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Cancer Prevention and Competitive Risks

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THIS PAPER is divided into three parts. The first part deals with methods of cancer control now available to us. The second part deals with cancer prevention. The third deals with competitive risks for cancer and other important diseases.

There's an old saying, "Let not the best be the enemy of the good." For cancer, the best would be prevention and the next best would be a remedy as quick and effective as the miracle drugs in relation to some infectious diseases. Lacking these, the ability to save many lives by more laborious means is surely good. Discouragement over our slow progress toward the best has led some people to disparage the good which has been achieved. Therefore, I will start by outlining what has been accomplished by means of early detection and modern methods of surgery and radiation therapy.

The greatest accomplishment has been improvement in cure rates and reduction in death rates from uterine cancer. In 1900, Cullen[1] reported on end results of 140 consecutive cases of uterine cancer seen at the Johns Hopkins Hospital. Half of these cases were treated by hysterectomy while the remainder were inoperable because of the extent of the disease. Four (2.8%) of the 140 patients were alive without evidence of the disease five or more years after therapy while another eleven were alive and symptom-free for from ten months up to 3½ years after therapy. Thus the five-year cure rate for these 140 patients could not have been greater than 10.7% but may have been as low as 2.8%. Dr. Cullen commented that the number of cases coming too late for effective therapy was "appalling" and attributes the "distressing" results largely to this fact.

During the ensuing 30 years, conditions improved both because of earlier diagnosis and the introduction of the Wertheim operation together with the use of improved methods of radiation therapy. However, as late as 1931 Bloodgood[2] wrote ". . . cancer of the cervix is today predominantly a hopeless disease." Like Cullen, he emphasized the need for earlier diagnosis and commented: "Apparently both surgery and radiation have reached their limits." He also suggested that it might be possible to prevent cervical cancer by treating "the lesion which precedes cancer."

In contrast to the distressing conditions of some decades ago, the relative five-year survival rate for patients treated in the period 1955 to 1959 is reported to be about 60%[3] (and for cervical cancer, five-year cure rates are not far below five-year survival rates). For the United States as a whole, age standardized uterine cancer death rates declined from 27.5 per 100,000 females in 1930 to 11.6 in 1966.[4] There is evidence that this progress has been made by the combination of: (1) earlier detection, (2) more effective therapy, (3) prevention of invasive carcinoma of the cervix by detecting and treating carcinoma in situ of the cervix, and (4) treating women who have a high risk of developing uterine cancer (hysterectomy[5,6] being a frequent method of treatment of conditions other than cancer).

Now let us turn to the picture for cancer as a whole (ie, all sites combined).

We do not have records covering the entire population of the United States, but the Cancer Registry of Connecticut provides information on nearly every case of cancer diagnosed in that state since 1935. Of all cases occurring from 1935 to 1940, excluding leukemia and generalized lymphoma, 50.4% were localized, 23.6% had regional involvement, and only 18.5% had evidence of remote metastasis at the time of

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diagnosis.[7] Early diagnosis had been made in half the cases. For cases diagnosed in that period, the five-year survival rate was 25% overall; 19% for men, and 29% for women.

The picture improved in Connecticut during the ensuing years, and the five-year survival rate rose to 32% for cancer diagnosed between 1947 and 1951. Analysis of data from the Cancer Registry shows that the improvement during that period of time was attributable only to a slight degree to earlier diagnosis. It was mainly due to more effective therapy.

Since we have to wait for at least five years to evaluate success, the 1947-1951 figures reported above were not available until 1956. In that year I estimated how much further progress could possibly be achieved in improving survival rates by maximum use of the methods then available.[7] I made three assumptions: (1) great progress could be made in the early detection of cancer of certain sites such as the cervix uteri; (2) moderate progress could be made in the early detection of cancer of internal organs such as the stomach and lungs; and (3) most important, the results achieved by the average surgeon and radiologist could equal the best results achieved by some surgeons and radiologists according to reports released at the Third National Cancer Conference in 1956.[8]

Calculations based on these assumptions indicated that it was theoretically possible to raise the five-year survival rate to approximately 50% with methods available in 1947 to 1951.[7] This upper limit was based upon certain assumptions about the natural history of cancer, assumptions which may or may not be fully justified. Since that time further progress has been made, especially with respect to cancer of the uterus. However, this progress has been partly offset by the great increase in the incidence of lung cancer for which survivor rates remain low.

Now let us turn to cancer prevention. Approximately 28% of all male cancer deaths and 7% of all female cancer deaths are due to lung cancer and the death toll is mounting every year.[9] The great majority of these deaths are preventable by persuading people not to smoke cigarettes. Not only would this prevent lung cancer, it would also reduce death rates from coronary heart disease, stroke, aortic aneurysm, emphysema, and peptic ulcer, as well as from cancer of the buccal cavity, pharynx, larynx, and urinary bladder. This is a challenge to public health educators.

Other known and preventable causes of cancer are exposure to ionizing radiation, prolonged exposure to sunlight, and occupational exposure to certain types of dusts and chemicals. The latter are well known to industrial hygienists.

Clearly, with present knowledge we are in a position to achieve a significant reduction in cancer death rates by preventive measures. Equally clearly, the majority of cancers occurring today are not preventable in our present state of knowledge. However, I am optimistic for the future.

There are two reasons for my optimism. From experimental, histologic and epidemiologic evidence it is apparent that the occurrence of carcinoma—that is, an invasive neoplasm arising in epithelial tissue—is preceded by what may be called a premalignant state lasting for many years. During that period, there is a gradual progression of changes which often (though not always) eventuate in cancer. Presumably, cancer could be prevented by breaking the chain of events at any time during those years. Some recent evidence on this subject is of practical importance.

In a study[6] carried out in Toledo, Ohio, a large number of women were examined routinely for cancer. Each examination included a history, recording of complaints, pelvic examination by a physician, and examination of at least two cytologic smears. Women whose smears were negative and who showed no signs of uterine cancer when first examined were traced for many years thereafter.

The women were divided into two groups; those who on first examination complained of abnormal bleeding or discharge from the vagina and those without this complaint on first examination. During the next five years, the incidence rate of corpus and cervical uterine cancer in women without cancer on entry into the study was well over three times higher among women with initial complaints as among women without

such complaints. This difference persisted up to 14 years. It is hard to escape the conclusion that the complaints reflected a disease state which in many instances eventuated in uterine cancer. If we can determine the nature of this disease state and correct it, then it should be possible to interrupt the progression of events leading up to uterine cancer.

Some of the women in the Toledo study with abnormal bleeding or discharge had the progression of events interrupted by a hysterectomy for reasons other than cancer. Until such time as we can intervene by means less radical than hysterectomy, the finding that abnormal bleeding indentifies women with a high risk of uterine cancer should be of great value in early detection as usually defined. We should make intensive efforts to examine these high-risk women periodically.

A different type of evidence also leads me to optimism. For a long time we have know of agents called co-carcinogens. These alone do not cause cancer when applied to experimental animals; however, they greatly increase the potency of true carcinogenic agents. This suggests the possibility that many human cancers may be caused by a combination of factors, a co-carcinogenic factor plus an otherwise ineffectual amount of a carcinogenic agent.

Tentative and as yet inconclusive evidence on this has come from an epidemiologic study of asbestos insulation workers carried out by Selikoff et al.[10] The lung cancer death rate was eight times as high among the asbestos workers as among men in the general population. The majority of asbestos workers were cigarette smokers, but some were not. No lung cancer deaths have yet occurred among the asbestos workers who did not smoke cigarettes. By now, there should have been at least several such deaths among the nonsmokers if the effects of cigarette smoking and asbestos exposure were independent of each other. This suggests that asbestos dust acts as a powerful co-carcinogenic agent for the production of lung cancer among cigarette smokers.

A much larger study including all members of the asbestos workers union in the United States is now being carried out to check these results. However, the tentative findings lead me to turn my attention to combinations of factors rather than to single factors.

Now I will turn to the problem of competitive risks. It may be illustrated by a few examples.

In addition to bronchogenic carcinoma, two other diseases can result from exposure to asbestos dust: asbestosis and mesothelioma of the pleura and peritoneum.[11] These three diseases are in competition, as it were, to claim the life of a person exposed to asbestos dust. There is evidence that with extremely heavy occupational exposure, asbestosis develops quite rapidly and the individual is likely to die of this cause or secondary pulmonary infection long before he reaches the age at which cancer is likely to occur. In one experience—and this was an extremely bad occupational situation—over 50% of the workers died of asbestosis within two years. With far lighter exposures, such as those to which insulation workers are subjected, asbestosis progresses very slowly and is not likely to prove fatal for many years if at all. Some 20 years after initial exposure, lung cancer, and mesothelioma begin to appear and a large percentage of insulation workers die of these two diseases which are in competition with each other.

Experimentally, it has been found that cigarette smoking produces at least three different serious effects on dogs.[12,13] Extremely heavy smoking produces acute toxic effects which can result in death within a short time. The smoking of somewhat fewer cigarettes—that is, just below the level where acute toxic effects cause death—frequently results in thrombus formation in the right auricular appendix with pulmonary embolism resulting in death. At the same level of smoking, some dogs escape pulmonary embolism but emphysema develops and progresses to a fatal or near-fatal degree within 400 to 500 days. During this period of 400 to 500 days, progressive changes occur in the bronchial epithelium of the dog similar to the changes in the remaining bronchial epithelium of human cigarette smokers who die of lung cancer. Whether or not these changes represent carcinoma in situ is still controversial among pathologists. With longer exposure there le

sions might progress to invasive carcinoma. However, this is effectively precluded by one of the other two causes of death mentioned earlier; that is, the dogs with heavy exposure to cigarette smoke die before cancer could develop. Presumably, with somewhat lighter exposure, the dogs would live much longer. Under these conditions some might eventually die of lung cancer (but this has not yet been determined).

In human beings, cigarette smoking leads to greatly increased death rates from a multiplicity of diseases including lung cancer, cancer of several other sites, emphysema, coronary heart disease and stroke.[14-16] In autopsy studies, we have found that most cigarette smokers who die of lung cancer also have emphysema and a considerable degree of coronary atherosclerosis. These diseases are in competition to bring about death of the smoker. Early death from one of these diseases precludes later death or disability from any of the others.

Now, suppose that we develop a pill which would prevent one and only one of these effects of smoking, or suppose that cigarettes were altered in such a way that they were harmless in relation to one and only one of these diseases. The risk of smoking would be somewhat reduced but life expectancy would not be greatly extended. On the other hand, avoidance of cigarette smoking eliminates all risks involved and life expectancy is considerably extended. (There is a difference of about eight years in the life expectancy of men who smoke 40 or more cigarettes and the life expectancy of men who never smoked regularly.)[17]

The same type of situation seems to apply to other factors which increase the risk of death from the principal atherosclerotic diseases—coronary heart disease, stroke, and aortic aneurysm.[18] For example, death rates from all three of these diseases are far higher among obese people than those not obese, far higher among people who take no exercise than among people who take a little exercise, and far higher among heavy cigarette smokers than among people who never smoked regularly. This together with other evidence strongly suggests that the same set of etiological factors is responsible for the occurrence of these three diseases (which may in fact be different manifestations of a single underlying disease rather than three separate diseases). It is fortunate since it implies that steps taken to reduce the rate of occurrence of any one of the diseases would probably reduce the rate of occurrence of all three of them—and together they account for a large proportion of all deaths in the United States.

