animals. The earlier age at death of exposed animals was considered compatible with a process of accelerated aging, possibly resulting from the stress of such exposure.

It is becoming increasingly evident that oxides of sulfur, in concentrations attainable in community air, may affect the human respiratory tract. A research team at the Harvard School of Public Health has shown that the acute response in human beings resembles that in guinea pigs. Normal persons who inhaled either sulfuric acid mist or sulfur dioxide for brief periods exhibited markedly shallower, more rapid breathing (*3,4*). More recently another team of investigators at Harvard measured pulmonary function in healthy volunteers exposed to controlled levels of sulfur dioxide (*5*). During administration of the gas, all measurements of resistance showed an increase, greatest for pulmonary flow resistance (PFR) on quiet breathing, intermediate for PFR on panting and for airway resistance, and smallest for total respiratory resistance. Pulmonary flow resistance showed no change at 1 to 2 ppm of sulfur dioxide; it increased an average of 19 percent above control levels at 4 to 5 ppm and 49 percent at 8 to 19 ppm; and when sulfur dioxide was combined with aerosol, the increase was 72 percent. However, investigators at St. Luke's Hospital in Cleveland observed no changes in resistance in normal subjects exposed briefly to sulfur dioxide in concentrations of 2.5 to 23 ppm, combined with particulates and aerosols, where emphysematous subjects exhibited a decrease in airway resistance (*5*).

Although the acute effects of exposure to high concentrations of carbon monoxide are well documented, the chronic effects from long-term subtoxic doses are controversial. Recent findings suggest that, besides its known effects upon hemoglobin, carbon monoxide exposure may affect the eye and nervous system adversely. Since 1955, carbon monoxide levels in the Los Angeles atmosphere have been increasing by about 1 ppm (0.0012 mg./m.^{3}) per year. It is estimated that gasoline engine exhaust is the source of about 75 percent of the total carbon monoxide content of Los Angeles air, with significant contributions also from metallurgic and oil-refining operations.

Research workers (*6, 7*) have found an average blood carboxyhemoglobin of 3.8 percent, not markedly different from average levels in groups with lesser degrees of exposure, in subjects exposed to carbon monoxide in their working environment, from smoking, or while commuting to work in private automobiles. The carbon monoxide concentration in a garage and automobile inspection center where the exposed group worked ranged from 10 to 150 ppm, (0.06 mg./m.^{3}); in the working environment of the control group, the ambient carbon monoxide level was less than 10 ppm (0.012 mg./m.^{3}). Although 17 of 68 exposed subjects, compared with 3 of 25 controls, complained of headache, dizziness, or unusual fatigue at the end of the workday, no relationship could be found between carboxyhemoglobin levels and occurrence of those symptoms.

In a preliminary study performed by Public Health Service scientists at Cincinnati, Ohio (*8*), the *levels of carbon monoxide in the passenger compartment of stationery vehicles in heavy traffic were greatly increased*, reaching a maximum of 370 ppm (0.44 mg./m.^{3}). Investigators at the University of Michigan (*9a, 10*) sought to determine whether atmospheric carbon monoxide levels in urban areas might interfere with the driver's ability to operate his vehicle. Data collected from appropriate sites in Detroit for 1 year showed that median daily values of atmospheric carbon monoxide ranged between 0 and 20 ppm. During periods of high atmospheric stability and heavy traffic, concentrations reached 100 ppm at some sampling sites and persisted at this level for several hours. A later study on Los Angeles freeways showed significant build-up of carbon monoxide in drivers in rush-hour traffic to dangerous levels with respect to driving judgment. In homes several hundred feet from street sampling sites, concentrations approximated those in the street. Analysis of reports of more than 4,000 consecutive accidents involving almost 5,000 persons failed to reveal a higher accident rate associated with occupations in which high carbon monoxide exposure would be expected. In an attempt to relate the carbon monoxide content of the blood to air levels, a cigar smoker and a nonsmoker traveled in a police scout car for 8 hours for a distance of 130 miles. The carbon monoxide in the vehicular air, monitored continuously—reflecting outside traffic conditions and not influenced by any tobacco smoke in the car—averaged 17 ppm with a peak of 120 ppm when the engine was idling. The smoker's loaded carboxyhemoglobin rose from 3.4 to 3.9 percent, the nonsmoker's from 0.8 to 1.2 percent.

In a study of 237 persons involved in traffic accidents (including both drivers and pedestrians) and brought to the hospital for treatment, 50 percent of the drivers had less than 3 percent carboxyhemoglobin, 50 percent of the pedestrians had less than 2 percent, and only 3 persons had levels of 10 percent or more; in