

HEALTH HAZARDS FROM BERYLLIUM*

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The chemical toxicity of the beryllium ion exceeds that of any material ever used in industry but, as is true for ~~any~~ ^{other} industrial hazards, the risk in the handling and processing of this metal and its compounds can be eliminated if proper precautions are taken.

It was with much reluctance that many toxicologists finally accepted the fact that beryllium is toxic. This was understandable in view of the many ways in which the pattern of beryllium disease differed from the better known occupational diseases. However, evidence for the toxicity of beryllium accumulated during the past decade is overwhelming and the criteria by which a substance can be labeled as potentially dangerous have been amply satisfied.

The earliest reports of disease among beryllium workers came from abroad (1) in a series of contributions from investigators who identified what we now recognize as acute beryllium poisoning. However, the role of beryllium in these early cases was not understood and the acute disease, which is a type of chemical pneumonia, was erroneously related to the presence of fluorides and oxyfluorides which were co-contaminants of the workroom atmospheres in which the European cases occurred.

The first known cases in this country were reported in 1943 (2).

The histories of these cases and of others which have been reported in more recent years have been amply reviewed in prior publications (3)(4)

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and will not be discussed in detail here. From these reports, however, has emerged general agreement among investigators that beryllium is capable of producing a variety of toxic manifestations in humans:

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- a. Acute irritation of the respiratory tract, including chemical pneumonitis (which we will refer to as the "acute disease").
 - b. A chronic disease "berylliosis" in which lung involvement is the principal feature but in which there are also a number of systemic manifestations.
 - c. A variety of skin reactions including dermatitis, ulcers and benign tumors.

The Acute Disease

Certain beryllium compounds have the ability to produce an acute chemical pneumonitis which is similar in form to that experienced from exposure to phosgene, nitrous fumes and cadmium oxide. The disease runs its course in a few weeks and unless death occurs during this period complete recovery may be expected. A large majority of the hundred or so cases that have been reported (including about 15 deaths) occurred in the plants in which beryllium compounds are extracted from ore. However, some cases have been reported from the fluorescent lamp industry and from laboratories engaged in beryllium metallurgy. In the largest published series of cases, 47 survivors of the acute disease were followed for as long as 12 years and the investigators report that no persons included in their survey developed chronic berylliosis (4).

Acute pneumonitis has been produced by inhalation of beryllium metal, beryllium oxide, sulphate, fluoride, the hydroxide and the chloride (5). Experience with other beryllium salts is lacking but it may be assumed that all soluble beryllium compounds are capable of producing acute disease. Two cases of acute pneumonitis have been reported (6) from the fluorescent lamp industry where it appears that the exposures involved beryllium oxide in the preparation of the phosphor rather than the phosphor itself. Beryl ore and the various beryllium containing phosphors and copper alloys have not produced acute disease, although detailed field studies have been made only for ore.

The toxicity of beryllium oxide is variable, a finding that was first made in field studies (5). Under certain conditions concentrations of beryllium oxide as high as 30 milligrams per cubic meter were associated with no acute cases among exposed employees, whereas in another plant, 4 milligrams per cubic meter was productive of a very high incidence of acute disease with a high fatality rate. Further study disclosed that the toxicity of beryllium oxide, like its chemical reactivity, is markedly influenced by the calcining temperature. Oxides produced above approximately 2800° F possess relatively low specific surface area and apparently do not possess acute toxicity. This has been confirmed by laboratory experiment which showed that animals developed acute beryllium poisoning when exposed to the low fired oxide but were able to tolerate high exposures to oxides formed at higher temperatures (7).

The size of the beryllium oxide particles formed in the oxidation process increases with calcining temperature. Agglomeration of the very small particles formed by firing at low temperatures may produce particulate

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whose dimensions are of the same order as the diameter of the dust of high fired oxide. However, these agglomerates will have a higher specific surface area and will be more reactive.

