Although automobile exhaust contains dozens of compounds, only a limited number of these are a potential public health hazard.

Health Hazards of Automobile Exhaust

JOHN R. GOLDSMITH, M.D., M.P.H., and LEWIS H. ROGERS, Ph.D.

THE GASES, vapors, and particles in automobile exhaust are considered by many to be the major contributing factors of Los Angeles' air pollution, which is so strikingly characterized by irritation of the eyes (*1,2*). Other west coast cities, including San Diego and San Francisco, appear to have a similar problem in less severe form. We have learned that when the nitrogen oxides and partly burned fuel of automobile exhaust are irradiated the characteristic photochemical type of smog is produced (*3*). Concern regarding this form of air pollution derives both from the widespread symptoms and from the possibility of immediate or long-term effects on health (*4*). If attention is focused on hydrocarbons and nitrogen oxides, in an effort to abate eye irritation, other adverse effects of automobile exhaust may unfortunately be ignored. This review attempts to place these other effects in perspective.

When only a few automobiles are driven on country roads, there is no public health problem, but when thousands of cars are operated in a small area with a limited supply of fresh air, automobile exhaust may constitute a potential hazard to the health of the community. This will be true even in the absence of irritated eyes and reduced visibility, so characteristic of photochemical smog.

Dr. Goldsmith is head of studies of health effects of air pollution, California State Department of Public Health, Berkeley, and Dr. Rogers is associate director, Vitro Laboratories, West Orange, N.J., and formerly senior chemist, Air Pollution Foundation, San Marino, Calif.

The solution of two imperative questions lies in the future: How much exhaust can be tolerated in how much air? When does this potential hazard become an actual one? For the present, we shall concern ourselves with what is known about the constituents of automobile exhaust and with an approach to answering these questions.

Composition of Automobile Exhaust

We are confining our attention to air pollution from spark-fired internal combustion engines and excluding that from diesel engines. In most urban areas, the latter, although sometimes a local nuisance, is of lesser importance to the community. In Los Angeles, for example, the air pollution from diesel engine exhaust is about 20 tons of organic emissions per day, whereas auto exhaust contributes about 1,200 tons of organic emissions per day (*5*).

When a petroleum fuel is completely burned, the products are carbon dioxide and water, together with nitrogen and unused oxygen from the air. However, in a spark-fired internal combustion engine some of the nitrogen is also oxidized producing the several oxides of nitrogen. In automobiles the ratio of air to fuel is seldom maintained at the theoretical value of 15:1 required for complete combustion. More frequently, this ratio amounts to about 12:1 at idling and 13.5:1 at cruising.

Under these conditions, other products appear in the exhaust, including carbon monoxide, hydrogen, aldehydes, and unburned hydrocarbons. In addition, oxides of sulfur occur in

exhaust depending on the amount of sulfur in the fuel, and lead compounds are present in a form determined by the additives used in the fuel. Because of tremendous differences in operating conditions, condition of cars, and other factors, it is difficult to give representative analyses. Some characteristic ranges of exhaust gas composition are presented in table 1. Of the constituents listed in table 1, we shall consider only hydrocarbons, oxides of nitrogen, lead compounds, and carbon monoxide.

Effects of Time and Weather

Since the composition of exhaust gas is so variable and is quickly diluted several hundredfold when it is released to the atmosphere, knowing the actual concentrations of exhausted gases in the atmosphere is important. These concentrations are affected by windspeed, presence and intensity of a temperature inversion, vehicle operating conditions, number of automobiles operating per square mile, and further reactions of the atmospheric constituents. To illustrate how high the concentrations may become, table 2 shows values of hydrocarbons and carbon monoxide at five busy intersections in Los Angeles during peak traffic and adverse weather conditions.

About 5,000 vehicles per hour passed the intersections at which the samples were taken except at Hollywood and Harbor Freeways, where about 11,000 per hour passed.

Community and Industrial Exposure Hazards

The substances listed in table 1 have been subject to conventional toxicological studies, especially from the viewpoint of occupational hazards (*9–11*). Although the toxicological procedures are well established there are several reasons why this approach is inadequate for our purposes.

