Motor Vehicle Emissions and Their Effects

CONFERENCE REPORT

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TWO CONFERENCES held in Los Angeles in December 1961 presented current research findings on quantities and types of air pollutants from motor vehicles and the effects of these pollutants on the health of man, plants, and laboratory animals. The Fifth Air Pollution Medical Research Conference, on December 4, was sponsored by the California State Department of Public Health. The Joint Research Conference on Motor Vehicle Emissions and Their Effects, held December 5-7, was sponsored by the California State Department of Public Health and the Public Health Service's Division of Air Pollution. Highlights of the papers follow. (Boldface numbers in parentheses refer to conference papers listed at the end of the report.)

In North America, photochemical air pollution occurs in parts of Canada, the major cities of 19 States, and Baja California, Mexico (1). Motor vehicles are the major source of this pollution.

Public Health Service investigators (1) are measuring vehicle emissions on parkway, arterial, neighborhood, and business routes in Cincinnati, Chicago, Detroit, Los Angeles, New Orleans, Philadelphia, San Francisco, and Washington. The sampler developed for the study collects a composite exhaust sample that is proportional to exhaust flow during all operations. From the total exhaust 0.25 percent is

collected in a noncontaminating bag and analyzed in the laboratory.

Scott Research Laboratories (2) is identifying diesel exhaust odorants and eye irritants and relating their concentrations to engine operations. Concentrations were presented for particulate, low-molecular-weight hydrocarbons, formaldehyde, acrolein, carbon monoxide, carbon dioxide, nitrogen dioxide, and nitric oxide. Infrared and colorimetry measurements were compared.

A study of the Boston Sumner Tunnel (3) in September 1961 provided information on its air quality, the quantity of pollutants emitted per vehicle-mile of tunnel travel, and the relative effect of vehicle exhaust on outdoor concentrations of various suspended particulates. Average carbon monoxide concentration in the tunnel was 70 parts per million. Average suspended particulate concentrations, in micrograms per cubic meter, were: total particulates, 600; organics, 225; lead, 45; benzo[g,h,i]perylene, 1; benzo[a]pyrene, 0.69; and coronene, 0.53. The relative effect of vehicle exhaust on outdoor concentrations for several suspended particulates was estimated. Ranked from most to least significant, they were coronene, benzo[a]pyrene, benzo[g,h,i]perylene, lead, total organics, iron, titanium, and total suspended particulates.

Hydrocarbon concentrations in Los Angeles air were compared with diluted, irradiated and nonirradiated exhaust samples (4). Relative concentrations of the individual hydrocarbon groups did not change significantly with total concentration. Total hydrocarbons were proportional to the sum of the amount of nitric oxide plus nitrogen dioxide. Comparison of

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atmospheric hydrocarbons with automobile exhaust indicated that natural gas could be a major source of low-boiling hydrocarbons in the Los Angeles atmosphere. Automobile blowby and carburetor and fuel tank evaporation contributed significantly to atmospheric concentrations.

Atmospheric Reactions

Sunlight acts on diluted automobile exhaust to form products that irritate eyes, damage plants, and form visibility-obscuring aerosols. Dilute mixtures of hydrocarbons, nitrogen oxides, and sulfur dioxide experimentally exposed to sunlight produce these same products (5).

Ozone and a newly identified series of compounds, the peroxyacyl nitrates, are the principal plant toxicants formed. The peroxyacyl nitrates are powerful eye irritants, as are formaldehyde and acrolein, also produced in the reaction. One species, peroxyacetyl nitrate (PAN), must be handled carefully. A dozen samples of prepared, purified PAN have exploded violently.

Only with some of the complex olefins are significant amounts of aerosol formed without sulfur dioxide. Small amounts of sulfur dioxide greatly enhance aerosol formation when straight-chain monolefins are used.

Recent photochemical research has concentrated on the highly reactive, eye-irritating, plant-damaging olefins. Because of their reactivity and concentration, the aromatic hydrocarbons are probably also important (6). Similarly, higher molecular-weight paraffins and organic oxygenates may be significant. Photochemical smog would probably persist even if all olefins were eliminated from the atmosphere.

