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Carboxyhemoglobin: Hemodynamic and Respiratory Responses to Small Concentrations

Abstract. *Hemodynamic and respiratory measurements were made on humans before and after inhalation of sufficient carbon monoxide to raise the carboxyhemoglobin to between 5 and 10 percent of saturation. Arterial and mixed-venous oxygen tensions decreased on average 7.3 and 13.3 percent, respectively. One of five subjects developed evidence of mild left-ventricular dysfunction.*

The recent report on smoking by the Surgeon General (*1*) summarized the known effects of smoking on the cardiovascular system. Most investigators have attributed these effects to the action of nicotine and miscellaneous coal tars, although certain observations are not thus conveniently explained. For example, the significant increase in the oxygen debt during exercise, observed in smokers (*2*), cannot be attributed to these agents. It is remarkable that carbon monoxide, a known toxicant constituting 4.2 percent of tobacco smoke (*1*), has not been considered as an explanation of the diverse effects of tobacco smoke on the circulation. Reluctance to indict carbon monoxide has probably derived from Hanson and Hastings's observations that saturation of carboxyhemoglobin in smokers ranged from 3 to 4 percent (*3*) and from Haldane's observations that physiologic effects did not occur until carboxyhemoglobin reached 20 to 30 percent of saturation (*4*). Recent evidence suggests that both these observations may be inaccurate (*5, 6*).

A sensitive and rapid gas-chromatographic method for the measurement of carbon monoxide in blood (*6*) has permitted extensive study of the concentration of carboxyhemoglobin in the blood of smokers. We have found the carbon monoxide content of blood in 28 normal nonsmokers to range from 0.01 to 0.36 percent by volume (average saturation, 0.9 percent). In 25 smokers, carbon monoxide content ranged from 0.15 to 2.39 percent (average saturation, 4.2 percent) and was related to the amount of tobacco smoked. However, smaller individuals, and those whose blood showed lower hematocrits, showed higher contents of carboxyhemoglobin in their blood after similar inhalation of tobacco smoke; in one heavy smoker the value was 17 percent of saturation.

Cardiorespiratory responses to small amounts of carbon monoxide were determined by transvenous catheterization of the heart. A cardiac catheter was positioned in the main pulmonary artery to measure intracardiac pressures and to sample mixed venous blood; arterial blood was obtained by Cournand needle from a brachial artery. Oxygen tensions of the bloods were measured (*7*) before and 5 to 7 minutes after inhalation of carbon monoxide at 0.4 percent in air.

Table 1 shows that oxygen tensions of arterial and mixed-venous bloods decreased on average 7.3 and 13.3 percent, respectively, when the carboxyhemoglobin rose to between 4.95 and 9.69 percent of saturation. Neither cardiac output, oxygen consumption, nor body-surface ventilation per minute changed consistently, but the difference in oxygen pressure in arterial and venous bloods, which reflects extraction of oxygen by tissue, increased in all five subjects. Intracardiac pressures did not change in four of the subjects; in the individual that received the greatest amount of carbon monoxide, left atrial pressure rose and cardiac output fell, indicating development of abnormal left ventricular function.

These studies demonstrate that small amounts of carboxyhemoglobin decrease oxygen tension in both arterial and mixed-venous bloods. Oxygen tension of arterial blood is a function of the oxygen concentration in the inspired air, the resistance to diffusion of oxygen imposed by alveolar membrane and red blood cells, and the shunting of venous blood directly to systemic arteries. Such shunts occur through collapsed or inadequately ventilated alveoli and through anatomic pulmonary arteriovenous comunications (*7*). The concentration of oxygen in inspired air was unchanged after the inhalation of carbon monoxide, and no evidence suggested an increase in venoarterial shunting. Two explanations may be advanced: (i) decrease in the capacity of the blood to carry oxygen can be shown to magnify the effect of physiologic venoarterial shunting, and (ii) carboxyhemoglobin containing red cells may impose abnormal resistance to diffusion of oxygen. Roughton and Forster have stressed the importance of oxygen distribution within the blood (*8*); we postulate that binding of heme groups with carbon monoxide may further hinder distribution of oxygen.

The partial pressure of oxygen in venous blood, an index of maximum oxygen tension in tissue, decreased to a greater degree than arterial oxygen tension because of changes in the hemoglobin dissociation curve. Haldane demonstrated in 1912 that the partial combination of hemoglobin with carbon monoxide makes the remaining hemoglobin bind oxygen with abnormal tenacity (*9*). Thus, unloading of oxygen results only from exposure to lower oxygen tensions. This effect may be particularly harmful in a vascular bed,

Table 1. Hemodynamic and respiratory responses of five normal subjects to carboxyhemoglobin. The first of each pair of lines shows values before the breathing of CO at 0.4 percent in air; the second, values after breathing. COHb, carboxyhemoglobin; sat, saturation; LA, left artium; PA, pulmonary artery; Ar, arterial; t_{O_2}, oxygen tension; Ven, mixed venous; Ar-ven diff, arterial-venous difference; Vent, ventilation per square meter of body-surface area per minute; t_{CO_2}, carbon dioxide tension.