Industrial exposure standards are usually determined by the American Conference of Governmental Industrial Hygienists. These standards, called "threshold limits," are based upon exposures of healthy adults to a single substance for a period not to exceed 8 hours followed by a recovery period of at least twice that long. In community air pollution from automobile exhaust, concentrations vary from hour to hour, but the exposure may be continuous, both in time and, for most persons, in place. Coming home from work does not mean an end of exposure to air pollution and may mean a great increase if the trip requires driving during peak traffic.

The extent to which exposure to one or more of the substances in automobile exhaust increases the effect of another is not known. Concern over this possibility is heightened by some examples of synergistic effects. Amdur (*12*) has shown the extent to which aerosols enhance the effects of SO_2 on the pulmonary airway flow resistance in guinea pigs, and Falk (*13*) has shown the effect of soots on deposition of carcinogens. Until further studies have clarified such interactions, it is necessary to suspect that components of exhaust may have synergistic effects.

While industrial exposures usually involve small numbers of relatively healthy people, community exposures affect, in varying ways, the entire population of a community, the sick with the well, the frail with the vigorous. In sufficient concentrations, exhaust from automobiles may lead to morbidity or even mortality in the sick and frail segment of the population whereas the same exposure might not noticeably affect healthy and vigorous persons. For persons with circulatory failure or with reduced competence of cerebral or myocardial circula-

Table 1. Composition of automobile exhaust

Constituent	Percent of concentration (volume/volume)	
	Minimum	Maximum
Aldehydes	0	0.03
Carbon dioxide	5	15
Carbon monoxide	0.2	12
Hydrocarbons	0.01	2
Hydrogen	0	4
Lead compounds	[1]	[1]
Nitrogen	78	85
Oxides of nitrogen	0	0.4
Oxygen	0	4
Sulfur dioxide	[2]	[2]
Water vapor	5	15

[1] Depends on lead additives.
[2] Depends on sulfur content of fuel.

SOURCE: References 6 and 7.

Table 2. Carbon monoxide and hydrocarbons in Los Angeles, Calif., near traffic arteries for days with low-level thermal inversion conditions,[1] September 1956 [2]

Street intersection	Carbon monoxide (ppm)		Hydrocarbons (ppm)		Number of samples	Days of sampling
	Maximum	Mean	Maximum	Mean		
Slauson at Figueroc	93. 2	30. 2	2. 33	1. 32	54	3
La Brea at Olympic	65. 2	34. 4	1. 74	1. 10	18	1
San Fernando at Highland	41. 7	15. 4	1. 56	. 76	36	2
Hollywood and Harbor Freeways	64. 1	36. 0	1. 83	1. 24	36	2
Vineland and Ventura	48. 5	30. 8	1. 50	. 94	36	2

[1] Low-level thermal inversion conditions refer to that condition in the atmosphere when the upper level of air over the earth, instead of being colder than the surface air, is warmer, thereby trapping the air beneath it.

[2] Average of 6 samples taken every 30 minutes between 6:00 and 8:30 a.m., Pacific Daylight Saving Time, at 3 distances from the curb.

SOURCE: Reference 8.

tion, slight impairment of the oxygen transport function of the blood may have serious consequences. In addition to these people, two other groups are at unusual risk from communitywide exposure to auto exhaust. They are the workmen who experience a similar type of exposure while employed, and persons who inhale tobacco smoke. If a member of these multiple exposure groups is also ill, or has impaired health, then the hazard may be compounded.

Finally, we cannot assume that termination of exposure terminates risk of ill effects. In this connection, data on lung cancer reported in Eastcott's study of emigrants from Great Britain to New Zealand are revealing (*14*). He found that the duration of exposure to urban air pollution in Britain before emigration was significantly associated with rates of development of lung cancer observed many years later. Since cigarette consumption was similar in Great Britain and New Zealand, the evidence points toward atmospheric pollution as a factor in the causation of the disease.

