III. BIOLOGIC EFFECTS OF EXPOSURE

Extent of Exposure

The primary function of the coke plant is the production of metallurgical coke for use at the blast furnace. A secondary function is the recovery of chemical byproducts during the transformation (high temperature carbonization) of bituminous coal into coke. Prior to World War I, the main source of metallurgical coke in the United States was the beehive coke oven. These ovens were used solely for the production of coke, and the volatile matter produced during carbonization was emitted into the atmosphere. The byproduct coke plant, which allows for recovery of tar, oils, and chemicals from the volatiles, was introduced around the turn of the century, and by 1931 byproduct ovens accounted for 80% of coke production. Except for brief periods during World War II and the Korean War, this figure has been about 95%, with production by beehive ovens steadily declining. [1]

In addition to the production of metallurgical coke, there are several other methods of coal carbonization which, although they are not used in the United States at present, are of interest because of similar exposure to the volatiles and their byproducts, and there are previous reports [2-8] of unusual cancer experience in workmen employed in these areas. Major among these processes are the production of household gas in vertical and horizontal retorts, and the generation of producer gas for industrial use.

The modern byproduct coke plant is a semicontinuous operation which may be subdivided into three rather distinct work areas in terms of function and potential exposure to environmental hazards. These are: (a) the coal handling area where coal is received by rail or barge, and where provision is made for the handling, storage, and blending of several types of coal before transfer to the coke ovens; (b) the coke ovens, grouped into one or more batteries, with equipment for charging and discharging the ovens and the quenching of coke; and (c) a byproduct plant for recovery of gas and chemical products. [9]

In 1970, according to the Bureau of Mines, there were 64 slot-oven plants in the United States operating 13,218 coke ovens. [10] In a study [11] of steelworkers employed in Allegheny County, Pennsylvania, 1,327 men were enumerated as coke oven workers in two plants composed of 1,754 ovens. Applying the "coke oven worker-to-coke oven" ratio from this study to the number of coke ovens reported by the Bureau of Mines for 1970 would put the direct coke oven worker population at approximately 10,000 persons.

Historical Reports

It has long been recognized that some agent (or agents) produced during the combustion or distillation of bituminous coal is carcinogenic for the skin of man, and since the turn of the century a variety of industrial populations exposed to coal tar products have shown a special liability to cancer of the skin. [12] More recent studies [3-8,13-16] of men employed in some of these areas indicate that exposure

to coal tar products and coke oven emissions also may result in increased liability for cancer of other organ systems.

The history of the "coal tar" cancers begins with the observations of scrotal cancer in London chimney sweeps by Percivall Pott [17] in 1775. Since that time, a large body of evidence has accumulated demonstrating that persons engaged in the carbonization of bituminous coal and those handling certain byproducts are at excess risk of scrotal and other forms of skin cancer. In a comprehensive study of the relationship between these tumors and exposure to coal tar products, Henry [12] reported an average annual scrotal cancer mortality rate of 21.1 per million in coke oven workers during the period 1911-1938 compared to a general population rate of 4.2. He also noted 84 cases of epitheliomatous ulceration or cancer of the skin (40 scrotal) in British coke oven workers for the period 1920 to 1943, and 11 fatal scrotal cancers among men with prior coke oven employment.

Epidemiological Studies

The first report of unusual lung cancer experience in men engaged in coal carbonization concerned Japanese producer gas workers. [2] Recent observations on this population show that gas producer men continued to show a lung cancer mortality rate 33 times that observed for other Japanese steelworkers many years after the facility was closed. [8] The excess lung cancer risk for gas producer men was confirmed by British studies of death certificates in England and Wales for the years 1921-1932. [18] Furthermore, this study showed that

other coal carbonization and byproduct workers experienced greater than expected lung cancer mortality. The excess indicated for "gas stokers and coke oven chargers" in an extended report through 1938 was approximately three-fold. [13] More recent estimates for this group indicate an excess of only 32%, [14] and a report on a population-based study of British coke oven workers, although consistent with an excess of lung cancer mortality, was not particularly striking (14 deaths observed compared to 10 expected among retirees). [15] Possible explanations for variations in the estimates of excess risk for lung cancer among coke oven workers have been discussed elsewhere. [11] Doll, [4] in a 1952 study of gas works pensioners (gas retort workers), observed an 81% excess of lung cancer deaths in comparison with the general population. In 1971, Lloyd [11] reported that men employed as coke oven workers in Allegheny County, Pennsylvania had a lung cancer mortality rate two and one-half times that predicted by the experience of all steelworkers (31 deaths observed vs. 12.3 expected). A more recent study [16] of men employed during 1951-1955 at coke plants located throughout the U.S. and Canada confirms these findings, reporting relative risks for lung cancer almost identical to those observed in the original report.