Acute pneumonitis has occurred from single accidental exposures to beryllium. In this respect, the acute toxicity of the beryllium compounds is again similar to the irritant gases like nitrous fumes and phosgene which also have the ability to produce pulmonary edema following brief but excessive exposure. In one episode for which detailed air analysis are available (5), a 20 minute exposure to beryllium fluoride produced acute disease in three individuals. The exposure which followed the sudden accidental release of beryllium fluoride was in the range of 400 to 650 micrograms of beryllium per cubic meter of air. The total amount of beryllium inhaled by the affected individuals could not have greatly exceeded 50 micrograms. Compared with the dose of other irritants required to produce equivalent pulmonary injury, this is an exceedingly small amount. To produce injury by phosgene in a comparable period of time one would have to inhale approximately 50 milligrams!

Studies of the occurrence of acute disease in beryllium production plants revealed that all cases could be associated with concentrations of soluble beryllium compounds in excess of 100 micrograms per cubic meter. When the concentrations exceeded 1 milligram per cubic meter, cases were persistently produced among almost all personnel.

Berylliosis

It has been shown that the levels of beryllium exposure capable of producing acute disease are of a low order in comparison with the doses of other chemical irritants which produce acute pneumonitis. The dose

which may produce berylliosis, chronic beryllium poisoning, is of yet a lower order.

There is now essential unanimity within the medical profession that berylliosis is a distinct disease distinguishable from other lung diseases by a unique complex of clinical roentgenographic, histologic and biochemical characteristics. Although much has been learned about this disease, the causative factors and the mechanisms by which injury is produced remain obscure. A number of striking features characterize the disease. Severe cases have been known to develop from exposures which would be considered inconsequential if we were dealing with even the most virulent of other toxic chemicals. For example, a concentration of lead of 150 micrograms per cubic meter is considered a safe exposure to lead, but chronic beryllium poisoning has been caused by concentrations of only a few micrograms per cubic meter. Because of the marked difference in atomic weights, it may be unfair to compare the toxicity of beryllium with that of a heavy metal such as lead. On a molar basis approximately six micrograms of beryllium are equivalent to 150 micrograms of lead. When considered in this way, the toxicity of beryllium is less striking although it remains somewhat higher than lead.

The extraordinary toxicity of beryllium is emphasized by the occurrence of berylliosis among residents in the vicinity of at least two plants in which beryllium was processed. Although instances of acute intoxication in the vicinity of industrial plants are known and are illustrated by such incidents as the one which occurred in the smog at Donora, Pennsylvania, the occurrence of berylliosis is apparently unique among the chronic diseases.

Despite the enormous tonnages of lead, arsenic and mercury processed in industry, and in spite of the marked capacity of these metals to produce disease among workers, there are no known cases of community intoxication attributable to air pollution from these sources.

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In one community a total of 16 cases including five fatalities has been reported. These cases occurred within $3/4$ of a mile from a plant producing beryllium compounds from the ore (8). None of these individuals had a history of occupational exposure to beryllium and their only exposure was therefore to the contaminated atmosphere surrounding the plant. When these cases were reported, an effort was made to estimate the concentration of beryllium to which people were exposed $3/4$ of a mile from the plant. This concentration was important because the absence of cases beyond $3/4$ mile suggested that the concentration at this distance could be used as the maximum permissible concentration for continuous (168 hours per week) exposure to beryllium.

Air analysis in the vicinity of this plant during a period of normal operation indicated that the $3/4$ mile concentration was certainly less than 1 microgram per cubic meter of air and probably ranged between .01 and .1 microgram per cubic meter of air.

One ordinarily expects that the incidence of disease will vary directly with the dose administered. The dose-response curve for a toxic metal is usually a sigmoid in which no injury is observed below the minimum toxic dose. As one increases the dose, the frequency of injury increases until it reaches 100% of the exposed population. Although complete data are lacking, there is evidence that the dose-response relationships in the case of chronic beryllium poisoning do not follow the pattern to which other chemical poisons conform. In the neighborhood

~~cases~~ described above, the usual dose-response relationships existed.

Individuals living closer to the plant were exposed to higher concentrations than those living at greater distances and it was closest to the plant that the greatest frequency of cases occurred. However, it is startling that relatively few individuals who worked in this beryllium plant developed chronic disease.

