The recent volcanic activity started with a relatively minor earthquake on March 20, 1980. This was followed by avalanches several days later and a plume of steam on March 27. The volcanic activity continued over the next few weeks with earthquakes, ash eruptions and blocks of ice being hurled above the rim of the crater formed when the crust blew on March 28. Despite the ominous bulging of the north side, there was little to suggest that the mountain would erupt with such incredible force. This it did on May 18 blowing 1,300 feet off the top of the mountain and leaving a crater mile wide and a mile from top to bottom.

With the eruption, the plume of ash was thrown to over 70,000 feet, and a pyroclastic flow, creating its own shock wave, stripped trees of foliage and laid them flat—virtually obliterating a large tract of virgin forest. The devastation was immediate and awesome. The cloud of ash cut a broad swath across the continent, then across the world. Worst hit by the ashfall were the cities of Yakima, Spokane, Moses Lake and Ritzville in eastern Washington. Two subsequent major eruptions on May 25 and June 12 resulted in a significant ashfall which affected Portland and Vancouver, Washington major metropolitan areas with a population of over a million.

To those in the ashfall areas, the immediate problem was how to get rid of the ash. It proved to be extraordinarily tenacious and did not wash off any surfaces readily. Furthermore, it seemed to be irritating to mucous membranes. Its abrasiveness proved very destructive to machinery and the ubiquitous dust, with its high iron content, proved a real hazard for electronic equipment. But was it in fact any more than an irritation? Answering this question has been very difficult. First, there is inadequate information about the composition of the ash, specifically, just how much free or crystalline silica does it contain? Second, are there potentially dangerous minerals adhering to the particles? Third, what is the size range of the particles and is there, in fact, an appreciable proportion in the respirable range? Also, are the particles radioactive? What is the hazard to the young and old and to those with pre-existing heart or lung disease?

These and similar questions have been the immediate issues faced by officials responsible for protecting the health of the public. Near hysteria and panic engendered by irresponsible pronouncements in the media about the possible health effects of the ashfall have been hard to counteract because we are dealing with so many unknowns and uncertainties that making scientifically valid guesses about risk is virtually impossible.

The purpose of this extemporaneous volcano session is to review the available information about the nature and composition of the Mt. St. Helens ash and by putting this together with the large body of data on the effects of similar exposures in occupational settings in man and of simulated exposures in animal models, arrive at a reasonable and scientifically valid guess as to the potential health effects, both immediate and long term.

Risk Assessment in Volcano Ash Exposure

*Hans Weill, M.D., F.C.C.P.**

Following the eruption of the volcano Mt. St. Helens in May of 1980, early concern for the exposed population centered around the possibility that the silica content of the ash would pose a risk which included, but was not limited to, silicosis. In my early discussions with public health officials and others in eastern Washington state, I indicated that to my knowledge the disease silicosis has been recognized only as the result of *occupational* exposure.

Determinants of Risk

I reviewed the determinants of a silicosis risk, which include the proportion of the dust which is free *crystalline* silica. There was early confusion concerning total silicon dioxide (SiO_2) content which, of course, would include the silicates and amorphous silica. It is likely that much of this early fear resulted from the wide quoting of high percentages for silicon dioxide.

A well recognized risk factor is *particle size distribution* with the respirable (less than 5 micron) sized particles being those of greatest concern. *Airborne concentration* of the respirable silica dust is of critical importance and in the occupational setting undoubtedly determines not only whether and to what extent the risk of silicosis exists, but also the form of the disease, from chronic, slowly progressive through accelerated and to acute silicosis. The *length of exposure* is a major determinant and even in the most heavily exposed workers, such as the silicotic sandblasters whom we have been following for many years, an exposure of at least one to three years is required. Finally, *respiratory protection* undoubtedly influences the dose of respirable silica particles inhaled, but complete protection is probably unobtainable even with air-supplied hoods.