It will take years to estimate accurately the possible delayed consequences of exposure of large numbers of persons to automobile exhaust fumes, but the possibility of harm exists and is difficult to evaluate by conventional toxicological methods.

Carbon Monoxide

The maximum carbon monoxide concentration reported in table 2 is 93.2 ppm, while the average values were 15 to 36 ppm. Average concentrations of 4 to 20 ppm, with a maximum of 80 ppm, have been reported in British cities during smog (*15*). Similarly, an average of 28.9 ppm carbon monoxide in Detroit during heavy traffic has been reported, with a maximum of 80 ppm (*16*).

More is known about the mechanism of absorption and action of carbon monoxide than about any other noxious substances found in exhaust. This tasteless, odorless, colorless gas is 250 times as firmly bound to hemoglobin as is oxygen. It may be calculated that at equilibrium for every part per million of carbon monoxide reaching the lung, 0.16 percent of the body's hemoglobin is combined with carbon monoxide, and hence inactive. Roughton (*17*) has shown further that in the presence of carbon monoxide-hemoglobin compounds, oxygen is bound more firmly to hemoglobin, thus further impairing oxygen transport.

While no health damage has been attributed to carbon monoxide exposures below 100 ppm, the presumption that such levels inactivate a small amount of hemoglobin is inescapable. Gaensler and his associates (*18*) have shown that urban nonsmokers have a CO level corresponding to saturation of 0.62 to 1.24 percent of hemoglobin, while smokers have 3.1 to 7.8 percent. The public health importance of this lies in the certainty that smoking and exposure to automobile exhaust are so common that a very large number of persons are affected in such a way that up to 8 percent of their hemo-

globin is unavailable for oxygen transfer. Accurate predictions of carbon monoxide-hemoglobin levels from environmental measurements are complicated by the fact that some time is taken to reach equilibrium and this time varies with the activity of the subject.

In studies on the adaptation of eyes to darkness McFarland (*19*) demonstrated an impaired adaptability in older persons which was duplicated in young subjects when breathing gases deficient in oxygen or when exposed to low levels of carbon monoxide. The importance to automobile drivers of visual sensitivity at night is obvious. However, it has not been shown that exposure to automobile exhaust at the levels found on the freeways of a modern city impairs the adaptability of eyes to darkness or alters the exchange of vital respiratory gases, although this is a plausible inference.

Some idea of the possible exposures of drivers is obtained by sampling air in the driver's compartment of motor vehicles, as shown in table 3 (*20*).

Another way of stating the effect of carbon monoxide is that its inactivation of hemoglobin is similar to withdrawing the corresponding amount of blood from circulation. From this viewpoint a concentration of carbon monoxide of 100 ppm, when equilibrated, is associated with inactivation of about one-sixth of the body's circulating hemoglobin, equivalent in volume to about one pint of blood, with corresponding loss for other concentrations of carbon monoxide.

Nitrogen Oxides

During acceleration and cruising, automobiles emit appreciable quantities of nitric oxide. On mixing with oxygen, nitric oxide is oxidized to nitrogen dioxide, so that a mixture of these two oxides is found in the atmosphere. The concentration of nitrogen oxides in the open air may be 1 ppm at times when CO is 50 ppm.

The toxicity of nitrogen dioxide is based on its irritant properties, often delayed in onset. In cases of significant exposure, signs and symptoms of pulmonary edema have been noted hours to days later (*21*). Nitric oxide also forms a stable compound with hemoglobin in vitro, which, if it occurred in vivo, would make the hemoglobin unavailable for transport of carbon dioxide and oxygen.

Nitrogen dioxide on dissolving forms some nitrite ion which is capable of reacting with hemoglobin to yield methemoglobin, also unsuitable for transporting respiratory gases. That this may occur under some circumstances is suggested by a report of methemoglobin levels of 2.3 to 2.6 percent in welders exposed to a mixture of gases including nitrogen oxides (*22*).

It is unlikely that toxic effects would occur solely from the levels of nitrogen oxides found in places with air pollution due to automobile exhaust. But the similar effect of the reaction of hemoglobin with carbon monoxide and with nitrogen oxides lends greater significance to studying the latter.