The conversion of nitric oxide to nitrogen dioxide is faster than nearly any other photochemical reaction observed in outside air or in laboratory irradiations of dilute automobile exhaust. Dr. P. A. Leighton described several photochemical reactions that produce nitrogen dioxide and concluded that more research is needed to determine the importance of each (7).

Nitrogen dioxide has an undesirable color, as well as being toxic, and this color becomes

more noticeable as particulate concentration is reduced. Detailed studies of nitrogen dioxide formation have in the past been deferred in favor of more pressing problems. Since present plans in California are to control hydrocarbons but not nitric oxide, its nature and characteristics become more important.

In another study alkyl and alkoxy radicals were reacted with NO_2 to determine formation rates of the various products (8). The major product was formed by a direct combination of a radical with NO_2 , giving RNO_2 . RONO was produced at less than one-third the rate of the major process. The process yielding $\text{RO} + \text{NO}$ was important only when R was an alkyl.

Smog-type aerosols are formed by the accumulation of photochemical reaction products on nucleating particulates ever present in the atmosphere, a process closely analogous to fog formation (9). The condensible substances result from the interaction of traces of oxidizing gases (NO , NO_2 , O_3) with the heavier unsaturated hydrocarbon molecules (aromatics, olefins) when irradiated by sunlight in the 320–400 millimicron range. Traces of SO_2 significantly enhance and stabilize such condensates. The smog particle diameters range predominantly from 0.3 to 1.0 micron at a concentration of about 1 billion particles per cubic meter. Since this size range falls into the wavelengths of visible light (0.36–0.72 micron), the formation of such aerosols reduces visibility effectively by diffuse light scattering rather than by absorption as do the larger dusts. Because its fallout rate is negligible, smog has a more lasting effect than dusts.

Smog irritation decreases as humidity increases. This may help explain why the lungs of normal individuals, with near 100 percent humidity, are not irritated much, even during severe eye irritation.

Carbon Monoxide

Sixty million tons of carbon monoxide were emitted into the U.S. atmosphere in 1953 (2). Los Angeles vehicles emit 9,000 tons of CO per day, or 3.3 million tons per year. Carbon monoxide concentration in vehicle exhaust is about 1,000 times greater than its concentration in air adjacent to heavy traffic (10). The average

carbon monoxide concentration during the last 3 months of the year in Pasadena and downtown Los Angeles is about 15 ppm. Weekday concentrations are about 25 percent greater than weekend concentrations. Variations in seasonal averages of carbon monoxide concentration and cardiopulmonary deaths occur in a phasic pattern. This pattern is about 180° out of phase with temperature and inversion height. Such phasic patterns need not indicate causal relationships. A mathematical model has been programed on a computer to estimate the carboxyhemoglobin concentration in blood as a function of fluctuating carbon monoxide concentration in air.

Total carbon monoxide exposure may be determined by combining CO exposure during commuting and working with that from air pollution, cigarettes, and other sources. Other important questions concerning the effects of the substance are the population at risk, the effect of carbon monoxide on systems other than hemoglobin, and possible synergistic effects with other vehicle exhaust pollutants.

Blood carboxyhemoglobin concentration values are useful for population field studies, CO diffusion studies, rapid diagnosis of acute CO intoxication, and "chronic effects" studies. Carboxyhemoglobin concentrations may be determined directly from a blood sample or indirectly by breathholding or rebreathing. In a suggested method (11), 4 liters of 100 percent oxygen are rebreathed for 2 minutes. This method takes less time and gives greater sensitivity than previous rebreathing methods.

Carboxyhemoglobin concentrations were determined hourly on a smoker and a nonsmoker in Los Angeles on two different days (12). On the first day atmospheric carbon monoxide concentrations (outdoors and in a municipal garage) ranged from 15 to 60 ppm. The nonsmoker's carboxyhemoglobin level ranged from 1 percent of saturation at 8 a.m. to 3 percent at midafternoon. During this same interval, the smoker's carboxyhemoglobin level rose from 2.5 percent of saturation to 9 percent. The smoker had smoked the usual 15 cigarettes during this first day. On the second day, the smoker smoked a cigarette each half hour, beginning at 8 a.m. The ambient air CO concentration ranged from 15 to 30 ppm (the subjects stayed

out of garages). From 8 a.m. to midafternoon, the nonsmoker's carboxyhemoglobin level rose from 2 to 2.5 percent of saturation, while the smoker's level rose from 3 to 12 percent of saturation.