An excess risk of bladder cancer among men employed at coal carbonization processes was first reported by Henry et al [19] in 1931. In their review of bladder cancer deaths for the period 1921-1928, they reported a greater than expected mortality for nine occupational groups exposed to coal gas, tar, and pitch. Furthermore, of

the fifteen occupational groups exhibiting an excess of 50% or more, five were among the "coal tar" occupations. Forty-five bladder cancer deaths were observed in "gas stokers and coke oven chargers" compared to 33.7 expected. Unfortunately, this group cannot be subdivided to obtain an estimate for the coke oven chargers (larrymen). More recent studies of British gas retort workers confirmed an excess of bladder cancer for this group and suggested that these tumors may be associated with exposure to beta-naphthylamine at the gas retorts. [6,7] The observed incidence of bladder cancer in 4661 American coke oven workers was not excessive [16]; but the possibility of excess mortality cannot be ruled out when it is recognized that this is a comparatively rare cancer site with a long latent period, and that the study population has an extremely high risk for cancer of several other sites. On the other hand, Redmond et al [16] report that U.S. coke oven workers are experiencing an excess of cancer of the kidney (8 observed vs. 2.6 expected). This observation is consistent with the 1951 report of the British Registrar General which shows an excess risk of bladder and kidney tumors for men employed as "laborers and unskilled workers in coke ovens and gas works." [14]

Excess mortality from cancer of other sites for coke oven workers and other coal carbonization workers has been reported from several sources. [3,5,11,14,18,20] However, these findings are generally based on very limited data and have yet to be confirmed. At the same time, it should be pointed out that such responses could be part of the picture of a general carcinogenic response among coal

carbonization workers who have already been noted to have extremely high risks of cancer from three sites (skin, [12] lungs, [2,4,8,11,13-16,18] and urinary organs [6,7,11,16]). Other cancers and cancer sites reported to be in excess from these limited studies are the larynx, [18] nasal sinuses, [3,5] pancreas [14] (which has been associated with exposure to beta-naphthylamine), [20] blood forming organs (leukemia), [14] and stomach. [14] Lloyd [11] has pointed out that American coke plant workers employed at work areas other than the coke ovens may be at increased risk of cancer of the digestive tract.

With the exception of the studies of lung and kidney cancer in American coke oven workers, data are not available to characterize the carcinogenic response in terms of extent of exposure. However, the accumulated evidence suggests the possibility of some difference in response according to carbonization process. As shown in Table VII-1, [9,21-25] there appears to be a positive relationship between temperatures attained during carbonization and the lung cancer response for men employed in the several areas. Doll et al reported that the bladder cancer response varies according to type of gas retort. [6] It has been noted, also, that the skin cancer response among persons exposed to coal tar products appears to be related to the level of distillation, with the rate increasing with successive distillations. [12] However, variation in response may be related to the materials and methods employed and nonoccupational factors such as personal habits and level of medicinal surveillance. For example,

general population rates for skin cancer have been much higher in England and Wales than in the United States. [26,27]

As regards cancer of the lung, Lloyd [11] and Redmond et al [16] have shown that the response is related to both length of exposure and relative level of exposure as determined by specific job assignments at the coke ovens. In Allegheny County, men employed at the coke ovens for five or more years exhibited a mortality rate for lung cancer 3.5 times the expected rate, while the rate for all coke oven workers was 2.5 times that expected. The same relationship between length of exposure and lung cancer mortality was noted for coke oven workers outside of Allegheny County. An even greater difference for lung cancer is seen when the coke oven workers are classified into broad exposure groups according to work assignments. The lung cancer mortality rate for men employed at the top of the coke ovens is seven times the expected rate, whereas the rate for men employed exclusively at the sides of the ovens is 25% greater than expected. Among men employed at the coke ovens for five or more years, there is a definite gradient in response by work area with side oven workers showing a 46% excess, men with mixed side and topside employment but less than five years topside experiencing a rate almost three times the expected rate, and men employed full-time at the top of the ovens showing a lung cancer mortality rate 10 times that noted for all steelworkers. The distribution among 4661 coke oven workers of the eight kidney cancers reported by Redmond et al [16] does not suggest any association with a specific work area or length of exposure, but the

overall relative risk for kidney cancer was 7.5. It should be noted that kidney cancers occurred predominantly in the parenchyma rather than in the kidney pelvis, the ureters, or bladder where the urine is more concentrated.