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Within the first quarter of a mile of the plant, where the concentrations of beryllium were on the order of 1 microgram per cubic meter, the incidence of berylliosis was approximately 1%. Within the plant, however, employees were exposed to concentrations as much as a thousand times greater but only 9 cases of berylliosis have developed in 15 years among the 1700 people who were at one time or another employed. This incidence of approximately .5% is lower than the incidence among the first quarter of a mile residents despite the fact that the inplant concentrations were higher by possibly three orders of magnitude. Another puzzling feature of the in-plant cases is that the nine individuals who developed berylliosis were employed only for relatively brief periods, i.e., less than 4 months. One ordinarily expects to find chronic occupational diseases among those whose histories indicate the longest exposure. The reverse seems true in this plant.

Berylliosis is peculiar in that individuals may develop the disease after very long latent periods. Cases have occurred up to 11 years after cessation of exposure.

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The small amounts of beryllium which are capable of producing disease are reflected in the exceedingly minute deposits of beryllium in affected tissues. In some fatal cases less than .1 microgram of beryllium was found to be present in a whole lung (4). Moreover, there appears to be no relationship between the degree of pathology and the amount of beryllium found in the tissues at autopsy.

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The unusual characteristics of this disease have led some investigators to abandon the hypothesis that berylliosis is a simple chemical intoxication and seek an explanation for the disease in the theory that a sensitizing reaction is involved (3). With this hypothesis, it is possible to explain many of the peculiarities of this disease.

One can demonstrate quite readily that beryllium oxide exposure was the specific compound involved in most of the reported cases of berylliosis, but this in itself is not conclusive because beryllium oxide is the compound most used industrially. It is the only compound of beryllium which has found application in the ceramics industry. In the machining of beryllium metal, the metallic dust particles form oxide coatings; and in beryllium copper founding, beryllium oxide is a constituent of the fume. There are, however, two important industries, beryllium extraction and fluorescent lamp manufacturing, in which beryllium compounds other than the oxide are widely used. The spotty occurrence of berylliosis in these industries may possibly be explained by the hypothesis that beryllium oxide is the causative agent of this disease.

In the beryllium extraction plants, the principal exposures are to the dust and mist of beryl, beryllium sulfate and beryllium fluoride. Only a small minority of the employees are exposed to beryllium oxide.

In one plant for which data over a 17 year period are available, all of the known cases of berylliosis occurred in individuals who had been exposed to beryllium oxide. This is true for the nine cases which have developed among approximately 1700 employees who have at one time or another worked in this plant. As noted earlier, this was the plant near ^{DOE ARCHIVES} which a number of residents developed berylliosis, presumably from atmospheric contamination. It is noteworthy that the principal beryllium compound emitted from this plant was beryllium oxide in the fumes emitted from the beryllium copper furnaces. Exposure to this fume existed within the plant as well but relatively few employees were involved by reason of its physical isolation.

In another beryllium production plant there have been no cases of berylliosis among approximately 200 individuals who have been employed during the 12 years this plant has been in operation. The complete absence of cases is not surprising when one considers the frequency of disease in the larger of these two plants where comparable exposures existed.

The largest single series of cases occurred in a fluorescent lamp plant in Massachusetts. It has been observed that all of the individuals who developed berylliosis were exposed to the phosphors used prior to 1942, during which time the beryllium oxide content was about 12%. (6)(9) The beryllium content of phosphors in subsequent use was restricted to about 2%, a change which was stimulated by the wartime requirements for beryllium. No cases have developed among employees who were hired subsequent to early 1942 when the 2% beryllium oxide phosphors were adopted.

It has been shown by X-ray diffraction studies (10) that the solubility of beryllium oxide in the zinc silicate host lattice is limited to 30 molar percent for phosphors having up to 1% manganese. The solubility of beryllium oxide decreases with increases in manganese content. The limited solubility of beryllium oxide in zinc-beryllium - ^{DOE ARCHIVES} manganese phosphors is thus no greater than 6.8% and would be considerably lower in the case of a phosphor containing magnesium or more than 1% manganese. The presence of free beryllia is thus a distinct possibility in phosphors in which the beryllium oxide in the original mix exceeds its limit of solid solution in zinc silicate. It appears reasonable to conclude, tentatively, that berylliosis in the fluorescent lamp industry was produced by free beryllium oxide present as an excess in the phosphor.