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Discussion

GEORGE B. HUTCHISON, MD, Chicago: Let me first speak about Dr. Hammond's initial comments relative to the effects of currently available therapy and what could be anticipated by optimal use of this therapy and proper distribution of medical facilities.

The data that Dr. Hammond has presented are a little difficult to grasp for the following reasons. He starts with some very bad results from some very early periods of time. The five-year survivors for this early period of time are "few and far between"—a difficult statistic to treat. In 1935 to 1940, he finds that the overall five-year survival figure for all malignancies is 25%. This apparently is to be taken as an improvement as compared with "few and far between." By 1947 to 1951, this figure rose to 32%. The difference of 25 versus 32 seems encouraging. Following 1951, it is recognized that one has to wait five years from the application of methods to see the five-year survival rate. The prediction is that this rate may rise substantially above 32%. Again, "may rise substantially" is a difficult statistic to grasp. Overall, we have a comparison between "few and far between" and "substantially above 32%."

The statistic used in all discussion up to this point is the five-year survival. This is the statistic most commonly quoted in assessing the effect of cancer therapy. It is a difficult statistic to handle for a number of reasons that have been discussed by many, including myself, in several papers.

The first difficulty with the five-year survival statistic is the problem of its use when early case finding is one of the activities expected to be effective. It is clear that early case finding gives an automatic improvement in five-year survival statistics, even if nothing is done as a result of early case finding.

Another difficulty with this statistic needs to be mentioned, though I am not at all sure that it is a potent factor. This is the problem of possible nonspecificity of early case finding detection activities. It is best illustrated by the possible hazard in evaluating Papanicolaou screening for cervical cancer whereby a number of the cases picked up and enumerated as malignancy by Papanicolaou screening may, in fact, be individuals that, if left alone, would never advance to what is recognized as invasive cervical cancer. That is, these two diagnostic procedures do not have a one to one correspondence. We have some evidence—I have never been entirely satisfied with the evidence, but there is a suggestion—that a substantial proportion, perhaps something on the order of 15% to 20%, of cases detected in Papanicolaou screening are not, in fact, cases that would progress to invasive cancer. If, then, these are thrown into five-year survival statistics, clearly you get an improved result simply because you are counting survival of a different disease or of a diluted category.

This is potentially effective in any early case finding activity. We have discussed it in a recent paper on mammographic screening for breast cancer. My personal feeling from our data at this point is that it probably has a negligible effect in mammographic screening. Lesions identified by mammography and verified by biopsy in fact essentially correspond with cases ultimately recognized as breast cancer. Our observations on this are small and early, and whether some nonspecificity will show up in mammographic screening I cannot say. I simply mention it as a factor that must always be held in mind in talking about and evaluating five-year survival as the statistical measure for cancer therapy effects.

Another difficulty that Dr. Hammond mentioned in using the all-sites-five-year survival in evaluating time trends is the fact that the components of this all-sites rate are changing over the years. Specifically, as Dr. Hammond clearly noted, the lung cancer component has risen in recent years while many other sites have remained stable and a few have fallen in incidence. This is obviously not a random rise as far as survival potential is concerned, because lung cancer is one of the worst sites in terms of mortality and survival.

One has a number of other statistics that can be looked at in evaluating trends in effects of cancer therapy. The general population-based mortality is a commonly used statistic, and this almost invariably leads one to a somewhat more pessimistic outlook than the five-year survival figures. I think this relates to the factors I have just mentioned in interpreting five-year survivals.

Another figure commonly used which seems

to be increasingly popular is the so-called "cure rate." Cure rates in previous periods have often been synonymous with five-year survival, but more recently a number of analytic statistical devices have been used to attempt to say from a given group of cancer survivors which shall be called cured. The most common interpretation of this cure concept is to say that one follows a cohort of cancer patients over however long a period of years it takes to make appropriate observations. If a point is reached, let us say perhaps ten years, after which survival is similar to that of a proper general population control group, then you may call this group of survivors the cured fraction. This is a useful statistic when it is available. It doesn't develop in all malignancy sites. There are sites of malignancy that, at least as far as current observations are concerned, never do come to a point where subsequent survival is the same for survivors as for a comparable general population.

Perhaps the best procedure to use in thinking about effectiveness of therapy is to attempt to evaluate all these methods of looking at the question. If, for instance, one looks at breast mortality, as has been done in the end results reports, you find the general population mortality is approximately stable, and in most recent years the incidence seems to be rising. If this is so, then one must conclude that, despite the stable general population mortality, something effective is being done in this disease and is working in just the measure to counteract the rise in incidence.

In cervical cancer, mortality is falling. Incidence is essentially stable. This, again, is a favorable combination of these statistics. If it jibes with the changes one sees in five-year survivals, it adds confidence to the conclusion that something effective is being done in management of this disease.

We often think of the clinical trial as being the thing really needed to get at a solid answer to a number of these questions. Clinical trials have become exceedingly popular perhaps a bit over popular, in the cancer field. Almost invariably, clinical trials of therapy have shown little, if any, difference between the one, two or three methods being compared. Do we need to be totally pessimistic when we continue time after time to see this result? I think not. Dr. Hammond has specifically mentioned that one of the things he is most interested in seeing is the best available methods of therapy made generally available. He has made a 50% survival prediction in terms of this concept. He has compared this with survival in earlier time periods. That isn't what is being compared in any of the clinical trials. The formal clinical trials are almost invariably comparing two treatments known before the trial is initiated to be not very different. They are things that highly competent groups of therapists are willing to try, with all the ethical considerations involved, on a randomized basis for different groups of patients. So we know at the start of these trials that vast differences are not going to be seen between them. At best, there can only be small differences; this is what we must be looking for. We are not at all testing the question that Dr. Hammond has raised about the difference between poor and good medical care.

There is another sort of test investigation to be used, and this is the clinical trial of early case finding. This contrasts with the clinical trial of therapeutic methods. A possible such study would be a randomized clinical trial, for instance, of Papanicolaou screening. So far as I know, this has not been done in any of the studies that we have of Papanicolaou screening.

We are at the moment doing a trial of this sort in breast mammographic screening, as already mentioned. We can say clearly, as can be said in Papanicolaou screening for cervical cancer, that cases are found early by this procedure, but finding cases early is not in itself a necessary equivalent of changing mortality. The mortality effect in our study in mammography still remains obscure. I agree with Dr. Hammond's overall optimism in this issue. I am optimistic for the mammographic study, but it is entirely wishful feeling at this moment. I have no data to present to you as to whether or not there is any effect on mortality in this study.

Let me shift to the second issue in Dr. Hammond's paper, prevention, and here I think we are in rather good agreement.

On the smoking issue, I indeed am optimistic. The data to support optimism remain rather few. Dr. Hammond himself has been one of the most active people in making available data on current trends in smoking habits, and this requires careful and sophisticated statistical analysis at this point.

However, over the long run, perhaps many of us will not live to see this because it will be a matter of a generation or two away. I believe smoking habits will be vastly different, a marked decrease from what we see now.

A number of industrial hazards associated with carcinogenesis are well known and must be controlled. Effective results can be anticipated from this, but this accounts for a very small proportion of the overall malignancy picture.

Two other issues that Dr. Hammond men-

tioned I think he didn't mean to emphasize: The breast feeding issue relative to breast cancer and the circumcision issue relative to cervical cancer. I think we agree that these issues are not at all well supported by data that we have now.

The substantive description of how to look at these data has been well covered by Jerry Cornfield's discussion which gets exactly at the issue in all discussions of this group. Cornfield's remarks in connection with his paper should be the guide in considering this session . . . In particular application to cancer prevention, he pointed up the problem that a very large effect, the penicillin effect in pneumonia, for instance, can be studied and has been studied without randomized clinical trials. But I don't think the most optimistic of us expects that sort of massive effect in cancer control. We are looking for the small effect we expect in cancer therapy, and these effects are so overburdened by possible selection that I am very dubious of being able to come to any firm conclusions by methods other than strictly controlled studies.

Prof Cornfield also pointed out the problems of distinguishing in the double-blind concept between blind or random assignment and blind assessment. If one is looking at total deaths from all causes as the end result, then one has the equivalent of blind assessment. This is what generally is being done when we are talking about cancer data. This is true when we are looking at the material presented by the American Cancer Society, by the End Results Groups, and most large statistical studies in the cancer field. It is not what the clinician is talking about. Clinicians are exceedingly optimistic about the sort of things they are doing. They are not looking at end results in terms of overall mortality. They are looking at early effects, so-called "responses" to one method or another. They are counting, for instance, the time of onset of recurrence, and this elusive concept of recurrence must be defined. They are talking about the disease-free interval, and this, again, involves recognizing the point of onset of recurrence.

These concepts are not worth discussing if they have not been studied with the most rigidly controlled sort of blind assessment, and this is not what is being done in the data that clinicians are looking at in their optimism with the methods they are using.

ABRAHAM LILIENFELD, MD, Baltimore: One of Dr. Hammond's comments relates to an earlier discussion on Mr. Cornfield's paper. Dr. Hammond states that if one selects cigarette smoke as a variable there can be many factors, A, B, C, etc, which can cause a series of diseases—lung cancer, coronary heart disease, emphysema, in addition to others.

He suggests that if we had a harmless cigarette, that is, if we remove the carcinogen from cigarettes, this would in turn have little influence on the total life expectancy simply because of the competing causes of death. Deaths from other causes produced by the different components in cigarette smoke may well fill up the vacuum created by the decrease in lung cancer deaths.

It seems to me the same situation applies to coronary disease discussed earlier. In this case we have a set of factors—cholesterol, blood pressure, cigarette smoke, and weight which produce coronary heart disease.

When we look at these various factors in relation to coronary heart disease, I do not think we necessarily conclude that the pathogenetic mechanism is the same for all factors. Essentially we are probably dealing with different pathogenetic mechanisms. If our diagnostic tools were adequate we could subdivide coronary heart disease into coronary heart disease one due to cholesterol; coronary heart disease two due to smoking; coronary heart disease three due to hypertension and coronary heart disease four due to obesity. Using Dr. Hammond's argument with respect to cigarette smoke from carcinogen free cigarettes and its subsequent influence on life expectancy, perhaps merely changing one risk factor involved in coronary disease will not have much effect on the total coronary heart disease problem.

Perhaps what one has to do is evaluate the whole set of risk factors in order to attempt to change the death rate from coronary heart disease.

PROF JEROME CORNFIELD, Bethesda, Md: I have a number of comments, but I think some of them are not going to be resolved this morning, and I would like to make one upon which perhaps we can all agree. We would all be better off if we stopped looking at life expectancy as an end point by which to evaluate intervention effects.