Lead Compounds

Nearly all gasoline used in automobiles contains lead tetraethyl, up to as much as 3 ml. per gallon. This lead is mostly discharged through the exhaust to the atmosphere, and this fact has caused much concern over the past 25 years as to the possible effects of lead on health. Lead occurs mostly in the particulate form, and a maximum concentration of 16 micrograms per cubic meter has been reported by Cholak and others in Los Angeles with average values of 7 micrograms per cubic meter (*23*) during a 4-month period, August–November 1954.

In general, analyses of urban air show lead concentrations which are low in relation to the amount of lead burned in gasoline. This may

Table 3. Levels of carbon monoxide in parts per million found in compartments of drivers of motor vehicles in California

Parts per million	Number of vehicles	Percent of vehicles
0–49	1, 014	91. 8
50–99	69	6. 2
100–149	13	1. 2
150–199	5	. 4
200–249	1	. 1
250–299	2	. 2
600–649	1	. 1
Total	1, 105	100. 0

be explained by the fact that the particle size of the exhausted lead ranges from 0.01 micron to several millimeters in diameter, and the large particles can be expected to settle rapidly when exhausted into open air. Moreover, high-speed driving tends to increase the number of heavy particles, and also tends to clean out the exhaust system of lead previously deposited during the light duty, stop-and-go driving conditions (*24*).

Estimating the retention of lead in the body is complicated by the problem of particle size and composition. Generally speaking the smaller (submicron) particles impinge on the deeper portions of the lung, where they may be rapidly absorbed, while larger particles are more likely to impinge on the mucous layer of the upper portion of the airway and subsequently be swallowed. Such a route is associated with loss of most of the lead in the feces.

One feature of the toxicology of lead indicates that its effect might be related to that of carbon monoxide and of nitric oxides. In chronic lead poisoning, there is found a low-grade anemia and also increased fragility of red blood cells; this may tend indirectly to impair transportation of respiratory gases.

When considered apart, lead exposure from observed levels in Los Angeles and other cities is not toxic, but lead exposures are so common in industry and in garages that the small and persistent increment in exposure associated with auto exhaust may be sufficient to render an otherwise tolerable lead burden a toxic one in a few persons.

Hydrocarbons

Although "hydrocarbons" have been classed as a group in table 2, it is apparent that the group includes many compounds such as paraffins, olefins, aromatics, cycloparaffins, and others. The particular mixture in automobile exhaust depends in part on the fuel used and in part on the way the motor vehicles are operated. The total hydrocarbon concentration in the open air seldom exceeds 2 ppm, hence the concentration of individual compounds will be much less than that. At these low concentrations, only those having known physiological activity are of concern.

One group of hydrocarbons of particular interest includes such compounds as 3,4-benzpyrene which under experimental conditions may produce cancer in animals. This compound, along with others of similar chemical constitution, has been qualitatively identified in automobile exhaust in trace amounts (*25,26*). Although polynuclear hydrocarbons produce tumors when painted on the skin of susceptible animals, their inhalation has resulted in no experimental lung cancers. Experimental tumors can be produced if the lung's mucosa is damaged, for example, by a transfixion suture soaked in the carcinogen or by a hooked capsule containing the substance which is retained in the bronchus of an experimental animal (*27*). From such studies one is led to the hypothesis that carcinogenesis occurs on the basis of damage to the mechanisms protecting the underlying tissues.

The possibility that these trace quantities do in fact have a relation to the occurrence of lung cancer is a debatable point, and one on which additional research is necessary. Moreover, even if 3,4-benzpyrene is a cause of lung cancer, it is present to a much greater extent in coal smoke than in automobile exhaust. Communities in which coal is used as a major fuel could expect to find far more 3,4-benzpyrene arising from coal burning than from combustion of petroleum fuels. For example, Los Angeles (*28*), where no coal is burned, has been found to have 3.3 micrograms of 3,4-benzpyrene per 100 cubic meters of air, while in London (*29*) values up to 47 micrograms per 100 cubic meters have been reported.