Unfortunately, this question as well as many others cannot be answered readily by animal experimentation. Berylliosis, like certain other human diseases, has not as yet been produced in laboratory animals despite many systematic attempts during the past 12 years. Within the past few years, some progress has been made by one investigator (11) who succeeded in producing the typical lesion of beryllium poisoning in porcine skin by implanting a phosphor containing 33% beryllium oxide. Free beryllium oxide was probably present in this phosphor. However, implantations of pure beryllium oxide and beryllium metal did not produce the characteristic lesion seen in beryllium workers. This experiment would seem to contradict the hypothesis that beryllium oxide is the specific causative factor. Only further studies of this type will tell.

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Another illustration of the unusual toxicity of beryllium is found in the occurrence of berylliosis in individuals residing in the same household with beryllium workers. A typical case is that of a housewife whose only exposure to beryllium occurred in the washing of her husband's work clothes (8). No other industrial toxin is known to have produced poisoning in this way. During the investigation of this case, work garments from the plant where her husband was employed were laundered in a manner simulating the laundering procedure she used in her home. It was concluded on the basis of this study, that the laundering of contaminated work clothes results in the inhalation of about 17 micrograms of beryllium. While this is a very meager exposure, it is certainly of a magnitude of hygienic significance when compared to other data given above.

Skin Manifestations

The soluble beryllium compounds have the characteristics of both primary irritants and sensitizing agents. In one large plant over 25% of the new employees exposed to BeF_2 developed a dermatitis within the first few weeks of exposure. BeF_2 when accidentally implanted into an abrasion or laceration of the skin may produce an ulcer which will persist until the beryllium is removed.

Fluorescent lamp phosphors and metallic beryllium, when implanted accidentally into the skin, have been known to produce a benign tumor at the site of the implant. The onset usually occurs from 2 to 4 months after initial implantation frequently following apparent healing of the wound. The lesion will persist until the implant is removed. Pathologically, this lesion is a granuloma of the type seen in the lungs of beryllium workers.

As noted earlier, it has been reproduced experimentally in the pig.

Prevention of Beryllium Poisoning

The above information provides a background with which it is possible to develop the basic procedures by which the various manifestations of beryllium poisoning can be prevented. The procedures adopted will, of course, depend on the physical or chemical properties of specific beryllium compounds or alloys, as well as the manner in which they are to be handled.

The skin reactions to beryllium are a low order hazard in comparison with the danger of lung injury, and this presentation will therefore not be concerned in detail with this problem. Dermatitis has been a serious problem only where the acid salts of beryllium (particularly BeF_2 and $(\text{NH}_4)_2\text{BeF}_4$) are handled. The strict standards of personal and process hygiene that are necessary to prevent lung injury to beryllium compounds will tend to minimize the dermatitis problem except in a small percentage of individuals in whom the sensitizing properties of the soluble beryllium compounds may be evident. Employee education and good medical supervision will prevent skin ulcers produced by implantation of the soluble and insoluble compounds of beryllium.

The forms of beryllium poisoning which characterize beryllium as an insidiously toxic substance are the lung diseases. As is true with other occupational diseases of the lung, inhalation of the beryllium compounds in excessive amounts is the primary factor in the causation of the disease. To produce injury the beryllium compound must be inhaled in amounts which exceed an individual's tolerance for the substance. Thus, in keeping with industrial hygiene practice as it relates to the

control of many other industrial diseases, the prevention of beryllium diseases of the lung depends on the control of atmospheric contamination.