The possibility that the unusual lung cancer experience of coke oven workers might be accounted for by factors other than occupational exposure has been examined in detail by Lloyd [11] and Redmond et al. [16] Account has been taken of selective factors such as age, race, place of birth, residence, and employability, and it has been demonstrated that the differences observed cannot be explained by these factors. Consideration has also been given to the possibility that the excess lung cancer mortality of coke oven workers might be explained by differences in smoking habits, since it is known that tobacco use (primarily cigarette smoking) is positively correlated with the incidence of lung cancer. [28] In the single study of coal-carbonization workers for which smoking histories were available, Doll et al [6] ruled out cigarette smoking as a selective factor by showing that the smoking habits of persons employed in the several areas of the gas industry were comparable to those in the general population. Unfortunately, information on cigarette smoking history is not available for the American coke oven workers so that a direct approach to the question is not presently available. However, comparison of the age-specific lung cancer mortality of coke oven workers with mortality observed for American cigarette smokers during the same time period provides an indirect measure of the possible extent of effects

due to cigarette smoking. The lung cancer mortality rates chosen for comparison come from a study of 293,000 U.S. veterans reported by Kahn. [28] These rates, according to smoking class and age, are contrasted with the lung cancer mortality rates for steelworkers and coke oven workers in Table VII-2. A detailed description of how these rates were determined is given elsewhere. [29]

Although different age groupings were used in the two studies, it can be seen in Table VII-2 that the steelworkers rate for men under age 45 is between the rate for total cigarette smokers ages 35-44 and 45-54. The same pattern is seen for each of the succeeding age groups. It appears that steelworker mortality may have been considerably higher than that for nonsmokers, somewhat higher than that for light smokers, but considerably lower than the mortality of heavy smokers. The lung cancer mortality rates for nontopside workers, although much higher than those observed for steelworkers, also are well within the limits defined for heavy smokers. On the other hand, the rates observed for topside coke oven workers are far greater than the rates for the veterans. For topside workers under age 45, for example, the rate of 141 is higher than that seen for heavy smokers of an older age (a rate of 95 for ages 45-54). The differential for the other age groups is even more striking. The topside worker rates of 819 for ages 45-54 and 1,356 for ages 55 and over are considerably higher than the veterans rates for any smoking class and age group. It is thus shown that the carcinogenic agent responsible for the excess lung cancer seen in topside workers has an

effect considerably beyond that predicted by heavy cigarette smoking. While these findings do not rule out the possibility of some difference in coke oven worker mortality associated with differential smoking patterns, it indicates that the marked differences in lung cancer mortality between topside workers and other steelworkers cannot be explained solely by this factor.

There is some evidence that exposure to emissions at coke ovens and gas retorts may be associated with an increased occurrence of bronchitis. Doll et al [6] compared the death rate from bronchitis among men who had heavy exposure to products of coal carbonization at gas retorts to the rates in the general population and among all gasworkers. In both cases, the mortality in the heavily exposed group was significantly higher. A differential in bronchitis response according to type of retort house also was observed, with the largest excess in vertical houses while a lower level of mortality occurred at the horizontal retorts. Thus, the pattern of mortality from bronchitis was the reverse of that for lung cancer previously noted in this study. The authors tentatively concluded that exposure to products of coal carbonization at the retorts was causally related to increased mortality from bronchitis. However, other evidence suggests that differences in demographic characteristics [30] and in cigarette smoking habits [31] may account for at least some of the excess.

The same relationship between extent of exposure and incidence of malignant and nonmalignant respiratory disease also is suggested by the most recent findings on American coke oven workers, [16] but, the

differences for nonmalignant respiratory disease are not significant and too few deaths have been observed to date to reach firm conclusions by specific respiratory disease.

In summary, evidence from other countries, involving a variety of processes, makes it clear that workers intimately exposed to the products from the carbonization of coal experience increased mortality due to cancer of the skin, [12] lung, [2,4,8,13-15,18] bladder, [6,7,14,19] and kidney. [14] There also is evidence from limited studies suggesting increased mortality due to nonmalignant respiratory disease [6,30]; and to cancer of the larynx, [18] pancreas, [14] stomach, [14] blood forming organs (leukemia), [14] and the nasal sinuses. [3,5] While not all of these diseases have been demonstrated in American coke oven workers, neither have they been eliminated as possible health problems. Among American coke oven workers, cancer of the lung, [11,16] and kidney, [16] and, among nonoven coke plant workers, cancer of the digestive system [11] have been shown to occur at an excessive rate. Since there is evidence that the disease response is related both to relative level and length of exposure, [11,16] reduction of the emissions or exposure to them should result in a reduced health hazard.