Eye Irritation

Eye irritation was measured on a panel of people exposed to 0.2 to 2.0 ppm concentrations of olefins and nitrogen dioxide in purified air (13). Trace concentrations of a branched, internal olefin (cyclohexene) produced significant amounts of eye irritants other than formaldehyde and acrolein. The culprit might be peroxacetyl nitrate. Stirred-flow conditions produced more eye irritation than quiet conditions. Small changes in flow about the eyes caused large changes in eye irritation. Sulfur dioxide slightly decreased eye irritation and caused an aerosol to form.

Lung Effects

Lung tissue is highly absorptive and particularly sensitive to air pollutants. Lung irritants, such as nitrogen dioxide or phosgene, produce acute pulmonary edema. Similar stimuli, repeatedly sustained, may cause chronic effects. Edema caused by lung irritants begins as patchy congestions throughout the lung. Blood pressure does not change. Sympatholytic drugs do not help. Meyers and Hine (14) suspect that the blood congestion is due to a loss of normal sympathetic tone. This could tie in with Stokinger's hypothesis (3) that a "conditioning" exposure to an irritating gas protects an animal against a second insult from the gas that would be fatal if the conditioning exposure had not toughened or scarred the lung to some degree.

Normal adults without respiratory disease were exposed to nitrogen dioxide concentrations of 1-50 ppm in research conducted by Meyers and Hine. They recorded subjective responses such as odor and eye irritation but no statistically significant changes in respiratory functions such as inspiratory reserve, vital capacity, expiratory reserve, and 10-second maximum breathing capacity. Changes of less than 28 percent were not considered significant when day-to-day variations were considered.

The bronchial tree fights chemical, physical,

and biological invaders by secreting mucus and by the movement of its hairlike cilia, which sweep the invaders up the tree and into the throat, where they are swallowed. High-pollutant concentrations paralyze cilia and destroy lung surface layers. The effects of lower concentrations may be apparent only by the subject's decreased resistance to a secondary stress such as an airborne infectious micro-organism. To test this hypothesis, mice were first exposed to ozone or nitrogen dioxide and later infected with aerosols of *Klebsiella pneumoniae* (15). Mortality increased up to 50 percent in the group of mice that were exposed to 4 ppm ozone for 3 hours or 5 ppm nitrogen dioxide for 2 hours and 6 hours later were challenged with *K. pneumoniae*. When the interval between insults was extended to 27 hours, the differences in mortality of exposed and unexposed mice were not statistically significant. These experiments indicated that the injury produced by these gases was not permanent and that recovery took place in less than 24 hours.

Another example of disease due to double insult has previously been reported in mice exposed to influenza virus and an aerosol of carcinogenic ozonized gasoline (4). Mice exposed to both stresses developed lung cancer, while those exposed to only one did not. The effect of multiple stresses is also evident in the increased lung cancer rate reported among persons exposed to both air pollution and smoking (5).

Chronic lung disease is more prevalent in Great Britain than in the United States and more prevalent in the eastern United States than on the west coast (16). Not much detail is available on this general trend, however. More definitive information is being obtained for New York City by measuring prevalence of obstructive lung disease in some well-defined segments of the population.

White men over 50 years of age living in highly polluted sections of Buffalo, N.Y., experience elevated death rates from asthma, bronchitis, stomach cancer, and lung cancer (17). This observation has sparked a study to test the hypothesis that air pollution is related to pulmonary function and other health measurements. Aerometry and health data have been gathered and are being analyzed.