Limits of Permissible Atmospheric Contamination

The U. S. Atomic Energy Commission which has been the largest user of beryllium compounds in recent years has established an advisory committee whose principal function is to recommend the maximum permissible concentrations of atmospheric beryllium. Beginning in 1949 it has been the policy of this committee to recommend values which are automatically revoked at the end of a 12-month period at which time the subject is reevaluated in the light of new information. However, the recommended values appear quite stable in view of the fact that there have been no changes since the subject was first considered by the committee in 1949. The recommendations of the committee are:

1. The in-plant atmospheric concentration of beryllium should not exceed 2 micrograms per cubic meter as an average concentration throughout an 8-hour day.
2. Even though the daily average might be within the limits of Recommendation #1, no personnel should be exposed to a concentration greater than 25 micrograms per cubic meter for any period of time, however short.
3. In the neighborhood of a plant handling beryllium compounds, the average monthly concentration should not exceed 0.01 microgram per cubic meter.

Those who are not completely familiar with the epidemiology of beryllium poisoning are startled by the fact that three separate values are recommended. This is unique in industrial hygiene practice

where a single maximum permissible concentration is ordinarily used in the control of a toxic substance.

The value of two micrograms per cubic meter is the only one of the three values that is not based on field study. It was recommended by

As noted above, 2 micrograms per cubic meter is recommended as the maximum daily average exposure. Thus, an individual could be exposed to 4 micrograms per cubic meter for four hours a day and, if he were not exposed to beryllium for the remaining 4 hours, his average exposure for the 8 hour period would be 2 micrograms per cubic meter, which is permissible. **DOE ARCHIVES** Similarly, an individual could properly be exposed for higher amounts for correspondingly briefer periods. The maximum exposure should not exceed 25 micrograms per cubic meter because higher concentrations even for brief periods may produce acute disease. As noted earlier, one episode which involved 20 minutes exposure to about 500 micrograms per cubic meter produced acute disease in three individuals.

It is possible that Recommendation #2 will eventually be waived for certain beryllium compounds. There is every indication that beryl is not capable of producing acute disease. To an only slightly lesser extent there is evidence that the high fired grades of beryllium oxide are not acute hazards. Moreover, for those compounds which can produce acute disease, it is quite likely that the hazardous concentrations will vary from compound to compound. However, the evidence is not sufficiently convincing to justify differentiating between beryllium compounds and for the time being one should continue to apply this recommendation regardless of the form in which the beryllium exists.

Recommendation #3 that the average monthly concentration not exceed .01 micrograms per cubic meter in the neighborhood of a plant handling beryllium compounds is based on actual studies in a community where air pollution was responsible for berylliosis among residents near a beryllium producing plant. This value probably has a safety factor of about 10.

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Methods of Air Analysis

Air analysis for beryllium is the basic tool with which an exposure can be evaluated. Careful sampling procedures will provide quantitative exposure data, will identify the need for control procedures, and will assess the value of hygienic procedures already in effect.

Air analysis for beryllium is performed by drawing a known volume of air through a suitable filter medium and then analyzing for beryllium by either spectrographic or fluorometric methods.

A useful assembly for obtaining the air sample is shown in Figure 1. (12) This unit contains a compact motor **DOE ARCHIVES** blower assembly which draws one cubic foot per minute through a 1-1/8" filter paper. Whatman #41 has been found to provide good retention of the dusts, fumes and mists commonly encountered and has the advantage of being an ashless paper. Air flow through this device is controlled by a needle valve and measured by a rotameter which is part of the assembly. If the concentration of beryllium is so low as to require a larger sample, the apparatus shown in Figure 2 can be used. This unit is capable of sampling at 20 cfm through a 4" Whatman #41 disc. Higher rates of flow can be obtained by using other sampling media.

In general, air samples should be collected in the breathing zones of individuals performing potentially dusty occupations and in the general workroom areas. These can be composited to yield a measure of the general level of contamination. Careful air analysis will not only reveal the troublesome operations, which are sometimes not visibly dusty, but will also provide a record of the exposure of an individual worker. Table I illustrates the method by which one can estimate the daily average