Interactions of Exhaust Components

On the west coast, where weather conditions may lead to poor dispersal of automobile exhaust, and where there is an abundance of solar radiation, an interaction of nitrogen oxides and hydrocarbons occurs. This results in the obnoxious mixture which we call photochemical smog. The principal products are ozone and organic intermediates, some of which irritate eyes and damage plants. Ozone sometimes reaches a concentration of 0.5 ppm or more.

It is known that ozone is a highly irritating

substance, and is capable of producing clinical signs of pulmonary irritation. Repeated daily exposures of animals to 1.0 ppm for about a year produced fibrosis of the lung in some species. Recently the hygienic standard for industrial exposure was reduced from 1.0 ppm to 0.1 ppm by the American Conference of Governmental Industrial Hygienists.

Of all the substances mentioned, ozone is the only one in Los Angeles which exceeds the hygienic standard for industrial exposure of 0.1 ppm. However, studies to determine whether respiratory disease or deaths in Los Angeles are more common on days with high air pollution levels have not so far demonstrated such an effect. In a review of the hazards of ozone, Stokinger (*30*) states that "if the response of the human lung to ozone may be assumed to be similar to that of the animals tested in these studies, one might reasonably conclude that no acute effects on human beings would be expected from ozone exposures occurring in Los Angeles-type smog, owing to the often repeated exposures to very low grade ozone concentrations of the order of a few tenths part per million." Nevertheless, the use of pulmonary function tests on large numbers of persons may assist in reaching a conclusion about the effect of ozone or other irritants on respiratory function.

In addition to ozone, reactive organic intermediates, including free radicals, are formed (*31*). Whatever the compounds may be that cause eye irritation, it has been proved that these compounds are produced by irradiation of automobile exhaust.

It remains to be shown, however, whether the same compounds irritate the respiratory tract or whether irritation by inhaled substances plus inhaled carcinogens could produce cancer of the lung. More data are needed on the exact nature of the reactions, the concentration of the products, and their physiological effects.

Discussion

Two important effects of the known constituents of automobile exhaust are (*a*) the conversion of hemoglobin into a relatively stable, inactive form which impairs the efficiency of the blood and circulation in transporting the respiratory gases, oxygen and carbon dioxide, and (*b*) the production of respiratory tract irritation or pulmonary edema, either of which interferes with the transport of gases between the blood and the external atmosphere. The severity of both of these effects on individuals would depend on concentrations, length of exposure, age and vigor of the subjects, and other conditions. Concentrations found in the Los Angeles atmosphere have not been shown to interfere with gas transport mechanisms.

We have described what seems to be a potential hazard. To demonstrate whether an actual hazard exists, it will be necessary to combine measurements of exhaust constituents in the atmosphere with estimates of the impairment they produce. For example, measurements should be made of the proportion of hemoglobin inactivated by carbon monoxide and nitric oxides in large numbers of persons exposed to air polluted by automobile exhaust.

We suggest that hygienic standards for automobile exhaust in a community should be set at levels which will produce no health effects on the most susceptible group of persons in the community, defined in terms of age or health status. Among the groups to be considered would be those ill with impaired cerebral or myocardial circulation or impaired pulmonary function.

It is of interest that the public health problems of automobile exhaust have been recently surveyed in the U.S.S.R. with essentially the same conclusions as were independently reached by us (*32*).

Despite the difficulties, all possible means should be taken to control atmospheric pollution from automobile exhaust. At present it seems unlikely that control devices for automobile exhaust will eliminate all of the potentially harmful substances. Until there is complete control, potential hazards should be recognized and efforts made to assess damage.

Summary

Of the substances which occur in automobile exhaust and their reaction products, hygienic standards have been established for industrial exposure to carbon monoxide, nitrogen dioxide, lead, and ozone. Establishing a full set of levels for community exposures to these sub-

stances is very difficult because of the sensitivity of frail or ill individuals, the indeterminate period of exposure, the effect of agents in combination, and the cumulative effect of exposure from other sources, such as cigarette smoking.