Obstruction is the basic denominator in some chronic respiratory diseases. The obstruction may be caused by loss of lung elasticity (emphysema), lung passage constriction (asthma), retained secretions, or a combination of these. During the gradual onset of chronic obstructive respiratory disease, many individuals are well enough to work and are not under medical care, but they do have disease symptoms. Rapid screening techniques have been developed to find such persons in order to treat them and slow the progression of disease. With one technique (18), 100 persons can be screened per hour. Of 4,000 apparently well California workers screened with this technique, 2.7 percent had chronic obstructive respiratory disease.

Pulmonary function was measured on 710 persons consecutively admitted to a Seattle tuberculosis hospital to determine the incidence of diffuse obstructive pulmonary syndrome (19). This syndrome was present to a mild degree in 15 percent of the group, to a moderate degree in 11 percent, and to a severe degree in 8 percent. Diffuse obstructive pulmonary syndrome was significantly related to tuberculosis, asthma, organic heart disease, lung cancer, and frequent, long-lasting chest colds.

The natural history of emphysema remains essentially unknown. It is generally believed to be caused by breathing cigarette smoke, industrial dusts and fumes, and smog. Chronic bronchitis is believed to precede emphysema, but some emphysema without chronic bronchitis has been observed. To determine the onset and progression of emphysema, 50 normal individuals, 50 with chronic bronchitis, and 50 with chronic bronchitis and emphysema are being studied for several years (20).

The emphysema death rate has increased 400 percent in the last 10 years, from 15 to 63 deaths per million population per year (6). Direct population surveys are being conducted throughout the United States to measure prevalence of chronic respiratory diseases, since these diseases are not fully reported.

Lung function measurements of 3,000 long-shoremen in the San Francisco Bay area showed that the prevalence of chronic respiratory conditions is greater among smokers than non-smokers (21). One limitation of the usual method (age-adjusted rate) for assessing the

influence of smoking is that it fails to take into account the duration and amount of the smoking pattern. On the basis of the pharmacological dose-response relationship, an alternative method for assessing the smoking effect was developed. One of the findings was that smokers at age 40 showed the same rate of respiratory conditions as nonsmokers at age 50, and smokers at age 60 showed the same response as nonsmokers at age 80.

Smoking is an important factor in the prevalence of respiratory disease in Berlin, N.H., a community of 18,000 population (22). Ninety percent of the heavy smokers (more than two packs per day) had respiratory disease, a rate 4.5 times greater than the rate for those who never smoked. Thirty percent of the heavy smokers had irreversible obstructive lung disease, a rate 3.5 times greater than the rate for nonsmokers. Seventy-five percent of the heavy smokers had chronic bronchitis, a rate five times greater than the rate for nonsmokers. The smoking variable may be so strong as to obscure effects of air pollution in this community.

Sixteen chronic bronchitis outpatients in Baltimore, Md., are being studied on a daily basis (23). During periods of high atmospheric suspended particulate concentrations, three-fourths of the patients became worse. During periods of low suspended particulate concentration, one-quarter became worse. On the whole, the patients tended to become better and worse together. The patients' physiological changes seem to be related to environmental factors, but not to any one pollutant. Response appears to be a complex function involving combinations of environmental factors with subtle variations. The large variation in lung function in any one patient over a period of time is rather disconcerting when one realizes that many lung function surveys are conducted on a "one-shot" basis.

Plant Damage

A variety of plants are damaged by certain products of the reaction between motor vehicle emissions and sunlight, such as ozone, peroxyacyl nitrates, and the unidentified products of ozone-olefin reactions. Annual crop damage from such air pollutants is conservatively estimated at \$8 million in California and \$18 mil-

lion on the eastern seaboard (24). Peroxacyl nitrate (PAN) is extremely toxic, damaging leaves at concentrations of 5 parts per billion. Since PAN is relatively stable, while the ozone-olefin products last only a few minutes, and plant symptoms in Los Angeles more nearly resemble those of PAN damage than of other types, PAN probably causes most "oxidant-type" damage. Nitrogen dioxide is also highly toxic for plants; it was found to damage pinto bean leaves during 4-hour exposures at concentrations as low as 3 ppm.