Animal Toxicity

Although coke oven emissions have not been directly tested experimentally for toxic or carcinogenic properties, many components have been studied separately. The carcinogenic properties of coal tar have been well known since the first chemically induced tumors in

experimental animals were reported in 1918 by Yamagiwa and Ichikawa, who obtained papillomas and carcinomas after painting the ears of rabbits with coal tar. [32]

Benzo(a)pyrene, a chemical carcinogen frequently used for the experimental induction of cancer, was identified as a carcinogen following its isolation by investigators seeking to identify coal tar's carcinogenic constituents. [33] Another commonly used experimental chemical carcinogen found in coal tar is beta-naphthylamine, which has been demonstrated to be a potent bladder carcinogen in a variety of species, including the dog, [34] and the monkey. [35]

A variety of skin carcinogens have been identified in coal tar. Combes [36] divides coal tar's primary cutaneous carcinogens into two groups based upon molecular structure. Benz(a)anthracene and its derivatives, benzo(a)pyrene; 9,10-dimethyl-1,2-benzanthracene; 1,2,5,6-dibenzanthracene; 20-methylcholanthrene; 5,9,10-trimethyl-1,2-benzanthracene; and phenanthrene comprise the most important of the two groups. According to Combes' classification, the other group contains the "azo" compounds in which are included 4-amino-2,3-azotoluene; 2,3-azotoluene; and p-dimethylamino azobenzene.

Co-carcinogenic factors can markedly affect the potency of a carcinogen. Bingham and Falk [37] reported that in skin painting studies utilizing C3H/He mice, the potency of both benzo(a)pyrene and benzo(a)anthracene was increased 1000-fold when the diluent used was n-dodecane, which has been identified as a product in the low temper-

ature carbonization of coal. [38] Coal tar itself apparently has co-carcinogenic or promoting activity. Friedewald and Rous [39,40] found that coal tar, benzo(a)pyrene, and methylcholanthrene all produced growths of essentially the same types in rabbits. However, the promoting effect of the tar was much greater, leading to the rapid production of fleshy, vigorous, and rapidly enlarging tumors.

Using weanling rats, mice, rabbits, and hamsters in a 90-day continuous dosage inhalation study at coal tar concentrations of 0.2, 2.0, 10.0, and 20.0 mg/cu m, MacEwen [41] reported that 0.1 to 1.5 mg of tar accumulated in the lungs of the animals exposed to the 20 mg/cu m level. When these animal tissues were examined microscopically for morphologic or cytologic change, immediately following cessation of exposure, no tissue lesions attributable to the tar deposited in the tissue could be identified. Although it was too early to detect a carcinogenic response, a significantly reduced rate of body weight gain was observed in all animal groups except the rabbits, which lost weight.

Inhalation studies of longer duration have demonstrated the induction of lung cancer by coal tar. In inhalation studies using C3H mice, Horton et al [42] showed that after 35 weeks of daily exposures to atomized tar at 100 mg/cu m of air, squamous-cell tumors had developed in five of 33 test animals. In the same study, irritation of the lungs by the prior inhalation of formaldehyde was not found to predispose the mice to subsequent development of squamous cell carcinoma after inhalation of coal tar fumes.

Other inhalation studies have demonstrated a promoting effect apparently due to irritation of the lungs. For example, Kotin and Wisely [43] reported that hydrocarbons (ozonized gasoline) failed to produce squamous metaplasia and epidermoid carcinoma in C57 Black mice when inhaled alone, but did so when inhaled with influenza virus. Tye and Stemmer [44] found that coal tars with phenols removed were less potent carcinogens than coal tars with phenols and attributed the co-carcinogenic potential of phenols to their irritant properties. Laskin et al, [45] reported that rats inhaling benzo(a)pyrene and sulfur dioxide in combination, but not when either was inhaled alone, developed bronchial mucosal changes and tumors of bronchogenic origin, which they considered to closely simulate lung cancer in man. Both benzo(a)pyrene and sulfur dioxide are present in coke oven emissions and may interact in humans as they apparently do in rats.