The reason for this, skipping all subtleties, is that life expectancy is, roughly speaking, average age at death. Now, if we are dealing with interventions that affect risks at earlier ages but have smaller or perhaps nonexistent effects at higher ages, and if half or more of the deaths occur at the higher ages, we may have a very important effect at the younger ages and no effect at the higher ages; and it may show up in a one or two-year decrease in overall life expectancy.

So let us clear some of the underbrush away in discussing these matters and talk about

effects on the age specific probability of death.

THOMAS FRANCIS, Jr., MD, Ann Arbor, Mich: I would like to comment that in this discussion of the many factors with which one might try to intervene, there is a very old legend of the physician who was advising his patient of all things he shouldn't do. He said to the physician, "Do you think it is going to make me live longer?" The physician said, "No. It will make it seem a lot longer." I think that is what some of this bokum is all about.

E. CUYLER HAMMOND, ScD, New York: A rather well known person on television recently struggled for almost all of his comments and answered questions by saying, "I want to make this very clear." Well, I would like to make it very clear that present methods I called lifesavers—I called them that to indicate that it was not the same thing as an unsinkable ship.

I will also say that I agree with a very large part of what Dr. Hutchison said.

Now, of course, we do not have figures available from way back; we couldn't, there is no way of getting them. You do not get decent statistics until you first have pretty good medical services in general. We have no knowledge of survival. I think it was very low earlier.

In the first place, there are innumerable papers in the literature, none supported by good figures, but all say that cancer was practically never cured. These are doctors speaking.

There is, however, better evidence now. In the late 1940's I made a study in Puerto Rico. At that time a very large proportion of the doctors on that island had their medical degree from a one-year course in medicine without the benefit of earlier college; and, rather properly, the population did not think much of its medical services and didn't go to its doctors when cancer was suspected.

Well, at that date in Puerto Rico we found many cases of breast cancer with massive fulminating lesions, and the people were dying of this sort of disease. We found many cases of death from other forms of cancer at a very advanced stage. Since that time, control activities of one sort or another, particularly improvement in medical care, have been initiated and they are now curing some people.

It may surprise you to know that I originally made my calculations not to show how wonderful early diagnosis was, but to point out limitations to some people who seemed overly enthusiastic who were, practically speaking, telling the public that if they just heeded the cancer warning signals they would prevent all cancer and need not worry about any further research.

I have taken the other view here at this meeting for the reason that as a result of earlier overselling of early diagnosis there is such pessimism about it that I am afraid we will lose the gains we now have. This would be the height of folly.

There are also areas in this country with practically no medical services where people are developing cancer that could have been cured, but gets to the stage where it cannot be cured.

When it comes to breast cancer, I am more pessimistic about early diagnosis than Dr. Hutchison. In this disease there is every evidence that a considerable proportion of the cases metastasize not through the axillary node but through the mediastinum; and they probably metastasize while still of a microscopic size.

As to the five-year survival rate, I believe Dr. Hutchison must have been thinking primarily of breast cancer when he made his remarks, because 20 years after "apparent"—I put the word in quotation marks—"apparent" cure of breast cancer, the death rate is still way above that of the general population.

On breast cancer I think it is quite fallacious to use the argument that, "early cases live longer than cases late when first treated"; this would be true if you treated early cases with distilled water or anything else, any quack remedy. If you treat them early, they are going to live longer after treatment than if you treat them late.

However, there are sites of cancer that are of a totally different nature, and this applies particularly to cancer of the uterus. This disease typically does not kill by metastasis, it kills primarily by local extension. It is those that kill by local extension in which we have our best results, and this is quite natural.

To my mind this is where we can make the greatest progress, simply preventing disease from going to the incurable stage. I would hate to see this activity limited.

As far as we have gone with cancer of the uterus, incidence rates have dropped somewhat and death rates have dropped. Personally, I think this is more attributable to prevention, if I may use the word, than it is to cure. I have evidence that carcinoma in situ can develop very quickly into cancer. I also have evidence that people with positive smears, about 90% of them, live for a long time and don't develop invasive cancer. But by taking out carcinoma in situ, by taking out the uterus, I think they have precluded the development of cancer in a group of people who are in a very high risk group. This is prevention rather than cure. Nevertheless, it has worked.

That dirty, dangerous place you call home—it's polluted

By RUTH WINTER.

Your home is polluted.

"We've been so concerned with outdoor pollution we haven't paid enough attention to inside pollution," says Dr. Paul Cornely, president-elect of the American Public Health Association.

"If anyone doubts their home is polluted, all they have to do is look at the kitchen exhaust fan," adds Dr. Igho Kornbleuh, chairman of the Department of Physical Medicine and Rehabilitation at the University of Pennsylvania. "It's caked with grease and dirt and shows what we are breathing." The Home Ventilating Institute maintains as much as 200 pounds of grease-laden moisture is given off every year in the average kitchen.

Dr. Kornbleuh and other scientists agree that polluted outside air does not stop at the doors of a house. In summer, when the windows are open, the air can circulate freely within. Even in winter, windows and doors are no real barriers to outside air because homes are not sealed.

Asbestos hazard

One of the health hazards that drifts into the house is asbestos. It is used in brake linings, kitchen tiles, shingles, and a variety of insulating materials, soundproofing material, floor coverings, ironing board covers, pot holders and heating and air conditioning ducts. An extensive study recently completed at Mount Sinai Medical Center's Environmental Sciences Laboratory in New York found that 45 per cent of the white collar workers studied, 75 per cent of the construction workers and 35 per cent of the housewives had asbestos in their lungs. These fibers, once inhaled may causes diseases, including cancer.

D. M. Vincent Mansou of the American Museum of Natural History in New York, points out that many aerosols have talc, a substance similar to asbestos, as a spray base. Deaths of youngsters, deliberately inhaling aerosol sprays for "kicks" are frequently reported, but what happens to all of us when we inhale minute amounts of deodorants, disinfectants, cleansers and other liquids which we freely spray around the house?

"No one knows what effects aerosols have when they are mixed together or even sprayed individually," Dr. Kornbleuh says. "The average kitchen today resembles a laboratory more than the old-fashioned place for preparation of the family's food."

There are approximately 300,000 toxic, or potentially toxic, trade name products on the market. Many of these are found under the kitchen sink or on a closet shelf. Numerous housewives have created lethal gases by mixing products together, such as bleaches and toilet cleansers.

The American Medical Association warns that "Ignoring the safety rules for using many insect-killers, solvents and fumigating spray may result in dizziness, blurring of vision, injurious effects to the kidney, heart, liver, brain or blood, and even death."

Marion Gleason, University of Rochester toxicologist, said poisonous fumes around the house may predispose people to accidents. How many falls from ladders, for example, occur because the victim grew faint or dizzy after inhaling paint fumes near the ceiling of a warm, poorly ventilated room?

One of the cardinal rules of safety engineers is: "If you can smell it, you're breathing too much of it."

Dr. Kornbleuh adds that the internal climate of most homes and offices in the United States is drier than a desert. "Many cases of migraine and other types of headches, rashes, premature skin aging, allergies, respiratory disturbances and irritability are the consequence of exposure to the arid, polluted air within the home," he says.

Monoxide intake

In the United States, the National Center for Urban and Industrial Research points out, "With the increase of air-conditioning, carbon monoxide has been found in autos, homes, motel rooms and other places where the air intake vent is located near a source producing carbon monoxide."

More than 7,000 Americans each year are killed or adversely affected by carbon monoxide. In addition, many people exposed daily to low concentrations of the gas in the home may experience headaches, dizzy spells, anemia, weakness, mental depression, nervousness, irritability and circulatory impairment.

In a recent Tennessee study, 40 per cent of the homes and establishments investigated had one or more appliances that were emitting unduly high levels of carbon monoxide. Gas ranges, ovens, gas floor furnaces and gas space heaters were most often at fault.

But gas is not the only culprit. No one is sure how much radiation we are receiving from and ever-increasing number of modern devices. Said Betty Furness when she testified before a senate hearing on radiation control: "Such innocent and useful things as exit signs on airplanes, luminescent keylocks, elevator alarm buttons, telephone dials, fuse box markers using radium — all can be potentially hazardous if they give off radiation in

excess of the limits set by the Atomic Energy Commission."

The danger of X-radiation in color TV sets has been widely publicized. The federal government has proposed radiation standards for sets manufactured after January 1, and advised viewers to sit no closer than six to 10 feet. Scientists say they do not know what effects low doses of radiation may have on the body over long periods, and they worry about exposure bringing a person closer to the unknown point were genes are mutated.

Another less publicized hazard comes from microwave ovens — new kitchen gadgets that cook food fast. A survey in Pennsylvania found 71 per cent of these ovens gave off radiation in excess of the safety standard.

Americans assume when they turn on a faucet in their homes, they will get pure, fresh water. The Public Health Service, however, maintains that 8,000,000 people served by municipal water systems consume water which exceeds the bacteriological criteria set in the U.S. Public Health Service's drinking water standards.

Water often bad

C. C. Johnson, Jr., administrator of the Consumer Protection and Environmental Health Service, found that bacteriological analysis of more than 700 samples of water coliform contamination. This means that householders were drinking water containing other people's wastes. Furthermore, he said, out of 189 water supplies studied in one area, 14 exceeded the arsenic standards, and 28 exceeded the nitrate limits. Of 79 samples tested, 76 contained pesticide residues.

If most home pollutants are silent, there is one major one that isn't — noise!

In the average suburban home, there are as many as 40 small motors which whir and buzz, along with radio, hi-fi, TV, power mowers, jet planes, the telephone and the voices of children.

The intensity of sound is measured in decibels. The noise level of conversation at three feet is 65 decibles. Sixty is the point at which the use of the telephone becomes difficult, and above 90 damage to hearing may result.

The living room is ordinarily satisfactory at 50 decibels. Running a vacuum cleaner raises the level to 73 when the nozzle is on the rug and 81 when the nozzle is raised.

The modern kitchen is the noisiest place in the house. The automatic dishwasher rumbles at 60 to 70 decibels, the garbage disposal unit at 70 and the refrigerator at 30 to 40.

The typical kitchen often reaches a decibel level of 80 — the equivalent to that of a big city street corner. One expert observed "the noise level in a modern kitchen is just below that of the cockpit of an old DC-3."

Food pollution is another problem. Food poisoning has increased 1,000 per cent since 1951. Intentional additives now amount to 500 million pounds, and then there are the unintentional additives such as nitrates and pesticides.

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SUMMARY AND EVALUATION

mailed: Aug

Subject: Speech on occupational health, including mention of asbestos hazards by a U.S. Public Health Service official. Of interest and importance.

Evaluation: The speaker is Administrator of Consumer Protection and Environmental Health Service of the Public Health Service. While the subject is occupational health in general, asbestos comes in for special attention, both as a hazard and as an example of positive action underway.