COHb (% sat)	Pressure (mm-Hg)				Ar-ven diff (% by vol.)	Cardiac output (lit./min)	Vent (liter)	t_{CO_2} (mm-Hg)
	LA (wedge)	PA (mean)	Ar t_{O_2}	Ven t_{O_2}				
0.48	28	9	89	45	3.40	5.23	4.23	34
8.84	28	9	81	42	3.82	4.46	4.23	36
			86	37	3.96	4.37	4.68	36
6.29			80	30	4.55	4.35	5.72	36
	3	14	74	42	3.92	4.31	2.55	36
	3	12	68	37	4.24	4.17	3.11	40
0.37	9	13	84	49	4.00	5.32	5.43	39
4.95	9	13	79	42	4.66	6.54	7.36	38
0.96	7	12	77	41	4.02	6.00	4.87	36
9.69	11	18	72	35	4.81	4.68	4.24	39

such as the coronary circulation, which normally extracts oxygen at low oxygen tensions. An amount of carboxyhemoglobin of 5- to 10-percent saturation, which could occur in heavy smokers, could lead during exercise to severe myocardial hypoxia in patients with coronary-artery disease. Myocardial oxygen is limited by flow, in that increased requirements of oxygen are normally met by increase in coronary flow rather than in the amount of oxygen extracted. Diseased coronary arteries cannot significantly increase flow, and hypoxia can be prevented only by increased extraction. Thus even small amounts of carboxyhemoglobin may hinder unloading of oxygen and produce hypoxia in tissue.

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Pox Pottery: Earliest Identified Mexican Ceramic

Abstract. *The earliest known ceramics from Mexico, termed "Pox Pottery," may mark the transition from a nomadic to a settled way of life. The presence of "Pox Pottery" in both coastal Guerrero and the Tehuacan Valley might provide evidence as to the type of environment in which this change first occurred.*

During the spring of 1960, my wife and I excavated two pits along the Guerrero coast at Puerto Marquez and Zanja, and found samples of the earliest ceramics yet uncovered in Mexico.

Puerto Marquez Bay is immediately to the south of Acapulco, and the modern village of Puerto Marquez is situated at the center of the bay on a narrow bar which separates the Pacific from mangrove swamp. The southern side of the bay is formed by a short mountainous peninsula rising from these swamps. It is possible to travel by canoe from the town of Puerto Marquez through the mangroves behind this peninsula to a sand bar fronting on open ocean. The archeological site is next to a fresh-water stream on a northward-facing cove of this promontory and consists of a poorly defined mound stretching back from the beach. Much of the site is now covered by the residence of Ing. Adolfo Orive Alba.

Zanja is located on a narrow branch of a large lagoon about 8 km to the southeast of Puerto Marquez and 5 km inland from the Pacific. Part of the single, low mound forming this site has been eroded by the lagoon so that quantities of potsherds and shells extending downward below the water can be seen. The site is distinguished from the immediately surrounding swamp and essentially barren sandy patches by the lush grove of coconut palms it supports. Today there are no permanent habitations on the mound, although temporary camps are frequently established there by people wanting to exploit the resources of the lagoon. Flooding forced us to abandon our excavation at Zanja after we had reached a depth of 4.60 m but before we had reached sterile soil.

At Puerto Marquez, Ing. Orive kindly granted us permission to dig a pit behind his house. No obvious stratification was present, so we excavated arbitrary layers, each 20 cm thick. The layers were numbered from top down until sterile soil was reached after 7.60 m at layer 38. The first 33 layers of refuse contained abundant pottery. The deepest meter (layers 34 to 38) produced no potsherds, and the cultural inventory was confined to little chunks of obsidian, some retouched flint flakes, and a single small flint core. When the age of material from the bottom of the pit (layer 38) was determined by carbon-14 dating techniques (*1*), a date of 2940 ± 130 B.C. was obtained; material from layer 33, the first ceramic-bearing layer, gave a date of 2440 ± 140 B.C. These tests indicate that the area was occupied for 500 years before pottery was manufactured. Starting with layer 29, sherds carrying distinct resemblances to ceramics from the earliest known Formative cultures were encountered (*2*). Below layer 29, many of the sherds were dissimilar to any ceramics from Mexico for which descriptions have been published. Fragments essentially identical to these were recovered from the deepest levels at Zanja. I have termed these sherds "Pox Pottery."

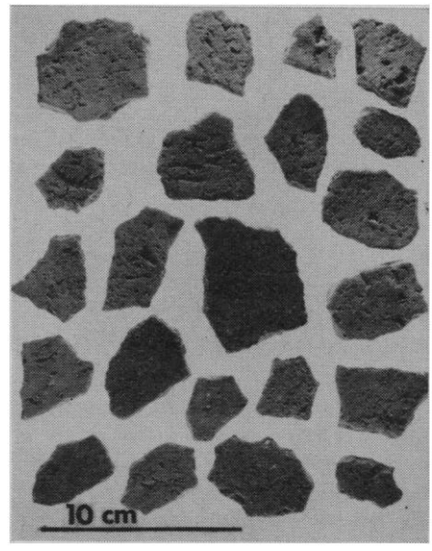

Fig. 1. Examples of Pox Pottery sherds from Guerrero, showing concave surfaces.

The diagnostic feature of Pox Pottery is the rough and pitted appearance of the inside or concave surface of the sherd (Fig. 1). The pitting was undoubtedly produced by roughly brushing or wiping the interior of the vessel after the clay had dried sufficiently to have lost most of its plasticity. This caused the irregularly sized particles of temper to be dragged along and away from the clay, leaving numerous deep gouges and crater-like indentations. This crude interior contrasts with the well-smoothed, occasionally red-slipped exterior or convex surface of these fragments. Pox Pottery is quite friable, and the generally small sizes of the sherds make shape determinations difficult. Where these could be made, the forms were either sharply incurving neckless pots or vessels with high straight necks. Similar shapes occur during the succeeding period at Puerto Marquez and Zanja, at other early Formative sites in Mesoamerica (*2*), and among the earliest ceramics from Tehuacan (*3*).