The variety of effects of irradiated automobile exhaust on plants indicates that at least four classes of phytotoxicants are present (25). The type and quantity of toxicant varies as a function of type of fuel used, the ratio of hydrocarbons to nitrogen oxides, total hydrocarbon concentration, light intensity, and light quality. A 4-hour dark period immediately following exposure prevents some plant damage.

Poa annua leaves were exposed in studies using low- and high-olefin fuels (26). Five times as much exhaust gas from low-olefin fuel combustion was required to produce initial damage as was required from high-olefin fuel combustion.

Effects on Laboratory Animals

Animals are injured by oxidant gases in proportion not only to exposure but also to such factors as past exposure, chemical or hormonal treatment, other physiological stresses, and inherited enzyme deficiencies.

Resistance to ozone and nitrogen dioxide has been altered in laboratory animals by several means (27). Noninjurious quantities of ozone or nitrogen dioxide produced tolerance and cross-tolerance to future insults. Pretreatment with mineral oil mist produced tolerance to otherwise lethal quantities of ozone and nitrogen dioxide. A remarkable resistance to injury by these oxidants was also produced with anti-thyroid agents. Conversely, susceptibility was increased with thyroid hormone: 1.3 ppm ozone for 4 hours killed half of the thyroid-treated mice. By neurohumoral mechanisms, exposure to ozone or nitrogen dioxide affects cells throughout the body as well as at the injury site.

Laboratory animals were exposed briefly to diluted, irradiated and nonirradiated vehicular exhaust at concentrations ranging from 1 to 10 times present community levels (28). Irradiated atmospheres caused greater change than nonirradiated atmospheres. The most sensitive indicators of physiological response were pulmonary function, spontaneous activity, and mortality of impaired animals. The death rate in impaired, exhaust-exposed animals was significantly greater than the death rate in impaired animals in nonpolluted air.

Air Quality Standards

Russian air quality standards have been developed with the philosophy that the concentration of atmospheric pollutants should be kept below a level which causes a human response (29). Changes in the eyes' sensitivity to light, which often occur before odor recognition, provide the basis for several of the Russian standards.

The Russian standards probably have at least one present use in the United States. In a given community, pollution concentrations that do not exceed the Russian standards could be tentatively considered not to be problems.

Vehicle Exhaust Control

The average American automobile operates with a rich fuel mixture which produces considerable carbon monoxide and about 5.5 percent unburned hydrocarbons (30). Thus it is far from meeting the California standard of 1.1 percent unburned hydrocarbons. Air pollution could be decreased significantly by providing a near-stoichiometric air-to-fuel ratio of about 15. This ratio could be achieved by improved carburetor design, improved production control, and improved maintenance of the carburetor and ignition system. If the conventional engine is considered beyond redemption without an afterburner, a lean-fuel engine could be used to meet California standards. Lean-fuel engines, such as diesel, gas turbine, and stratified charge (a rich mixture near the spark plug and a lean mixture elsewhere in the cylinder), can meet both carbon monoxide and hydrocarbon standards.

During the past 30 years propulsion engineering has advanced from internal combustion engines through gas turbines to rockets. During this time American motor vehicle transmission and body design have changed markedly, but the engine has not (31). With the need for an automobile with less air-polluting emissions, consideration should be given to various propulsion systems such as fuel cells, storage batteries, gas turbines, and improved internal combustion engines.

Atmospheric emissions from internal combustion engines could be reduced by encouraging lighter vehicles, lean-mixture operation, semi-annual vehicle inspection, and stratified-charge operation. If the smog-free automobile is to be achieved, a new sense of mission, a lot more money, and additional competent researchers will be required.