The hazard of automobile exhaust to the population of a large community will depend, among other things, on the extent and way that vehicles are used, and the meteorology of the area.

In the absence of effective control for air pollution from automobile exhaust, the public health hazard should be evaluated.

REFERENCES

(1) Haagen-Smit, A. J.: Progress in smog control. Engin. & Sc. 21: 5–11, June 1958.

(2) Faith, W. L., Renzetti, N. A., and Rogers, L. H.: Automobile exhaust and smog formation. Air Pollution Foundation Report No. 21. San Marino, Calif., October 1957, 103 pp.

(3) Schuck, E. A.: Eye irritation from irradiated auto exhaust. Air Pollution Foundation Report No. 18. San Marino, Calif., March 1957, 85 pp.

(4) California State Department of Public Health: Clean air for California. Report I. Berkeley, March 1955, 57 pp.

(5) Faith, W. L.: Air pollution: What causes smog? Refinery Engin. 28: c–3, c–6, c–8, October 1956.

(6) Sheldrick, L. S.: Exhaust gas composition of internal combustion engines. General Motors Diesel Power Engineering Bulletin No. 24. Detroit, Feb. 10, 1954.

(7) Fitton, A.: Exhaust gases from motor vehicles. Roy. Soc. Promotion Health J. 76: 664–677, October 1956.

(8) Renzetti, N. A.: Analysis of air near heavy traffic arteries. Air Pollution Foundation Report No. 16. San Marino, Calif., December 1956, 28 pp.

(9) Henderson, Y., Haggard, H. W., Teague, M. C., Prince, A. L., and Wunderlich, R. M.: Physiological effects of automobile exhaust gas and standards of ventilation for brief exposure (part 1). J. Indust. Hyg. 3: 79–92, July 1921.

(10) Sayers, R. R., Fieldner, A. C., Yant, W. P., and Thomas, B. G. H.: Experimental studies on the effects of ethyl gasoline and its combustion products. U.S. Bureau of Mines Report. Washington, D.C., 1927, 447 pp.

(11) Kehoe, R. A., Thamann, F., and Cholak, J.: An appraisal of the lead hazards associated with the distribution and use of gasoline containing tetraethyl lead. J. Indust. Hyg. 16: 100–128, March 1934.

(12) Amdur, M. O.: The influence of aerosols upon the respiratory response of guinea pigs to sulfur dioxide. Am. Indust. Hyg. A. Quart. 18: 149–155, June 1957.

(13) Falk, H. L.: Elution of 3,4-benzypyrene and related hydrocarbons from soot by plasma proteins. Science 127: 474–475, Feb. 28, 1958.

(14) Eastcott, D. F.: The epidemiology of lung cancer in New Zealand. Lancet 1: 37–39, Jan. 7, 1956.

(15) Wilkins, E. T.: Exhaust gases from motor vehicles: Some measurements of carbon monoxide in the air of London. Paper presented at the meeting of the Royal Society of Health, London, England, June 13, 1956.

(16) Castrop, V. J., Stephens, J. F., and Patty, F. A.: Comparison of carbon monoxide concentrations in Detroit and Los Angeles. Paper presented at the American Industrial Hygiene Association Annual Meeting, Buffalo, N.Y., April 27, 1955.

(17) Roughton, F. J. W., and Darling, R. C.: The effect of carbon monoxide on the oxyhemoglobin dissociation curve. Am. J. Physiol. 141: 17–31, Mar. 1, 1944.

(18) Gaensler, E. A., and associates: A new method for rapid precise determination of carbon monoxide in blood. J. Lab. & Clin. Med. 49: 945–957, June 1957.

(19) McFarland, R.: Human factors in air transportation. New York, McGraw-Hill, 1953, pp. 301–303, 379–380.

(20) Russell, J. P., Zelk, G. S., and Ingram, F. R: Carbon monoxide survey: A report on the carbon monoxide hazard in relation to highway traffic safety. California Highway Patrol, with supplemental report on the automobile survey. Sacramento, Calif., September 1939, 64 pp.