Early Information on Exposure

How, then, can we apply these risk factors to what we know at this early point concerning the nature of the volcano ash in the Pacific Northwest? Although a subject of substantial controversy, it seems likely that up to 5 or 6 percent of the dust (by weight) is free crystalline silica. This percentage would be at the very lowest of the spectrum of proportions of crystalline silica in occupational settings which might produce silicosis over a working lifetime of exposure. The particle size distribution appeared to have been favorable for deep pulmonary penetration of dust. Also, airborne concentrations were substantial (in comparison with known occupational hazardous exposures) although

*Professor of Medicine, Tulane University School of Medicine, New Orleans.
Reprint requests: Dr. Weill, 1700 Perdido Street, New Orleans 70112

these levels varied widely by geographic area and were high for very short periods of time.

So far, analysis of the risk factors which I enumerated seems not to favor a sanguine view concerning exposure to volcano ash. The good news presumably will be in consideration of the length of exposure. Assuming the data discussed above, the length of exposure would certainly have to be measured in *decades, not days,* for there to be a measurable risk for the development of silicosis in this exposed population. Even with repeated eruptions, such continuing regular exposures must be considered extremely unlikely. It is this probability that does indeed lead to the optimistic judgment that a negligible or nonexistent risk of silicosis exists in this population. While it is true that for some time there will be resuspension of settled ash and certain potentially "high risk" populations might include lumbering and maintenance workers, the length of exposure will certainly still be far short of that likely to be associated with a silicosis risk.

Potential Respiratory Responses

Are there other potential respiratory effects? Acute or long-term airways responses with or without pre-existing disease have been discussed. It is not unlikely that individuals with bronchial hyperresponsiveness (*eg,* asthmatics) might have transitory irritative bronchoconstriction, and perhaps a high proportion of heavily exposed individuals had some degree of irritant respiratory complaints. Long-term adverse airways effects are unexpected. Might there be an adverse parenchymal effect resulting from exposure to silicates (the feldspar, plagioclase, an aluminum silicate, is an important constituent of the volcano ash)? Evidence that nonfibrous silicates cause respiratory disease, such as pulmonary fibrosis, is extremely weak and I would dismiss this as a probability. It is of interest that no substantial fiber content has been identified in the volcano ash. The same optimistic conclusion for the silicates could be extended to amorphous silica or glass. Finally, it has even been suggested that the risk of respiratory cancer may be associated with this exposure and again with the recognized constituents in the ash this fear seems totally unfounded.

Initial Data on Biologic Response

What was the early post-eruption experience of exposed humans and animals? While there may have been an increase in emergency room visits in areas heavily exposed, primarily for minor irritative symptoms, hospitalizations for significant respiratory disease did not seem to be associated with this exposure. Autopsies of animals dying in the post-eruption period have failed to demonstrate significant adverse respiratory tissue or cellular responses. It was suggested that human autopsies also be used to monitor potential respiratory abnormalities associated with pulmonary deposition of volcano particles. Early *in vitro* studies of the volcano ash (*eg,* macrophage toxicity) have shown that while some minimal effect is at times demonstrable, this is far from the effect expected from silica and consistent with a dust containing the small crystalline silica composition indicated.

Public Health Approach

Can we then dismiss volcano ash exposure as irrelevant as a public health problem? It seems to be more judicious to reassure the exposed population while monitoring some high risk groups and initiating inhalation studies using volcano ash in the animal model. Additionally, temporal measurements of airborne particle concentrations and particle size distributions (as settled dust is resuspended) would be useful in further evaluating the risk of certain occupational groups such as foresters who may have somewhat prolonged exposure. Finally, a unique opportunity to study the potential biologic effects of volcano ash exposure justifies a well-designed centralized approach since unexpected findings, perhaps nonrespiratory in nature, are always possible. Dr. Buist and colleagues at the University of Oregon are, in fact, embarking on a multicenter, multidisciplinary approach to longterm surveillance.