Pages 5 & 6 mention the hazards of asbestosis, lung cancer and mesothelioma as reported among studies of insulation workers. Page 9 reveals that the Bureau of Occupational Health is preparing position papers on several critical issues. "The first of these reports, which I understand is nearing completion, will be on the subject of asbestos. I think it will give us a much better understanding of what we can do now, and what we should do in the future, to control this growing threat to workers. Page 12 includes a laudatory reference to the cooperative program under way at Mount Sinai, mentioning Dr. Selikoff, J-M and the Asbestos Workers Union. The Insulation Industry Hygiene Research Program, as such, is not specifically named.

Author: Johnson, C. C.

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Title: "Remarks"

Summary: As given above.

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Remarks
by
Charles C. Johnson, Jr.
Administrator
Consumer Protection and Environmental Health Service
Public Health Service
U. S. Department of Health, Education, and Welfare
Washington, D. C. 20204

I am pleased to have been given the opportunity to participate in this important meeting, and I certainly want to commend the Industrial Union Department of the AFL-CIO for sponsoring these discussions. Certainly the problems associated with occupational safety and health need to be clarified, for they are too little understood by all concerned -- by the public, by union members, by industry, and by the government agencies who have responsibility in these areas.

But it seems to me that, in every important matter, there is a time for conversation and a time for action. In the occupational safety and health field, we have had quite a lot of conversation in the past, but very little action. I believe it's time we translated some of this talk into action.

As you know, the occupational safety and health program of the Public Health Service is now a part of the Environmental Control Administration, one of three constituent units of the Consumer Protection and Environmental Health Service. The other two Administrations are the National Air Pollution Control Administration and the Food and Drug Administration. We in CPEHS are committed to developing sound, coordinated, action programs that will have a real impact on the various environmental and consumer ills that are so disturbing, and even frightening, an aspect of our contemporary life.

*For presentation at the Occupational Safety and Health Conference, sponsored by the Industrial Union Department, AFL-CIO, June 4, 1969, Washington, D. C.

Certainly occupational safety and health are a major -- and urgent -- area in which action has been too long neglected. And, I truly feel, that we are entering a new era of public awareness of, and public concern for, these problems.

True, the general public -- even the working public, if you can make such a distinction -- is so far more concerned about air and water pollution than about the hazards of the workplace. Someone said a few years ago, "The American public really isn't concerned about atomic fallout, because so far it has not affected television reception." Well, I think people are beginning to feel, and recognize, the effects on themselves of the high-risk environment we are so heedlessly creating, and there is a growing demand for whatever changes are necessary, including adequate protection for workers.

Of course, I don't have to tell you that occupational health and safety -- and a good many other environmental problems -- remain the subject of controversy. No one, not even the most cynical opponent, would dare to say publicly that he is <u>against</u> worker health and safety. The trouble begins when we start to talk about how to go about achieving it.

In one camp are those who maintain that all work involves, to a greater or lesser degree, some element of risk and that we must accept fatalistically the resultant carnage. In this view, the worker should look out for himself, stay alert, be responsible -- because most accidents (and occupational disease too, I presume, although this problem is usually glossed over) are the result of human failure.

Then there are those who would issue hard hats, safety shoes, respirators, and what have you, to every employed American and would set and enforce standards for every conceivable hazard for every type of occupation. The aim would be to make the industrial world perfectly "idiot-proof."

The truth surely lies somewhere in between, but I think these two divergent views could be brought closer together if all could come to share a common view of the fundamental nature of man and of the physical environment in which modern man works.

Let's look for a moment, then, at what we know about the occupational setting today. There are, in reality, two facets of the problem:

On the one hand, we still have with us old problems -- health hazards that in some cases go back as far as the Bronze Age in man's industrial life. Lead, zinc, mercury, silica sand, carbon tetrachloride, carbon monoxide, and many other industrial exposures have been well studied, and there is sufficient knowledge available to control them. But still they offer threats to workers. Control procedures break down, are ignored, or are allowed to deteriorate. Our occupational health people recently restudied lead and zinc smelters in one State, and found that controls instituted almost 20 years ago had deteriorated to the point where they were little better than those they had replaced. In other words, here we are in the Space Age, and find ourselves still suffering some of the old problems that were obvious to the medieval alchemists and first became widespread at the dawn of the Industrial Revolution.

In the meantime, new technology has created a whole new kind of work environment, with a whole new set of hazards, and potential hazards. In this new world, the threats themselves are little understood and control technology, in many cases, is not perfected.

For example, high energy sources, such as lasers, which just a few years ago were tools of the scientist, are now commonplace in industry.

Beryllium, a highly toxic metal which was used in the manufacture of fluorescent light bulbs until chronic beryllium poisoning was recognized as a health hazard to workers, is now finding new applications, primarily in the atomic energy field and the space effort.

in soap powders. They may take the work out of washday, but the unprotected worker in the manufacturing plant may develop dermatitis or respiratory irritation.

I don't need to tell you that new processes and massive, high-speed, even automated, industrial machines have created possibilities for accidental injury that go far beyond the worker's capability for self-protection.

The question we must ask then is: "To what extent may the individual be realistically expected to protect himself from the hazards of the modern work environment?"

The answer, obviously, is that it is patently impossible for the worker to take responsibility for his own health and safety in a complex world of physical and chemical hazards which are little understood even by the experts, and which are clearly beyond his control.

The fact is that man has created a new environment but he has not created a new man. We are the products of a slow evolutionary process, and we still have the same physical characteristics that our caveman ancestors had. We still need clean air to breathe, clean water to drink, and are still helpless against unseen or uncontrollable hazards in the environment. We have developed no second sight that tells us when an invisible beam or a seemingly harmless vapor is doing insidious damage to our health.

And we still have the same psychological makeup that those far-off ancestors had too. I suppose, like most of us, the caveman was ready to blame fate for every accident. And, if they'd had golf courses in those days, he'd have felt personally responsible -- like us -- if he ever made a hole-in-one.

So, to come back to our original two divergent views -- certainly every worker needs to be made aware of the hazards he confronts in the workplace and educated to exercise caution -- to "watch his step," to wear earplugs and a hard hat and safety shoes where required. It will be a never ending job for the union shop steward, for we are not likely to change human nature.

But the thing we can do -- and must do -- is to create a working environment in which such a person, exercising reasonable care, can be assured that sudden death or the slow process of industrial disease will not be the wages of his work.

Certainly the occupational respiratory diseases, in terms of severity, represent one of our most important occupational health problems, and one in which adequate controls would pay big dividends, both in reduction of human suffering and in dollars and cents savings to the workers affected, to industry, and to the taxpayer.

Three and a half million American workers are exposed to asbestos in their jobs. They face a dual threat: not only are they subject to the lung scarring pneumoconiosis of their trade, asbestosis, but they are

endangered by lung cancer associated with the inhalation of asbestos fibers. Recent studies of asbestos insulators showed that one of every 5 deaths in this group were caused by lung cancer -- 7 times the expected rate. Half of the men who had worked in the trade had X-ray evidence of asbestosis. One in every 10 deaths were caused by mesothelioma, a cancer of the lung pleura, so rare that it strikes only 1 in 10,000 in the general working population.

Black lung, or coal miners' pneumoconiosis, is a term which has become very familiar to us in the past few months. This disease -- which the British call simply "chronic bronchitis" -- is caused by the inhalation of soft coal dust. It is a progressive, crippling lung disease, often complicated by emphysema in the later stages. The death rate from respiratory disease in soft coal miners is 5 times that of the general working population. The Surgeon General, in Congressional testimony a few months ago, stated that "black lung", at a conservative estimate, affects more than 100,000 soft coal miners in the U. S. Last December after studying all the data, I announced a recommended standard for respirable coal dust in soft coal mines of 3.0 milligrams per cubic meter of air, and there is now legislation before the Congress in which this standard is proposed as a goal.

There are other industries whose workers run an extremely high risk of respiratory disease. In uranium mining, there is, as you know, a very serious problem. Of the 6,000 men who have been uranium miners, an estimated 600 to 1,100 will die of lung cancer within the next 20 years because of radiation exposure on the job.

Over 3,000 cases of silicosis are reported yearly from exposure to free silica -- the major ingredient of all rocks, soils, sands and clay.

For years, we had the comfortable illusion that byssinosis, the lung disease caused by inhaling cotton dust, was not a problem for American textile workers. Even though British workers using American cotton came down with this crippling lung disease, we relied on a limited X-ray survey done years ago which did not reveal a byssinosis problem. Now, our scientists are discovering that America's 230,000 cotton textile workers are also threatened by this respiratory disease. In one mill, employing 500 people, 12 percent were found to have byssinosis. Thirty percent of the workers in the carding room had the disease. In another mill, 26 percent of the workers in the carding and spinning room were victims of byssinosis. The disease begins with "Monday morning chest tightness" and progresses to chronic bronchitis and emphysema.

Talc, diatomite, carborundum, sugar cane fiber, even dust from moldy silage all produce their own form of lung damage, wherever dust control and worker protection are inadequate.

Our National record in preventing occupational respiratory disease is not a good one. We have allowed too many productive people to become disabled, and finally to die prematurely, because we have not paid attention to controlling the dust, fumes, and vapors to which they are exposed. Must we wait until we can count the corpses before we are sufficiently aroused to take preventive action? In West Virginia, it took a walk-out of 40,000 miners to bring about the passage of a comprehensive State workmen's compensation law. It took a mine disaster killing 78 men in Farmington, West Virginia, to make the passage of a national mine health and safety act a possibility.

We must know by now that the chronic inhalation of any foreign material -- cigarette smoke, polluted air, coal dust, of asbestos fibers and many others -- can harm the lungs and cause disease. Moreover, since for the most part, occupational respiratory diseases are entirely preventable diseases, I think we can all agree that we have a responsibility to take action to prevent them.

We in the Consumer Protection and Environmental Health Service are restructuring our occupational health programs with a view to creating as strong and effective effort as our resources will permit -- an effort that will begin to have, as soon as possible, an impact on workers health and safety.

We feel that it is our primary responsibility -- in occupational health, and in all areas of environmental control and consumer protection -- to define as well as possible, and to enunciate as clearly as possible the effect on man of the various environmental impacts to which he is subjected. We are, therefore, giving first priority in all areas of our responsibility to the development of criteria which can be used as a baseline throughout the Nation to avert damage to human health.

With this end in view, our Bureau of Occupational Health is presently reviewing the current status of a number of urgent problems affecting the health and safety of workers. Our purpose, first, then, is to develop, wherever the current knowledge is sufficient, sound, reasonable recommendations to limit worker exposure, or alternatively, to

determine what steps are necessary to lead to an early enunciation of such criteria and to the control or elimination of the hazard.

The Bureau is preparing position papers on several such "critical issues". The first of these reports, which I understand is nearing completion, will be on the subject of asbestos. I think it will give us a much better understanding of what we can do now, and what we should do in the future, to control this growing threat to workers.

Secondly, we have taken steps to assure that all of our occupational health research is mission-oriented, that is, that its purpose is early identification and control of known or potential hazards, and that it is directed at problems which appear to be most widespread and severe. With a limited budget, long-term, basic research is a luxury we cannot afford. We are developing our research priorities in accordance with strict guidelines which take into account the number of workers currently or potentially exposed, the severity of the hazard, and the magnitude of individual exposures.