TABLE I

Method of Estimating Average Daily Exposure

Job: Sintering Furnace Operator

Operation or Operating Area	Time Per Opera. (Min)	Opera. Per Shift	Time Per Shift(T) (Min)	No. of Samp- les	Concen- tration(C) $\mu\text{g}/\text{m}^3$ (Average)	Avg. Con Times Total Ti (TIC)
*Placing Compact in Furnace	6	1/3	2	4	5.3	10
*Dislodging Graphite Blocks	90	1/3	30	3	1.5	45
*Removing Compact & Transferring to De- contamination Room	6	1/3	2	2	3.4	6.8
*Tearing Down Compact	9	1/3	3	3	15	45
*Vacuuming & Washing Compact, Removing Com- pact from Decontamina- tion Room to Cooling Room	8.5	1/3	2.8	4	15	42
*Loading Blender	40	1/2	20	3	11.7	234
*Placing Blender on Roller & Cleaning Hood	6	1/2	3	3	20	60
*Loading Compact	166	1/2	83	4	5.4	450
*Taking Sample from Compact	.67	1/2	.3	3	75	22
*Leveling Compact & Placing Cover on	13	1/2	6.5	5	1.5	9.8
*Putting Plunger in Horizontal Sintering Furnace Die	9	1/3	3	1	11	33
GA Sintering Furnace Area			284.4	3	0.3	85.3
GA Lunch Room			40	4	1.5	60
GA Locker Room			28	6	0.9	25.2

 $\Sigma T = 508$ $\Sigma (T \times C) = 1090$

$$\Sigma \frac{(T \times C)}{\Sigma (T)} = 2.1 \mu\text{gm Be}/\text{m}^3 \text{ (Average Exposure)}$$

* These samples collected in breathing zone of operator.

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exposure to an individual. This requires a careful time study of the individual's work habits and is useful in characterizing the exposure associated with many repetitive operations involving toxic substances.

Methods of Chemical Analysis

A number of satisfactory spectrographic, fluorometric, and colorimetric procedures for the determination of micro-quantities of beryllium have been published.(13)(14)(15) It is important to note that these air samples frequently involve as little as 10^{-8} grams of beryllium and that scrupulous care is therefore necessary to avoid contamination of the samples either in collection or analysis. Because of this inherent difficulty, many laboratories associated with the chemical or metallurgical processing of beryllium are unable to undertake their own analysis. However, with suitable precautions this can be accomplished. This is evidenced by one large beryllium production plant which has successfully undertaken an air analysis program.

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Design of Working Facilities

The specific precautions which should be observed when beryllium is handled will depend on the type of work contemplated. There are laboratory operations in which gram amounts of beryllium compounds can be safely handled in a well operated and properly designed chemical hood. On the other hand, other laboratory procedures involving less than a gram of beryllium may require elaborate precautions if the work is such that the dispersion of beryllium as a dust, mist or fume may occur. Very few, if any, precautions are necessary for many machining operations involving beryllium copper alloy. This contrasts sharply with the extent to which precautions are required in the founding of the master alloy or in industrial scale operations involving the extraction and refining of beryllium.

The area in which a beryllium exposure exists frequently can be limited by physical separation of the work. One or two employees fabricating beryllia crucibles can contaminate the workroom air breathed by others who are not directly concerned with their work. Here, as in many other examples which could be cited, is the opportunity to reduce the extent of the exposure by providing a separate room in which this operation can be performed.

Beryllium, like other toxic materials should be handled in a work environment which is conducive to good general housekeeping. There is much to be said for the advantages of a well illuminated, freshly painted, neatly arranged workroom. Clean working practices can be more effectively encouraged when one starts with a workroom that is of itself properly designed.

Vacuum cleaning of floors and other surfaces is an important part of a program to control dust exposure. In just a few minutes, dry sweeping can produce dangerous exposures in a workroom where the beryllium is otherwise safely handled. Wet sweeping is less dusty but in practice is difficult to supervise. Vacuum cleaning provides a thoroughly satisfactory method of removing accumulations of dust but certain precautions should be observed.