(21) Lowry, T., and Schuman, L. M.: Silo-fillers disease, a syndrome caused by nitrogen dioxide. J.A.M.A. 162: 153–160, Sept. 15, 1956.

(22) McCord, C. P., Harrold, G. C., and Meek, S. F.: A chemical and physiological investigation of electric arc welding. III. Coated welding rods. J. Indust. Hyg. & Toxicol. 23: 200–215, May 1941.

(23) Cholak, J., Schafer, L. J., Yeager, D. W., and Kehoe, R. A.: The nature of the suspended matter. Air Pollution Foundation Report No. 9. San Marino, Calif., July 1955, pp. 201–225.

(24) Hirschler, D. A., and associates: Particulate lead compounds in automobile exhaust gas. Indust. & Engin. Chem. 49: 1131–1142, July 1957.

(25) Kotin, P., Falk, H., and Thomas, M.: Aromatic hydrocarbons. II. Presence in the particulate phase of gasoline engine exhausts and the carcinogenicity of exhaust extracts. A.M.A. Arch. Indust. Hyg. Occup. Med. 9: 164–177, February 1954.

(26) Lyons, M. J., and Johnston, H.: Aromatic hydrocarbons from vehicular exhausts. Brit. J. Cancer 11: 60–66, March 1957.
(27) Kuschner, M., Laskins, S., Cristofano, E., and Nelson, N.: Experimental carcinoma of the lung. *In* Proceedings of the Third National Cancer Conference. Philadelphia, Lippincott, 1957, p. 485.
(28) Kotin, P., Falk, H., and Thomas, M.: Production of skin tumors in mice with oxidation products of aliphatic hydrocarbons. Cancer 9: 905–909, September-October 1956.
(29) Commins, B. T., Waller, R. E., and Lawther, P. J.: Air pollution in diesel bus garages. Brit. J. Indust. Med. 14: 232–239, October 1957.
(30) Stokinger, H. E.: Evaluation of the hazards of ozone and oxides of nitrogen. A. M. A. Arch. Indust. Health 15: 181–190, March 1957.
(31) Leighton, P. A., and Perkins, W. A.: Photochemical secondary reactions in urban air. Air Pollution Foundation Report No. 24. San Marino, Calif., August 1958, 212 pp.
(32) Nedogibchenko, M. K.: The current state of pollution of the air of cities by motor transport exhaust and the problems of its control. Gig. San. 23: 6–9, August 1958.

Stebbins New Chairman of PHR Board of Editors

Dr. Ernest Lyman Stebbins, dean of the Johns Hopkins University School of Hygiene and Public Health for the past 13 years, is the newly appointed chairman of the Board of Editors of *Public Health Reports*. He succeeds Dr. Edward G. McGavran, dean of the University of North Carolina School of Public Health, who has served as chairman since 1952.

Dr. Stebbins entered the public health field in 1931 as an epidemiologist with the Virginia State Department of Health after completing internship and residencies at Clara Barton Hospital in Los Angeles and Presbyterian Hospital and the University Clinic in Chicago.

In 1934, he transferred to the New York State Department of Health and was appointed New York City Health Commissioner in 1942. In 1945, he received the Lincoln Award for distinguished service to the city. From 1940 to 1946 he was a professor of epidemiology at Columbia University.

He served as a medical director with the Public Health Service during the war and retains status as a reserve officer.

Dr. Stebbins is a fellow of the American Medical Association and the American Public Health Association; 1958–59 president of the Advisory Board of Medical Specialties; chairman, American Board of Preventive Medicine; and former president of the American Epidemiological Society.

Publications by Dr. Stebbins include, in addition to reports of scientific research, "Epidemiology and Social Medicine—Social Medicine: Its Derivations and Objectives," Commonwealth Fund, 1949, and "Introduction to Public Health," written with Dr. Harry S. Mustard.

After being graduated by Dartmouth College in 1926, Dr. Stebbins received his medical degree from the Rush Medical School of the University of Chicago in 1929. His master's degree in public health was earned at Johns Hopkins University School of Hygiene and Public Health in 1931.