We believe that, with careful direction of our research toward the purpose of establishing health criteria and control methodology, we can greatly improve our results.

Certainly, we propose to continue studying these problems. We are continuing our research in coal worker's pneumoconiosis, because there is

and new knowledge could lead to better and easier methods of control. But we are convinced that, in occupational safety and health, as in air pollution control and water hygiene and many other difficult environmental problems we face today, the time has come when we must begin to put to work the scientific knowledge we now have to reduce the very real and growing threat to human life and health. If we wait until we have dotted all the scientific "i's" and crossed all the scientific "t's" before we take positive action, it may well be too late. We have to recognize that science is never immutable, and the time will never come when we have all the answers.

We have taken another small, but important step in the direction of a more effective, action program, and one in which you will be especially interested since it will enable us to work more closely with State and local governments, with industry, and with union groups. This year, we will have occupational health representation in two of the Department's regional offices -- in Chicago and San Francisco. These two regions include approximately 37 percent of the Nation's manufacturing work force. And, I might add, the occupational health activities in the States covered range from non-existent to highly sophisticated programs. As soon as we can see our way clear to do so, we want to assign occupational health staff to each of the nine regional offices.

I hope that you will bring your occupational safety and health problems to the attention of these regional representatives, because I am sure that effective liaison at the State and local level can help us get an understanding of good occupational safety and health practices out of the laboratory and into the plant where it will really make some difference to the workers.

We are making progress in another area that I think may be of some interest to you. As you know, there has never been any sort of national reporting system for occupational illness and injuries, so that our Public Health Service program has been weakened by a lack of adequate data. Moreover, our inability to precisely assess the scope and magnitude of the occupational safety and health problem has stood in the way of public understanding and support of a National effort to protect workers.

To try to get more adequate information, we are beginning to develop an effective national monitoring and surveillance system. The Bureau of Occupational Safety and Health, some years ago, began conducting surveys in seven major metropolitan areas, covering some 2,500 plants and over 400,000 employees. The surveys were so designed that they can give us a statistical picture of environental threats to 2,150,000 workers in 34,500 plants.

We are currently examining the data that we have gathered thus far, and taking steps to insure that continued work in this direction will provide information that will be of value to all who are concerned with occupational health. A preliminary appraisal of the information we have gathered suggests, to no one's surprise I am sure, that many varities of working environments are beset with health and safety problems that require correction.

We believe that we can, by following this pattern of surveillance, and drawing on the information available from workmen's compensation and Social Security records, develop a far more complete picture of worker health and safety throughout the Nation than we have ever had before. We are also planning a reporting system through which a number of industrial physicians will provide us with case histories and other information obtained in their practice.

All of us who are concerned about the health and safety of workers have a great many problems confronting us today -- and they are going to grow rather than diminish in the years ahead unless we make a very great effort to reduce them.

The problems associated with asbestos exposure -- about which Dr. Selikoff will speak later -- is one of them. It is also a good example of how essential it is in the occupational health field for Government, industry, and unions to give their support to a dedicated university-based investigator like Dr. Selikoff, who has added so much to our knowledge of the asbestos problem. Dr. Selikoff's current study of insulation and other workers in the construction industry, conducted under the auspices of Mt. Sinai School of Medicine, is being jointly financed by the International Association of Heat and Frost Insulators and Asbestos Workers and the Johns Manville Corporation. Our Bureau of Occupational Health is providing technical assistance, consultation, training, and loan of equipment. This is the kind of cooperative effort that will be required if we are to make significant progress in the many areas of concern to the worker.

Virginia are cooperating with us now on a study of health problems in their industry, as are the International Chemical Workers in Florida. We have had a long-standing agreement with the United Mine Workers of America in connection with our research on hazards to coal workers. We look forward to developing other projects with labor and industry that can be mutually beneficial.

I hope you will agree with me that we in the Consumer Protection and Environmental Health Service are, in fact, marshalling the resources we have to build a stronger, more effective occupational safety and health program, and one that will begin to have a real impact on the problem.

But, I will be the first to say that the future will judge the whole pattern of our efforts today inadequate. I mean not only the efforts of government, at all levels, but the efforts of industry, and of labor. All of us, the public included--and we are, after all, a working public--have neglected to place on occupational safety and health the emphasis and the resources that this important problem merits.

We have made progress in recent years in our unsteady and halting march toward an improved environment. Certainly all of us have shown a desire for clean air and pure water. There is growing public demand for more sensible use of our environment, which, in our rush to gain the benefits of technology, we have heedlessly exploited, defaced, and polluted. But I have not noted a similar impulse toward improvement with regard to the work environment--which is, after all, the place where virtually all of us spend approximately one-third of our lives. And it is, unfortunately, this part of the environment in which contamination and other hazards to our health and well-being are most severe.

Society, judging by the response to the Air Quality Act of 1967, seems unwilling to continue to accept present levels of particulate pollution in the city air. Yet, at this moment, there are those who contend that it

is permissible for coal miners to breathe, every working day, disease-producing dusts at levels that exceed by many times anything we have ever endured in the ambient air.

Last year, the Congress passed the Radiation Safety and Health Act to protect citizens from X-ray and other hazards resulting from color television sets, microwave ovens, and other marvels of our new technology. We may be sure that this Act was passed in response to the public will. But such exposures are most acute in the work environment. I'm not suggesting that we do not need the protection in our homes that this important legislation will afford. But I offer it as another piece of evidence which suggests to me that public concern for the total environment--though late in arriving--is still far ahead of our concern for the occupational setting. All of us must redouble our efforts to insure that the rising interest in the environment encompasses a full appreciation of occupational hazards.

Interest in environmental health and consumer protection is quickening at a very gratifying rate. We are hopefully moving toward the day when Americans everywhere will be able to breathe clean air, bathe and boat in clean water, and find true recreation in a countryside unblemished by the blight of automobile graveyards and smoking dumps. We will reach that day when we have learned to employ our wondrous technology to solve the byproduct problems that technology has created. But what a hollow victory we will have achieved if a clean and healthful environment is to be enjoyed on weekends only.

I assure you that we in the Consumer Protection and Environmental Health Service will do everything within our power to make environmental health a seven-day-a-week, 52-week-per year reality.

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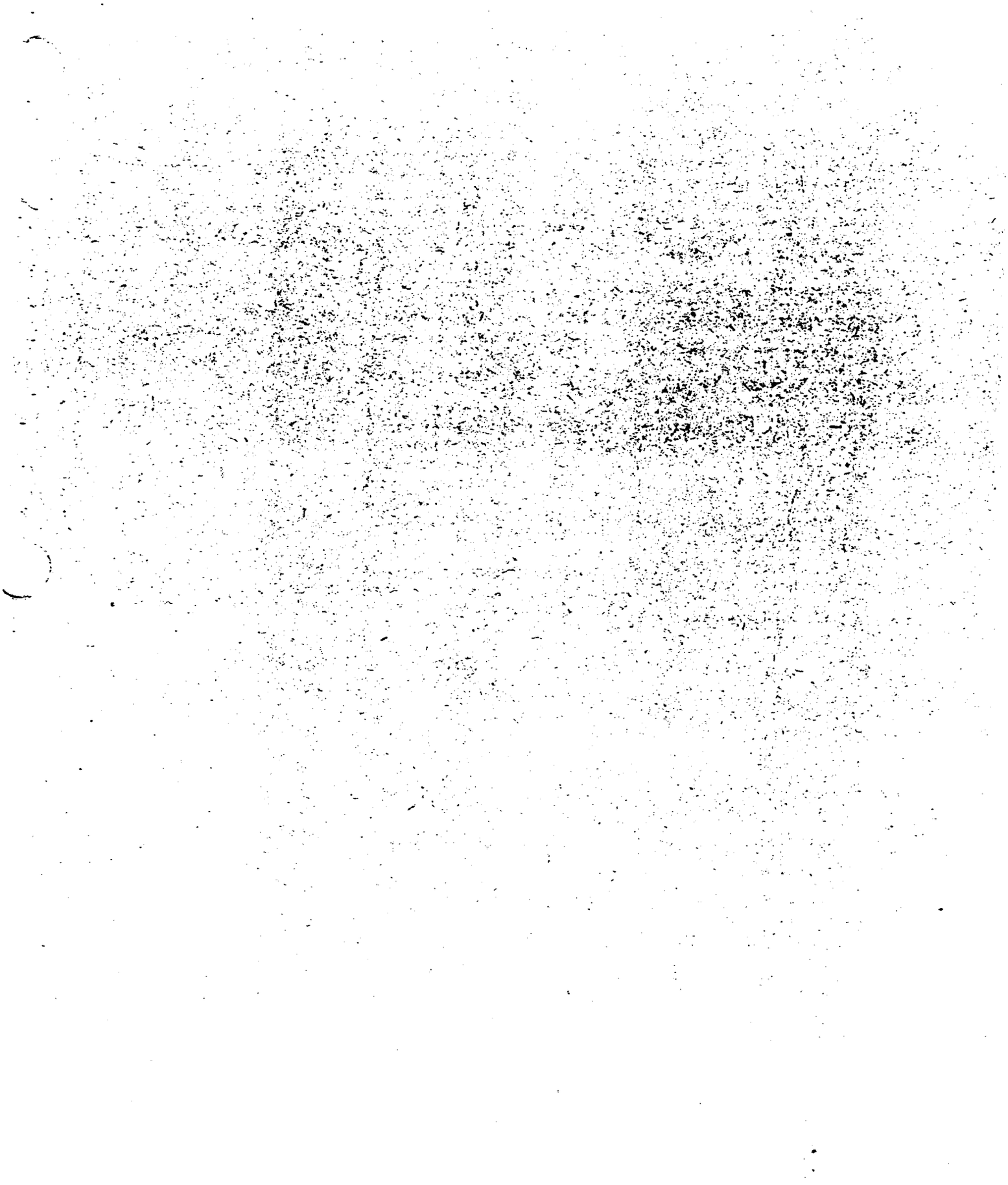

Panel Finds Danger To the Environment From Technology

By ROBERT REINHOLD
Special to The New York Times

DALLAS, Dec. 28—A panel of experts in environmental science warned, today that the "unanticipated" hazards of spreading technology threatened man's existence.

For five hours, one scientist after another stood up and discussed what they termed the grave consequences implicit in the continued use of nuclear power, herbicides in South Vietnam, asbestos in building materials and deep wells for storage of chemical wastes.

"We are altering man's environment in ways we do not understand and in ways that may be disastrous," said Dr. Margaret Mead, the anthropologist, who opened the session at the annual meeting of the American Association for the Advancement of Science at the Statler-Hilton.

Many Scientists Present

A standing-room-only audience of scientists attended the session, a reflection of the rising concern of scientists for the consequences of the technology they have spawned.