The type of vacuum equipment will vary from a household vacuum cleaner which may be adequate for a small laboratory to a central heavy duty system that costs tens of thousands of dollars and services relatively large industrial areas. In some cases the method of choice will be intermediate and involve a portable industrial type vacuum cleaner. It is important, regardless of the kind of unit which is adopted, that the vacuum cleaner

be discharged to outside air. This is not only to safeguard against sudden tears in the filter medium but also recognizes the fact that even under the best of operating conditions the discharge air, although relatively clean, may be hazardous for breathing purposes. In the small laboratory, it is possible to pass the discharge air through a flexible hose terminating in a laboratory hood. It is important even though the discharge from the vacuum system passes to outside air, that appropriate consideration be given the possibility of atmospheric pollution exterior to the plant. This may or may not be a problem depending on the amount of beryllium being discharged and the subject will be discussed in more detail later.

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Ventilation of Beryllium Processing

Well designed local exhaust ventilation is frequently the most essential part of a program to minimize the hazard from beryllium. Local exhaust ventilation provides a method of capturing an aerosol (dust fume or mist) as close as possible to its source of dispersion, and conveying the contaminant through a system of ducts to outdoor air. Where necessary, means must be provided for removing the contaminant in an aircleaning device prior to outdoor discharge of the air.

Figure 1 illustrates the design of a ventilated enclosure which permits manipulation of moderate amounts of toxic materials.

A number of principles are illustrated by this simple enclosure. The air supply required for ventilation is minimized by reducing the access area in front of the hood. In order to control dust dispersed within this enclosure, ventilation at a rate of 150 to 200 cubic feet per minute per square foot of hood opening is required. Minimizing the access area improves the performance of the hood by reducing the effects of cross drafts

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and the volume of air required for ventilation. Therefore the cost of the ventilation system is reduced in proportion to a reduction in the area of the hood opening. Good visibility is provided by large windows and installation of illumination within the enclosure. The latter is particularly important as it encourages the proper use of such a hood.

Figures 2 and 3 illustrate the manner of ventilating a lathe in which metallic beryllium is machined. In both methods a high velocity (2000 to 3000 ft. per minute) exhaust orifice at the end of a 2" flexible hose is mounted on the tool post. In this way most of the dust formed at the point of operation is properly controlled. Dust which escapes the influence of this high velocity exhaust nozzle is captured by a secondary hood of which 2 types are shown. Note that in Figure 3 ventilation economy is achieved by providing plastic shields which reduce the volume of air required for effective ventilation.

A method of ventilating a rotary kiln is shown in Figure 4. Annular enclosures at both ends of the kiln provide control over dusts and fumes which would otherwise be dispersed into the workroom air during normal operation. The ventilation hood at the discharge end of the kiln is integral with the discharge shoot for which ventilation is to be provided.

The field of local exhaust ventilation is a place in which it is advisable to obtain expert design advice. All too often a system fails completely or is over-designed. The latter very frequently involves unnecessary equipment and installation costs. Of equal importance but not often appreciated is the excessive cost of heat which must be supplied to make up air in a ventilated area in the winter time. The operation illustrated in Figure 1 is properly exhausted with a flow 300 cubic feet per minute. By other methods which provide less satisfactory protection, from 1000 to 2000 cubic feet per minute might be required.

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Air Cleaning Equipment

The occurrence of berylliosis among residents in the vicinity of plants using beryllium has emphasized the need to guard against excessive contamination of gaseous discharges from plants and laboratories using beryllium. Of perhaps equal importance is the danger of contaminated air being recirculated back into the workrooms. This can be caused by down drafts carrying virtually undiluted discharge into open windows, or by poor planning which places the intake of air supply systems in proximity to the discharge from the process ventilation system.

Although the problem of air pollution under some conditions is a very real one when beryllium is being handled, it should be emphasized that many beryllium operations can be ventilated without the need for aircleaning systems. If the beryllium is being discharged in amounts not exceeding a few grams per day, it is likely that no requirement for air cleaning exists unless the point of discharge is poorly located. The ventilation system should preferably discharge at a point well above the roof level. The maximum concentration at street level will occur at approximately 10 to 15 stack heights from the point of discharge and the magnitude of this concentration will decrease with the square of the stack height.