These studies [32-45] demonstrate the carcinogenicity of coal tar and some of its components for a variety of animal species. Especially convincing are studies such as that by Laskin et al, [45] which report a response similar to human lung cancer. In the face of these studies and the occupational experience previously discussed, there can be little doubt that coal tar and some of its constituents are human carcinogens. Thus, the presence of coal tar and volatiles from coal tar in coke oven emissions may account, at least in part, for the increased incidence of cancer observed in coke oven workers.

IV. ENVIRONMENTAL DATA and RESPIRATOR USE

The exact nature of the airborne particulates generated by the production of coke is very difficult to define and may vary with atmospheric conditions, the type and mix of coal used, and coking time. Particulates consist primarily of coal and coke dust plus condensed particles resulting from the distillation of the coal. According to the American Iron and Steel Institute (AISI), [46] during the charging without steam aspiration of an 18-ton oven emissions consisted of: particulate matter - 2.2 pounds/charge; water vapor - 15.4 pounds/charge; and tar vapor - 0.66 pounds/charge. Condensed coal tar vapor thus comprised 3.6% of total emissions or, eliminating water vapor from consideration since it would be evaporated from a filter used in air sampling, 23% of the total particulates discharged. In the same report, AISI states that polycyclic aromatic hydrocarbons constituted 2.1-3.1% of the total particulates and 3.2-5.8% of the benzene soluble fraction, which includes not only polycyclic aromatic hydrocarbons but also the soluble fraction of coal dust and other particulates, in samples collected from the front platform of a larry car.

Studies of the characteristics of particulate coke oven emissions have indicated a bimodal distribution of particle sizes, with peaks at 1-2 microns and greater than 10 microns aerodynamic equivalent diameters. [46] Coke oven emissions were described as a mixture of irregularly shaped coal and coke particles and spherical carbonaceous-organic material with tarry, sticky material intermixed. This mixture

is further complicated by the agglomeration of the smaller aerosols onto the larger, and adsorption of vapors. It was further reported that the benzene soluble fraction of total particulates increases with decreasing particle size.

In an effort to determine levels of exposure in coking operations, AISI [46] surveyed member companies having coke plants and requested exposure data relative to workers on and around coke ovens. Exposure data reported to AISI were collected during the period 1968-1972 by the member companies using standard industrial hygiene sampling procedures and represent 8-hour time-weighted average exposures. Although the data represent a wide range of coke oven operations, including both new and old batteries, and different operating procedures, the relative exposures for the eight job categories reported are apparent.

Data are summarized in Table VII-3. The range of concentrations of the benzene soluble fraction of coke oven particulate emissions, reported in milligrams per cubic meter, represents the maximum and minimum averages of the plant averages submitted. The mean shown (column 3) is the overall average of the individual plant averages. Included in Table VII-3 are less extensive data from studies made by the Pennsylvania Department of Environmental Resources beginning in 1966. [47]

A respirator is used to protect the wearer from the inhalation of harmful atmospheres. The conditions to be protected against range from those which are mainly a nuisance, as odor or irritation, to

those which are immediately dangerous to life. The hazard may be due to one or more toxic contaminants or to an atmosphere significantly deficient in oxygen. The contaminants may be in the gaseous or particulate state or in combination. Protection may be needed for only minutes, as in rescue operations, or for hours, as in routine use.

For adequate protection against the variety of conditions which may be encountered in different operations, many types of respirators have been developed. Each has a particular field of application and limitations from the viewpoint of protection, as well as advantages and disadvantages from the viewpoint of operational procedures and maintenance. Detailed information on the selection and use of respirators can be obtained from the Respiratory Protective Devices Manual published in 1963 by the American Industrial Hygiene Association and the American Conference of Governmental Industrial Hygienists. The American National Standard Practices for Respiratory Protection, ANSI Z88.2-1969, also classifies, describes, and gives the limitations of respirators.

Respirators generally fall into the following classifications:

(a) Atmosphere-supplying respirators

(1) self-contained

(2) hose-mask

(3) air-line

(4) combination self-contained and hose-mask or air-line

(b) Air-purifying respirators

(1) gas and vapor (gas mask and chemical cartridge)

(2) particulate (dust, fog, fume, mist, smoke, and sprays)

(3) combination gas, vapor, and particulate

(c) Combination atmosphere-supplying and air-purifying respirators.