"I believe that unless we begin to match our technological power with a deeper understanding of the environment we run the risk of destroying this planet as a suitable place for human habitation," said Dr. Barry Commoner, director of the Center of the Biology of Natural Systems at Washington University in St. Louis.

He said, for example, that the

Continued on Page 38, Column 5

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S-M.-no. 198
Emailed: Jan 1/69

Scientists' Panel Warns of Peril Of Technology to Environment

Continued From Page 1, Col. 4

use of nuclear reactors had to be evaluated in the light of "hidden costs" to human health from the release of iodine 131, a radioactive substance that could settle in the thyroid gland and possibly cause cancer.

The environment exacts a price for "technological intrusions," such as iodine 131, he said, adding that "the powerful illusion that we can avoid payment of this price is fostered by the enormous accomplishments of technology."

"Because of our illusions," he continued, "we have become unwitting victims of environmental pollution." He cited the damaging effects of automobile smog, insecticides, detergents, radioactive fallout, chemical weapons and the wide spraying of herbicides in South Vietnam as examples of "technological affronts" that are made "not out of greed, but out of ignorance."

Use of Herbicides Scored

Prof. Arthur W. Galston of Yale University charged that the use of herbicides in South Vietnam had violated the normal criteria for the safe use of these agents. The herbicides were used for the defoliation of enemy-held areas.

"Immediate investigations are required to determine the extent and nature of the undeniable ecological damage produced by our spraying activities in Vietnam," he said. "Extension of such activities to new areas should be strictly circumscribed until we have accurate knowledge of the consequences of our action."

The biologist also said that some of the herbicides in use in South Vietnam, such as Picloram, were not broken down by the soil and persisted in the earth for some time after spraying. He suggested that these poisonous agents were finding their way into human food supplies in South Vietnam.

Well Use Assailed

Another speaker, Dr. David M. Evans, a geologist at the Colorado School of Mines, protested against the widening practice of pumping industrial wastes down deep wells.

"The surface of the earth is not just rock but also liquid," he said. "Any time we change the pressure of those fluids we are changing the balance of the ground we stand on."

The number of disposal wells in the United States, up to more than 110 from one or two in 1950, is expected to double in the next few years, he said. Already, the geologist contended, these wells are causing extensive permanent damage to farmland and urban areas in the Southwest.

He cited the events in Denver in 1962, when the Army drilled a 12,040-foot-deep well to dispose of unwanted chemical warfare agents. "The following month, Denver experienced the first earthquake felt in 80 years. There is a perfectly beautiful correlation between the number of earthquakes and the number of gallons poured into the ground."

He also charged that salt water, a waste product of oil drilling, was seeping out of waste wells in Texas with disastrous effects. He noted that one barrel of oilfield brine could contaminate 20,000 gallons of drinkable water and pointed to buildings damaged by sinking land in Houston.

View Is Disputed

This view was disputed by Dr. Boysie E. Day, a herbicide expert from the University of California at Riverside, who said: "To view with alarm, to decry, deplore, and denounce herbicides in the scientific and public press without taking the trouble to learn the facts will not make herbicides go away and allow us to revert to a state of nature. Herbicides are a fact of the world."

The "widening spectrum" of diseases traced to the use of asbestos in building materials was discussed by Dr. Irving J. Selikoff, chairman of the Division of Environmental Medicine at the Mount Sinai School of Medicine in New York. He said that recent studies had detected an unusually high incidence of lung cancer and other diseases among construction and insulation workers.

There are 3,500,000 men, or 5 per cent of the total employed population of the United States, working in construction and building industries. Dr. Selikoff said that "significant exposure may even extend to families of these men and perhaps to residents in the neighborhood surrounding their places of employment."

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Ferruginous Bodies in Human Lungs

Prevalence at Random Autopsies

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Ferruginous bodies with a transparent central core or no visible central filament were present in 97 of 100 autopsies; they were found in 55 of 56 men (98%) and in 42 of 44 women (95.5%). The youngest subject with ferruginous bodies was a 24-year-old woman. The bodies were generally more abundant in the lungs of the men. Thirty-two patients in this series had some form of malignant disease, but there was no apparent association between the malignancies and the relative abundance of ferruginous bodies. There were two cases of primary lung cancer in men, but no case of mesothelioma, nor of asbestosis.

The significance of these findings from an epidemiologic point of view must await identification of the central core of the ferruginous bodies, but in any event, pulmonary fibrosis or neoplasm is rarely associated with their presence in the lungs of these autopsied city dwellers not occupationally exposed to asbestos dust.

IN RECENT years studies have been conducted in a number of cities of the prevalence of ferruginous bodies in the lungs of city dwellers at autopsy.

In 1960 in Cape Town, South Africa, Thomson et al[1] reported finding that 30% of the men and 20% of the women at autopsy were found to have asbestos bodies present in their lungs.

The following year, Thomson and Graves[2] reported finding a similar prevalence in Miami, Fla. Men and women showed positive results in 31.6% and 20.4% respectively.

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In 1964 in Pittsburgh, Cauna, et al[3] reported finding 47% and 34%, respectively, in men and women autopsied.

The same year, Anjilvel and Thurlbeck[4] studied autopsies in Montreal, and found 57% in men and 34% in women.

Meanwhile, Meurman[5] was finding an overall prevalence of 70% in the urban element of a regional survey for asbestos bodies in Finland.

In 1966, Ghezzi, et al[6] reported finding 54% and 44%, respectively, in men and women autopsied in Milan, Italy.

As it appears that all the series studied so far have been of comparable age range and occupational backgrounds, the most likely explanation for this trend is that progressively more intensive searches are being conducted. The techniques employed in the foregoing studies, with the exception of the Finnish, have been basically the same: examination of unstained air-dried smears of juice expressed from cut portions of fresh postmortem lung or scraped from a cut lung surface. Some took their samples from the lung bases (Thomson et al[1,2]) and some from both upper and lower lobes (Cauna et al[3]). Anjilvel and Thurlbeck[4] sampled only left lungs. Meurman[5] examined 20 micron sections stained for iron by Perle's stain in his search for bodies.

In all the above studies, their authors have defined the bodies morphologically in similar terms, and have described them as "asbestos bodies" on the grounds that they considered only asbestos fibers to be responsible for the formation of such bodies and to be at their core. However, as Meurman[5] points out, bodies with a striking or even

complete resemblance to asbestos bodies ... been found to have at their core, not a...stos, but elongated or fibrous particles of a variety of other minerals including biotite-fusain, hornblende-rutile, diatomaceous earth, graphite, and carborundum. Moreover, it has recently been shown by Gross et al[1] that bodies morphologically indistinguishable from true asbestos bodies can be produced in hamsters by three different man-made, nonasbestos fibrous materials (namely, silicon carbide "whiskers", ceramic aluminum silicate, and fibrous glass). The term "ferruginous body" has, therefore, been adopted by the authors, to describe all such bodies unless and until the central fiber has been positively identified as asbestos. This generic term encompasses both asbestos and pseudo-asbestos bodies as previously employed.

In this paper, the ferruginous body is defined as an elongated golden to reddish brown structure usually with clubbed ends; the shaft which often shows a segmented or beaded appearance is usually straight, but sometimes curvilinear with a tendency towards symmetry; usually it is from 3 to 5μ ...ameter and 20 to 100μ in length. The ...ng contains iron demonstrable by Perls' stain (prussian blue reaction), and which is probably composed of ferritin or a ferritin-like protein material (Davis[2] and Collet, written communication, June 1966); it may cover the structure completely, masking the central fiber from direct view, or may be incomplete in the central portion of the shaft or in the interstices of the body, revealing an expanse of naked fiber. In this study, only ferruginous bodies with transparent or no visible central fibers were counted.

Materials and Methods

The 100 lungs comprising the material for this study were supplied to us, formalin-fixed, through the central autopsy room of the Presbyterian University Hospital, Pittsburgh, and came from autopsies performed on patients who died in five metropolitan Pittsburgh hospitals during the period from late 1966 through June 1967. The lungs were not, therefore, from consecutive autopsies and were unselected by us. There were 56 men, age range 37 to 87 years (mean age 65), and 44 women, age range ...to 90 (mean age 59).

Thirteen of the men and six of the women were Negroes. Death had resulted from a wide variety of causes; 32 died from malignant conditions, including two cases of primary lung cancer (both men). There was no evidence of mesothelioma in any of the patients at autopsy. The lungs were examined superficially for calcified pleural plaques, and none were found; however, as some of the lungs were incomplete when received, there is a possibility that their presence may have been missed in some instances.

The lungs were sliced longitudinally on a commercial electric "bacon slicer," into slices approximately 3 mm in thickness and a total of about 300 gm (wet weight) was sliced in this way in each case. The slices were floated on the surface of about 2½ inches of domestic laundry bleach, undiluted (active constituent 5.25% sodium hypochlorite), contained in individual plastic pails of 9½ inches internal diameter at the base. Immediately as the lung tissue came into contact with the bleach, there was vigorous effervescence with the evolution of chlorine, which ceased after a few hours. Only the surface in contact appeared to be attacked. The pails were left undisturbed in a fume hood for 16 to 24 hours. At the end of this period the floating lung slices were seen to have shrunk considerably in area. The attacked undersurface was seen to be bleached yellowish white. At the bottom of the bucket, a dirty-looking grey to black sediment had formed. In some cases this was firmly adherent to the bottom and sides of the bucket as a greyish film. In others, the sediment appeared to be quite loose and readily resuspended in the spent bleach. In the former cases with an adherent film it was possible, after picking off and discarding the shriveled lung tissue, to decant all the spent bleach which was also discarded. The film was then dissolved off the inner surface of the pail by gentle agitation with about 200 ml of a 50% mixture of chloroform and 95% ethanol. The turbid, grey suspension so obtained was then decanted into a liter-separating funnel and shaken with an equal volume of clean water and allowed to stand. In the cases in which the sediment was nonadherent, about three fourths of the spent bleach only was carefully decanted, leaving most of the sediment in the remaining one fourth bleach. This was then mixed in the bucket with a similar volume of the chloroform-alcohol mixture and the whole swirled around the pail a few times and then decanted into a separating funnel.

On standing for ten minutes or so, the contents of the separating funnel were seen to separate into three layers. The lower, denser chlo-

...roform layer was ... colored and contained ... ganic, insoluble, ... gested portion of the ... bodies that might be pres... layer was turbid and gre... um chloride, any undeco... chlorite and other soluble ... tion of lung tissue ... variable amount of ... sion. At the interf... layers was invariably ... layer, from 1 mm to ? ... the amount of carbonac... the original lung, consi...