Some idea of the quantitative relationships involved in the dilution of stack effluents can be obtained from measurements in the vicinity of one plant where careful studies were made. This plant was discharging approximately 5 lbs. of beryllium per day from a stack located at 35 feet above the street level. This discharge produced a concentration of about 0.2 micrograms per cubic meter 350 feet from the stack. Under similar conditions therefore, it would be possible to discharge one quarter

pound of beryllium per day without exceeding the maximum permissible concentration for outdoor air. As a guide to estimating the order of magnitude of the concentrations to be expected in the vicinity of effluents containing toxic materials a number of techniques utilizing the theory of turbulent diffusion are available.(16)

The choice of aircleaning equipment will depend on the physical and chemical form in which the beryllium exists, the air temperature and whether or not corrosive co-contaminants are present. The choice should also be influenced by the required collection efficiency.

A well operated cloth bag type collector of the type available from a number of manufacturers will provide collection efficiencies of over 99% and are satisfactory for many requirements. Similar efficiencies can be achieved with a variety of fibre glass, cellulose, and other fibrous media which are commercially available in the form of replaceable mats. However, they are only practical for light dust loadings. Electrostatic precipitators of the Cottrel type are expensive in comparison with cloth or the glass filters and offer no appreciable advantages for the relatively low capacities required in ventilating a beryllium processing operation.

It is sometimes desirable to precede any of the above type dust collectors with a centrifugal separator (such as Cyclone) in order to remove coarse material, thereby minimizing the load on the high efficiency separator. For cleaning air containing mists, a variety of types of equipment are available. Here in particular the question of corrosion is frequently an important factor in determining a choice of equipment. Packed towers, Venturi scrubbers and other types of wet collectors have been used successfully. The choice of balancing the cost of the equipment against

the expected performance is frequently a difficult problem, particularly since the latter is often unpredictable. For this reason, it is desirable to seek expert guidance in the selection of equipment and to anticipate the need for careful performance tests and possible alterations to the system after the initial installation is made.

Some General Hygienic Precautions

Laundry: As discussed previously, the toxicity of beryllium is such that at least one well documented case is known where the only contact with beryllium was in the washing of work garments. **DOE ARCHIVES** It is therefore desirable to provide "on site" laundry facilities if at all possible. Where only a few employees are involved, home type laundry equipment is sufficient.

Special Problems in Maintenance Procedures

The most difficult operations to control are those having to do with plant and laboratory maintenance. Innumerable opportunities for exposure exist when beryllium contaminated equipment is being dismantled or repaired. In a plant where all routine work is well controlled it is possible for an individual with a welding torch to produce a lethal exposure in a few minutes. It is desirable that all maintenance work be carefully supervised by an individual who is conversant with the hazards of beryllium poisoning and can competently improvise protective procedures to suit the needs of the job.

Respirators: A good principle is that respirators should not be prescribed for a routine operation but rather for an unusual short term exposure where positive methods of dust control are unfeasible for mechanical or economic reasons.

Where the concentrations of a toxic substance are so high that brief exposure may be lethal, even an air supplied respirator should be avoided.

At least one fatality from acute beryllium poisoning is known to have occurred where an individual working in a cloud of beryllium chloride had to remove his respirator because the air supply lines became fouled. As an emergency device, for use where acute beryllium poisoning is a possibility, a self-contained breathing apparatus should be used. In this equipment, an air supply for a limited period is assured from either a tank of oxygen carried by the wearer or by re-circulation and chemical treatment of the expired breath.

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For less hazardous exposures a variety of respiratory protection is available. Referring to Table I it will be noted that the operation of "removing the sample from a compact" involves (a brief 1/2 minute) exposure to 75 micrograms per cubic meter. For exposure of this kind a toxic dust respirator would be satisfactory.

SUMMARY

Beryllium is a highly toxic material capable of producing a variety of diseases in humans. Acute pneumonitis and berylliosis are the principal effects which must be guarded against.

The principal methods of control are those which limit the concentration of beryllium in air to safe levels. Maximum permissible concentrations for prevention of acute disease and berylliosis have been tentatively recommended by the U. S. Atomic Energy Commission to its contractors and to date have been successful in the control of these lung diseases.

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