Requirements for approval of many types of respirators have been established by the Secretary of the Interior and the Secretary of Health, Education, and Welfare, and are published as Title 30, Code of Federal Regulations, Part 11, (30 CFR 11). Copies of the most recent requirements may be obtained from the Publications Distribution Section, U.S. Bureau of Mines, 4800 Forbes Avenue, Pittsburgh, Pennsylvania 15213, or from the NIOSH Testing and Certification Laboratory, 944 Chestnut Ridge Road, Morgantown, West Virginia 26505.

An air-purifying respirator for protection against particulates is equipped with a mechanical filter designed to remove particulate matter from the inspired air by capture on the filter, which is usually a fibrous pad. These respirators consist of a soft resilient full, half, or quarter mask facepiece to which is attached one or two filters through which the inspired air is drawn. Check valves, present in most mechanical-filter respirators, prevent exhaled breath from passing through the filters and allow it to be forced out through an exhalation valve in the facepiece to the surrounding atmosphere. The facepiece is held securely to the wearer's face by a head harness or headband.

The useful life of the mechanical filters is limited by the buildup of resistance to inhalation as the contaminant is removed by the filter. The higher the concentration of contaminant in the air drawn into the filter, and the greater the wearer's activity when using a nonpowered respirator, the more rapidly the resistance to inhalation increases. Because of this, the filters must be changed more frequently. In high dust concentrations, the Type C positive-pressure supplied air respirator must be selected.

A powered air-purifying positive-pressure respirator contains a motor-blower which draws the contaminated air of the workplace through a mechanical filter, and discharges it into a facepiece, hood, or helmet. The device can be designed to ensure a positive pressure in the facepiece, so that high protection can be achieved. Since leakage through the facepiece seal is reduced and inhalation resistance is eliminated, lower facepiece seating forces can be used. Additionally, the air sweeping through the mask may provide some facial cooling while reducing moisture build-up in the mask. Therefore, acceptance of this device by the wearer should be higher than that of the conventional air-purifying respirator.

The air supply system for the powered air-purifying respirator may be designed to be worn by a worker or separately mounted with an air-line connection to the worker. For mobility, the power pack is mounted on the worker. This additional weight and bulk, however, may be unacceptable to the worker. As with the nonpowered air-purifying

respirator, the filter service life will depend upon the concentration of air contaminant.

A battery-powered air-purifying respirator for coke oven workers has been developed and evaluated [48] under the direction of William A. Burgess and under the sponsorship of the American Iron and Steel Institute. This half mask respirator has been evaluated by AISI [46] as having an average protection factor of 30.

The Type C positive-pressure supplied air respirator is an air-line respirator consisting of a half mask or full facepiece to which respirable air is supplied through a small diameter hose. The air-line respirator is the most comfortable to wear. There is little or no resistance to inhalation, and the flow of air usually provides a cooling and refreshing effect. The wearer supports little weight other than the facepiece and connecting hose. A limitation is the necessity of trailing the small diameter hose connecting the facepiece to the air source, which limits the travel of the wearer.

The problem of providing adequately fitting respirators is complicated by the wide range of facial sizes and shapes which must be accommodated. Differences in facial sizes and shapes result from a wide variety of factors, the most significant of which include age, sex, and race.

Facial hair, such as beards or sideburns, make it impossible to achieve an airtight seal between the facepiece and the face, particularly with the half mask respirator. Even the stubble resulting from failure to shave daily can cause serious inward leakage

of contaminated air. Therefore, workers who are to wear respirators with half or full facepieces should be cleanly shaven.

Providing respiratory protection for individuals wearing corrective glasses is a serious problem. The ability to wear corrective glasses with a half mask depends on the face fit. It is possible to obtain a seal with a poorly fitting respirator, but quite often the device will rest so high on the face as to make the wearing of glasses impossible. For a full face mask, a proper face seal cannot be established if the temple bars of the eyeglasses extend through the sealing edge. Some full facepiece designs provide for the mounting of special corrective lenses within the facepiece.

Any respirator affects the wearer's ability to see. The half mask and the attached elements can restrict normal downward vision appreciably. Diminished vision in the full face mask may be caused not only by the facepiece, but also by the design and placement of the eyepieces.

Speech transmission through a respirator can be difficult, annoying, and fatiguing to workers. Mere movement of the jaws in speaking may cause leakage between the facepiece and face, especially with the half mask respirator.

The wearer's comfort, and his acceptance of the distress caused by wearing a respirator, are no less important than the device's effectiveness. All factors such as improperly fitted respirators, uncomfortable resistance to breathing, and limitation of vision and speech transmission affect a respirator's acceptability.