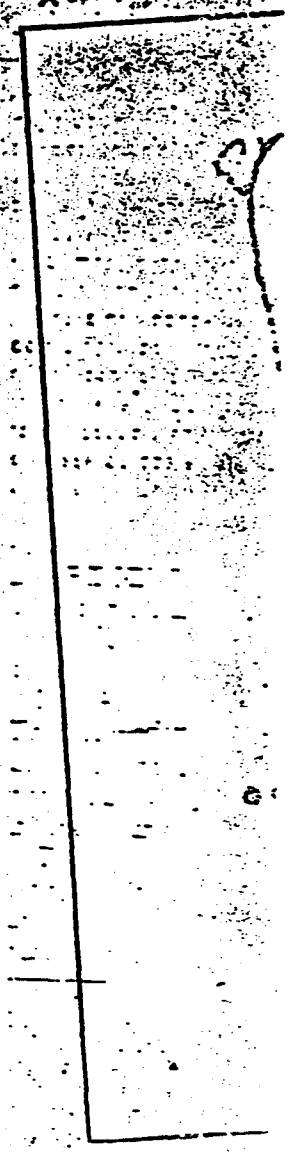
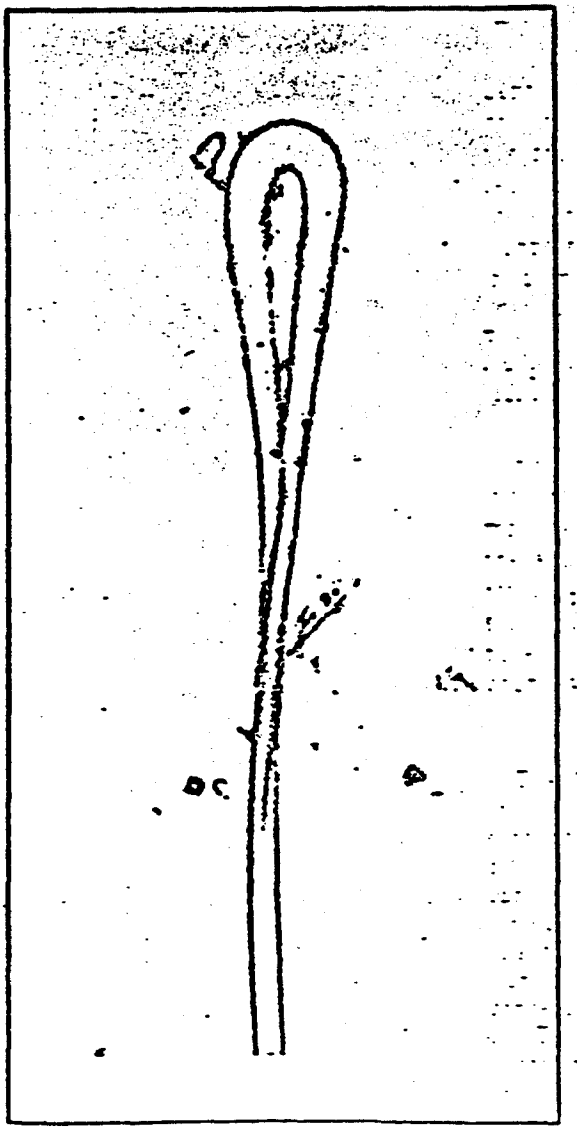


Fig 1.—End of picked smear from human lung ... implies fusion during ... ×1100).

roform layer was more or less clear and amber colored and contained the denser, mainly inorganic, insoluble, mineral residue from the digested portion of the lung, plus any ferruginous bodies that might be present. The upper watery layer was turbid and grey, and contained sodium chloride, any undecomposed sodium hypochlorite and other soluble products of the digestion of lung tissue and blood, formalin, and a variable amount of organic debris in suspension. At the interface between the two main layers was invariably a dark grey to jet black layer, from 1 mm to 2 cm in depth, according to the amount of carbonaceous material present in the original lung, consisting mainly of carbon

Fig 1.—End of asked fiber seen in concentrate smear from human lung in this series. Appearance implies fusion during formation of fiber (unstained, ×1100).



together with a certain amount of lipidic material (there was also evidence that some lipid material was held in solution in the chloroform). The lower layer only was run off into an elongated glass tube with ground-glass stopper, mixed with a similar volume of fresh clean water and vigorously shaken. This time the emulsion formed was usually much more stable and was poured into 50-ml centrifuge tubes and spun for ten minutes at 2,000 rpm. Once more there was a three-layer separation with relatively little carbonaceous material at the chloroform-water interface, plus a dense deposit at the very apex of the tube, from 1 to 3 mm in depth and ranging in color from dirty white through greyish pink to a distinct reddish brown. Occasionally this deposit was almost black, indicating the persistence of carbonaceous material adherent to the denser matter. In such cases, a further separation was performed by resuspending the deposit in more chloroform-water emulsion and recentrifuging. Both chloroform and watery layers were finally decanted and discarded, and any carbonaceous-lipid ring adherent to the walls of the tube removed with cotton wool swabs before the tube was righted. The apical deposit was suspended in two or three drops of distilled water, plus two drops of dimethyl sulphoxide as a preservative and was then ready for microscopy as a smear.

Smears were prepared from a drop of the suspension of as near constant size as possible, using uniform pipettes held vertically, and allowing a free-falling drop to form and fall onto the slide. The smear was allowed to form by the natural spread of the drop only. It was found feasible to examine the smears initially in the wet state under low power (16 mm), under which conditions the majority of the ferruginous bodies were very readily seen and counted under transmitted light. Subsequently, as they became air dried, they were coverslipped with a mounting medium (Permount) and reexamined under high power for confirmation of very small bodies or otherwise doubtful structures.

Quantitative Estimation of Relative Abundance.—It is appreciated that employing a technique of this kind, with possibilities of sampling variation at almost every stage, only a very crude estimate as to the relative abundance of ferruginous bodies in the original lung tissue can be formed. However, the specimens did seem to form naturally into three categories. There were those in which examination of several smears (five was the maximum number practicable with the amount of material avail-

able) was necessary to find a single ferruginous body. These were arbitrarily classified as "Abundance Category 1" and were taken to include those where no more than one body was found in any one smear.

Next, those specimens in which from two to five bodies were found in any one smear were placed in "Abundance Category 2" and lastly, those yielding over five bodies in any one smear were classified as "Abundance Category 3." The three negatives in this series yielded no bodies in five smears (approximately 2,000 low-power fields). The specimen in category 3 with the most bodies showed 32 in one smear. For comparison, a smear was prepared by the same technique, from the lung of a known clinical case of asbestosis. The resulting density of asbestos bodies was so high that they could not be counted with any accuracy, but would be of the order of many hundred in one smear.

Routine histological sections stained with hematoxylin and eosin were prepared from 20 of the lungs showing the most ferruginous bodies by the digestion technique.

Results

Ferruginous Bodies.—Ferruginous bodies were found in all but one of the lungs taken from men (98%), an 89-year-old white man. Ferruginous bodies were found in all but two of the lungs taken from women (95.5%). The two negative results were found in the lungs of white females: one 16 years of age, and the other 42.

The Abundance Categories, mean ages in each category, and percentage in each category with malignant disease for both sexes are given in the Table.

The relative abundance of ferruginous bodies is significantly higher in men than in women ($\chi^2 = 8.973$, indicating that sex and abundance category are associated, at a 97.5% significance level). The age range for men was 37 to 87, and for women, 24 to 90. There seems to be a slight tendency for the relative abundance to increase with age, but the range of mean ages between the different abundance categories is small compared to the overall age ranges, and it was not possible to demonstrate statistical significance for this tendency.

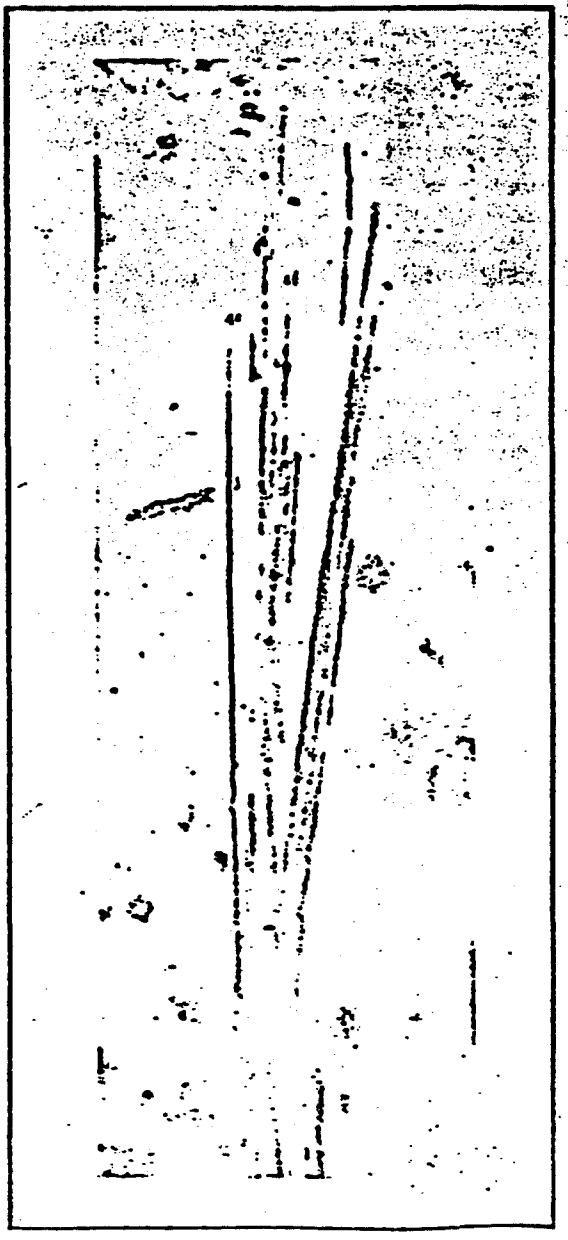
There does not appear to be any definite association between malignancy in general and relative abundance of ferruginous bodies in the lungs in this series.

There was definite histologic evidence of interstitial pulmonary fibrosis in only two of the lungs examined in category 3, but this was not of the type seen in asbestosis.

The two cases of primary carcinoma of the lung were both in category 2.

Naked Fibers.—The digest concentrates contain not only ferruginous bodies, but varying amounts of insoluble and relatively dense debris, denser, that is, than 1.5 gm/cc, the density of chloroform. Where carbon

Fig 2.—One end of naked fiber showing multiple longitudinal fission as seen in asbestos fibers (unstained, × 1100).



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persists in the concentrate from heavily pigmented lungs, it presumably does so by adhesion to denser mineral matter. Of particular interest, however, is the presence of what appear, in the light microscope at least, to be naked fibers of various types. Nearly all are translucent and rectilinear. Their range of dimensions is similar to that for the ferruginous bodies. Most appear to have fractured ends, and the fracture is usually conchoidal in appearance. Some, however, have fused ends or a "shepherd's crook" configuration (Fig 1). The latter we consider to be undoubtedly man-made, either "mineral wool" or fibrous glass, both of which materials are made from fused material which is spun and drawn out in a jet of steam or air. One naked fiber was found which shows the characteristic longitudinal fission associated with asbestos fibers (Fig 2). Whether such fibers were destined to become ferruginous bodies, but for the supervening death of the host, we cannot say.

