weeks prior to his initial evaluation he consulted his local physician with the above
symptoms. His hematocrit at that time was reported to be 53 per cent (no other
laboratory data are available) and a phlebotomy of 950 cc. was done. Two weeks
later a second 500 cc. of blood was removed. Neither phlebotomy improved his
symptoms. Physical examination at the time of his initial evaluation was unreveal-
ing except for prolongation of the expiratory phase of expiration. Laboratory data
included: a hemoglobin of 13.9 gm.%, a hematocrit of 42 per cent, a normal WBC,
differential, platelet count, blood volume and electrocardiogram. Chest fluoroscopy
showed evidence of emphysema and mild fibrotic changes. Pulmonary function
studies revealed moderate obstructive impairment with improvement after broncho-
dilator (Isoproterenol aerosol) administration.

Using the gastrometric method, the arterial oxygen content was 15.4 Vol. % and
oxxygen capacity 17.1 vol. %, thus an oxygen saturation of 90 per cent. However,
when the same content was related to the total capacity (18.9 vol. % calculated from
the hemoglobin concentration and 13.9 x 1.36 = 18.9 vol. %) the saturation was only
81 per cent. Following the administration of 100 per cent oxygen, the arterial oxygen
tension was 422 mm. Hg. At this tension the oxygen saturation was 92 per cent
using the total hemoglobin concentration for capacity but 100 per cent plus .3 vol.
%, using the gastometric method. This discrepancy suggested that part of the hemo-
globin was inactive as far as oxygen transport was concerned. Subsequent measure-
ments of alveolar carbon monoxide concentration showed an average of 19 ppm
which corresponds to a carboxyhemoglobin concentration of 12-13 per cent.

Following one week of abstinence from cigars his subsequent symptoms had
almost completely disappeared and his alveolar carbon monoxide was 10 ppm, which
is within the normal range. Further arterial gas studies were not done.

Discussion

In these cases carbon monoxide intoxication from cigar smoking is sug-
gested by the temporal relationship between the clinical and laboratory
findings and excessive smoking. In Case I the reversal of all clinical and
laboratory findings except the electrocardiographic abnormality after ces-
sation of smoking and the ability to markedly elevate blood carboxyhemoglo-
bin with increased cigar smoking tend to corroborate this relationship.
In Case II, after cessation of cigar smoking, the alveolar carbon monoxide