CONFERENCE PAPERS

- (1) Rose, A. H., Jr., and Smith, R.: A direct measurement technique for automotive exhaust emissions.
- (2) Linnell, R. H., and Scott, W. E.: Diesel exhaust gas analysis techniques and preliminary results.
- (3) Larsen, R. I., and Konopinski, V. J.: Sumner Tunnel air quality.
- (4) Nelligan, R. E.: A comparison between the hydrocarbons in automobile exhaust and those found in the Los Angeles atmosphere.
- (5) Stephens, E. R.: The reactions of auto exhaust in sunlight.
- (6) Altshuller, A. P., Sleva, S. F., and Kopczynski, S. L.: Photo-oxidation of aromatic hydrocarbons.
- (7) Leighton, P. A.: Some remarks on the nitric oxide-nitrogen dioxide conversion.
- (8) Ausloos, P., and Rebbert, R. E.: Reactions of free radicals with NO₂.
- (9) Goetz, A.: Visibility restriction by photochemical aerosol formation.
- (10) Goldsmith, J. R.: Some epidemiological problems of community carbon monoxide exposures.
- (11) Hackney, J. D., Kaufman, G. A., Lashier, H., and Lynn, K.: The estimation of carbon monoxide hemoglobin by rebreathing.
- (12) Hackney, J. D., Lashier, H., Lynn, K., and Spier, C.: Comparative effect of two sources of exposure to carbon monoxide on carbon monoxide hemoglobin levels.
- (13) Doyle, G. J., Endow, N., and Jones, J. L.: The effects of photochemical aerosols on eye irritation.

- (14) Meyers, F. H., and Hine, C. H.: Some experiences with nitrogen dioxide in animals and man.
- (15) Ehrlich, R.: Effects of atmospheric pollutants on resistance to respiratory infection.
- (16) Densen, P. M., Breuer, J., and Bass, H. E.: Organization problems in the New York City survey for chronic respiratory disease.
- (17) Winkelstein, W., Jr., and deGroot, I.: The Erie County air pollution-pulmonary function study; study design and selected preliminary findings.
- (18) Leftwich, C. I., Manson, R. M., and Rowan, R. D.: The use of rapid simple pulmonary function tests as screening devices in the detection of chronic respiratory disease.
- (19) Hallett, W. Y., and Martin, C. J.: An epidemiologic study of obstructive pulmonary disease and a review of physiologic methods.
- (20) Balchum, O. J., and Gierston, H. W.: A longitudinal correlative clinical and physiological study of chronic bronchitis and emphysema.
- (21) Hechter, H. H., Goldsmith, J. R., Perkins, N., and Borhani, N. O.: Pulmonary function in longshoremen.
- (22) Ferris, B. G., and Anderson, D. O.: The prevalence of chronic respiratory disease in a New Hampshire Town: I. Methodology. *Am. Rev. Resp. Dis.* 86:165-177, August 1962.
- (23) Spicer, W. S., Jr., Storey, P. B., Morgan, W. K. C., Kerr, H. D., and Standiford, N. E.: Variation in respiratory function in selected patients and its relation to air pollution.
- (24) Darley, E. F., Dugger, W. M., Mudd, J. B., Ordin, L., Taylor, O. C., and Stephens, E. R.: Effect on plants of air pollutants derived from motor vehicle emissions.
- (25) Brandt, C. S., Hindawi, I. J., Seidman, G., and Dunning, J. A.: Preliminary observations on the effects of irradiated auto exhaust on plants.
- (26) Noble, W. M.: The relation of plant damage to fuel composition.
- (27) Mountain, J. T.: Biochemical effects related to ozone and nitrogen dioxide injury and tolerance in laboratory animals.
- (28) Murphy, S. D., Leng, J. K., Ulrich, C. E., and

Davis, H. V.: Effects on experimental animals of brief exposure to diluted automobile exhaust.

- (29) Ryazanov, V. A.: About the criteria for evaluation of effects of atmospheric pollutants at low concentrations upon an organism.
- (30) London, A. L.: The application of research to motor vehicle emission control—Can we avoid afterburners?
- (31) Stern, A. C.: Prospects for exhaust control by engine modification.

Copies of some of the conference papers are available from Dr. John R. Goldsmith, Head, Air Pollution Medical Studies, California Department of Public Health, Berkeley 4, Calif.