Comment

Fibrous dust particles would appear to be part of the modern urban environment, at least of industrial cities. Cralley et al[9] comment upon their ubiquity in a recent paper. They give a long list of materials of animal, vegetable, and mineral origin, both natural and synthetic, which have the capability of forming fibers of respirable dimensions. As far as the naked fibers in our concentrates are concerned, organic materials may be ruled out as being unlikely to survive the prolonged action of sodium hypochlorite, and also being of lower density than chloroform. However, once such a fiber has become coated to form a ferruginous body, it may well survive into the concentrate.

Ferruginous Bodies.—In this series, only ferruginous bodies with transparent or invisible central filaments have been counted, as these are potentially, though not necessarily, asbestos bodies. However, in many smears additional ferruginous bodies were seen with opaque black central filaments (Fig 3).

A small number of ferruginous bodies were seen which exhibit longitudinal fission of the central filament and multiple extremities. Figure 4 shows such a body with one

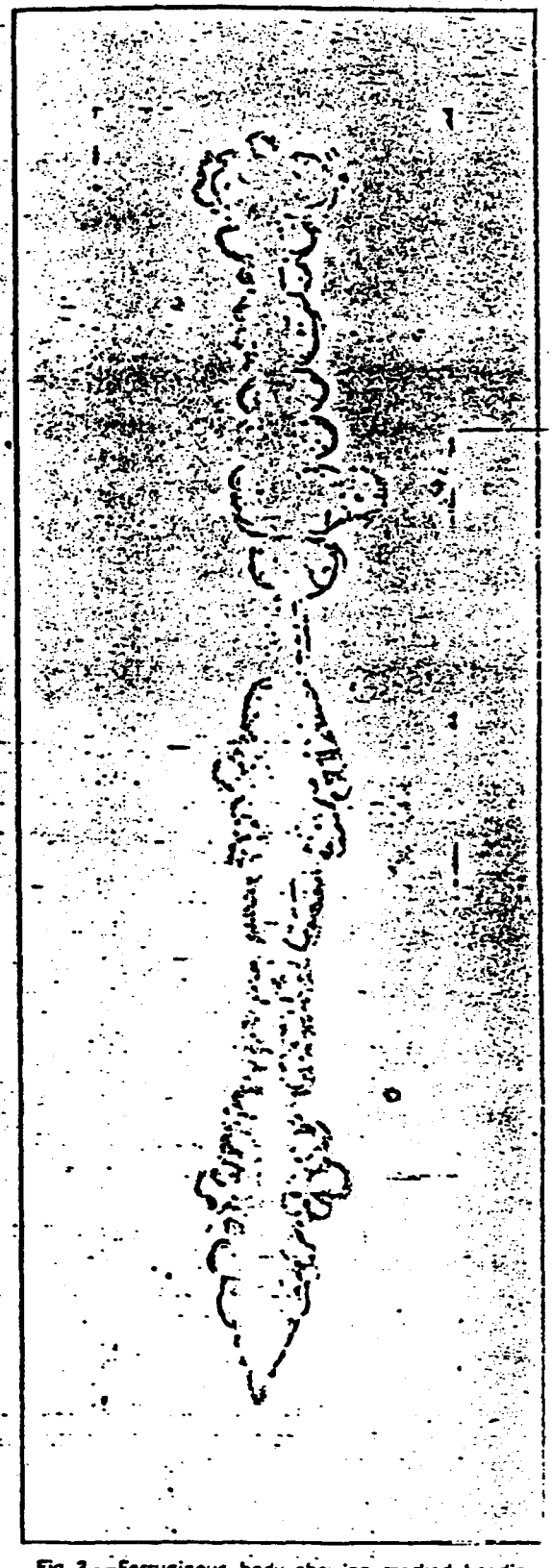

Fig 3.—Ferruginous body showing marked beading or segmentation in coating, and opaque central filament (unstained, × 1100).

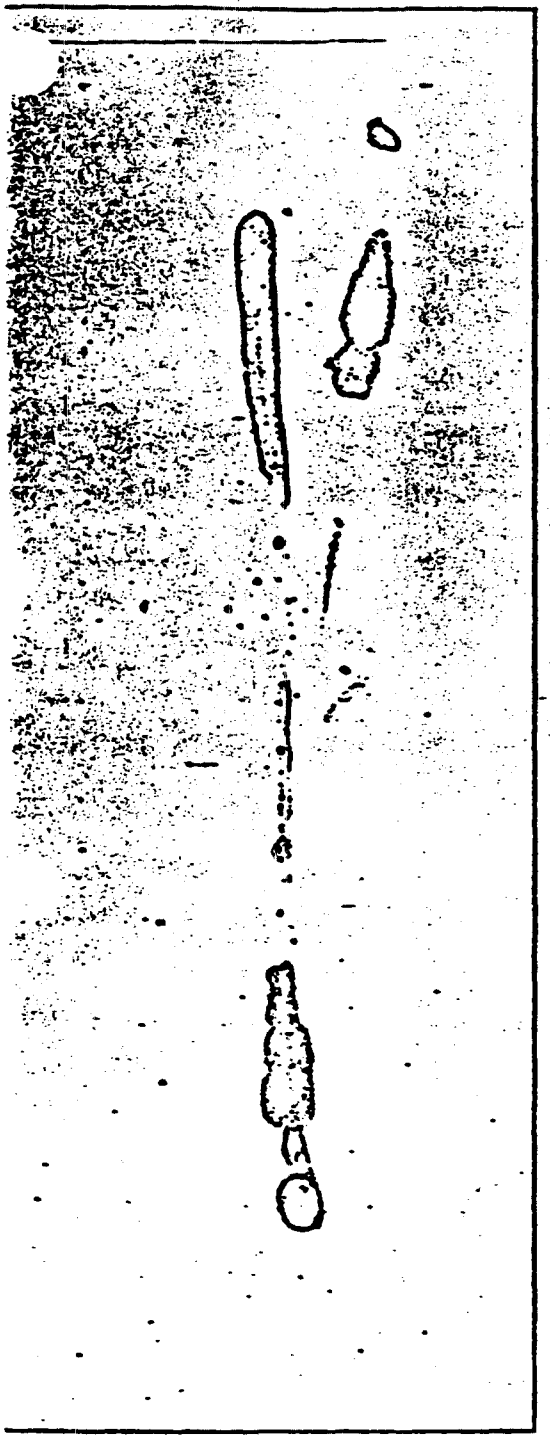

Fig 4.—Ferruginous body showing bifurcation of shaft and one double end. Appearance strongly suggestive of asbestos body (unstained, ×1100).

ouble end. Such bodies are almost certainly sbestos bodies, for we know of no other maerial which undergoes longitudinal fission n this microscopic scale.

*Distribution of Cases**

Abundance Category	No. of Cases	Distribution (%)	Mean Age	With Malignancy (%)
Men				
1	21	38	62	33
2	15	27	64	40
3	19	35	70	21
Total	55	100	65	33
Women				
1	29	69	57	31
2	8	19	68	38
3	5	12	64	20
Total	42	100	60	31

*Mean age, prevalence of malignancy by sex, and abundance of ferruginous bodies.

However, full and positive identification of both central filaments and naked fibers will have to await the further development of physical or physicochemical techniques. Positive identification of asbestos fibers by their distinctive electron-diffraction pattern is already underway in this laboratory.

Significance of Findings.—There is an urgent need for more data in order to form any estimate of the significance of ferruginous bodies and naked fibers in the human lung in terms of pathogenesis, actual or potential. The definite facts are few. It is now widely accepted that asbestos dust, if inhaled in sufficient quantity such as only occurs in those occupationally exposed in the absence of adequate dust control, may be carcinogenic to the human lung. In rats, the inhalation of chrysotile asbestos dust in high concentrations has been associated with a 35% prevalence of lung cancer.[10] There is also strong evidence that crocidolite, a particular variety of asbestos largely mined in South Africa and little used in the United States, may cause pleural mesothelioma—a rare tumor in man, after a time lag of many years.

Lastly, there is the established fact that primary carcinoma of the lung has increased in incidence in most industrialized countries over the past 50 years. However, little or nothing is known at present about the quantitative relationships involved. In the case of asbestos, what is the minimum pathogenic or carcinogenic dose? The only data on this question are afforded by the observation that the excess experience of lung cancer shown by certain asbestos industry workers in the 1930's and 1940's has been greatly re-

duced or altogether eliminated by the implementation of dust-control measures in the plant.[11] However, it is unlikely that such dust-control measures have reduced the level of exposure of the workers to respirable asbestos dust as low as that of the general population. Is the asbestos fiber rendered completely and permanently innocuous to the human body once it has become coated to form an asbestos body so that only the naked asbestos fibers are pathogenic? Which if any of the nonasbestos fibers, coated or uncoated, are pathogenic to the human lung and in what quantity? Can ability of nonasbestos fibers to provoke ferruginous body formation be related in any degree to pathogenicity? There is evidence from animal experiments that it cannot. Gross et al[7,12] have shown that fibrous glass, ceramic aluminum silicate fibers, and silicon carbide whiskers are relatively inert in animal lungs, beyond the fact of ferruginous body formation.

In conclusion it can be said that at this time there is no evidence that the presence of asbestos dust particles or dust particles of other fibrous materials, coated or uncoated with ferruginous material, in the lungs of individuals not occupationally exposed to these dusts in industrial urban communities, is making any contribution to pulmonary disease, inflammatory or neoplastic.

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Chairman Emmanuel Farber, MD, Department of Pathology, University of Pittsburgh School of Medicine, provided the lungs investigated in this study.

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THE MONTREAL STAR, TUESDAY, OCTOBER 14, 1959

Can asbestos cause cancer?

Canadian Press

TORONTO — An article to appear in the Autumn issue of the Canadian Cancer Society's national news letter says that a cancer which occurs in the lining of the lung may result from exposure to asbestos.

The cancer, called mesothelioma, may cause a re-examination of modern building techniques which call for the use of asbestos.

The article says this was the opinion of Dr. E. Cuyler Hammond, vice-president of the American Cancer Society, who discussed this cancer at a recent Cancer Orientation Institute for Science Writers at Annville, Pa.

The article says that in Canada a national committee to study the asbestos problem has beeen organized under the sponsorship of the occupational health division of the federal department of national health and welfare.

It says special studies are being conducted in the asbestos-producing areas of Thetford Mines and Asbestos in Quebec and Cassiar in British Columbia.

In addition, the International Union Against Cancer has organized a committee under Dr. J. C. Gilson of Cardiff, Wales, to collect evidence on the disease from across the world.

The article quotes Dr. Hammon as saying that until recently only 15 to 20 cases of mesothelioma were uncovered annually in the United States.

But he said the rate is climbing in the U.S. and Canada and has been found frequently among asbestos miners in South Africa, Poland, Finland and Czechoslovakia.

Dr. Hammon said it is a serious problem because the use of asbestos in building has increased in the last few years.

"On the face of it, the use of asbestos would seem to be more serious than smoking as a threat to health because cigarettes are not essential, but it would be extremely difficult to build skyscrapers, or power plants, or any one of a number of constructions without using asbestos for insulation," the article quotes Dr. Hammond as saying.

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