REFERENCES

- (1) Middleton, J. T., and Haagen-Smit, A.: The occurrence, distribution, and significance of photochemical air pollution in United States, Canada and Mexico. *J. Air Pollut. Contr. A.* 11: 129-134, March 1961.
- (2) Rupp, W. H.: *In* Air pollution handbook, edited by Magill, Holden, and Ackley. McGraw-Hill Book Co., New York, 1956, pp. 1-5.
- (3) Stokinger, H. E.: Themes and variations in experimental toxicology. *In* Proceedings of the Fourth Air Pollution Medical Research Conference, 1960. California State Department of Public Health, Berkeley, Calif., 1961, 3d day's proceedings, p. 126.
- (4) Wiseley, D. V., Kotin, P., Fowler, P. R., and Trivedi, J.: The combined effect of repeated viral infection and exposure to carcinogenic aerosols on pulmonary tumor induction in C57 mice [Abstract]. *J. Occup. Med.* 3: 547-548, November 1961.
- (5) Air pollution manual. American Industrial Hygiene Association, Detroit, 1960, part 1, p. 44.
- (6) Speizer, F., and Landau, E.: Epidemiologic studies of obstructive ventilatory disease of the lung; a follow-back approach for studying mortality from obstructive ventilatory disease of the lungs: Results of a pilot study. *Am. Rev. Resp. Dis.* 83: 826-841, June 1961.

Responsibility for Welfare of Aged

Census data suggest that two- and three-generation families are increasingly taking advantage of economic advances and the growing housing supply which enable them to live in separate homes. We can only conclude from the existing evidence that this is usually the preferred relationship for all concerned, except when economic or health needs dictate otherwise.

During the 1950's the nation's housing stock increased by more than 12 million units, while the total number of families increased by only half that amount. Together with a general rise in real incomes, this enabled many American families, who had previously been forced to live "doubled up," to acquire separate quarters. This change was reflected dramatically in the housing situation of older persons. In 1950 about 31 percent of those aged 65 years and over lived in households headed by others. By 1959 this proportion had declined to less than 23 percent. And the end of the decline is not yet in sight, because between 1959 and 1961 the proportion decreased an additional 1 percent.

Although a substantial part of the decrease was accounted for by a decline in the percentage of older people living in relatives' households, there was an even more dramatic decrease among elderly lodgers in the homes of nonrelatives. Today, the percentage of elderly lodgers is only about one-third that in 1950. There has also been a decline in the percentage of older persons living in institutions, which may provide some statistical backing for the frequent contention that institutionalization is sometimes a last resort when more suitable accommodations are unobtainable.

Many people have concluded from facts such as these that the old family relationships are dissolving. However, recent research suggests quite a different situation. For example, national surveys by Dr. Ethel Shanas of the National Opinion Research Center, University of Chicago, reveal that only one older person of seven lives more than a short ride away from his or her nearest child, and a majority live

within walking distance. Although fewer families live together at present, many still live close enough to visit frequently.

Furthermore, the popular notion that children are abandoning their parents to the mercy of the State has been sharply rebuffed by findings of surveys which indicate that children consistently show a greater feeling of responsibility for their parents' welfare than the parents themselves appear to desire. The parents are more inclined to want independence of their children, especially in a financial sense. And they are generally disposed to feel that financial support, if needed to supplement their own resources, should be provided through government channels.

Factual knowledge discloses still another fallacy in popular thinking about the responsibility of children for their parents' welfare. As medical science increases the lifespan, we are developing a situation for which there is no precedent; that is, substantial numbers of elderly parents whose children are past retirement age. In this instance, there is really no meaning to the admonition that the "young" should support the "old," for in fact both generations are past the age where they can support themselves by their own labors.

In Shanas' surveys, one of nine older persons, when asked to whom other than the spouse he would turn for aid in a health crisis, named someone 65 years of age or older. An additional 13 percent named someone aged 55-64 years. These data coupled with the fact that about one older person of every five has no living children clearly reveal the basic flaws in the still disturbingly current notion that the needs of the aged in housing, health, and even income could be met without government intervention if only children would recognize their responsibility to their parents.—*Excerpt from an address by Donald P. Kent, Ph.D., Director, Special Staff on Aging, Department of Health, Education, and Welfare, at the 15th annual University of Michigan Conference on Aging, Ann Arbor, June 18, 1962.*