The Significance of Irregular Opacities on the Chest Roentgenogram*

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Irregular opacities are not uncommonly observed on the chest roentgenogram in the absence of interstitial fibrosis of the lungs. In such circumstances the irregular opacities, when present, tend to be relatively scanty and seldom, if ever, exceed an ILO category of 1/1. They are found in association with cigarette smoking, especially when emphysema is also present. The development of irregular opacities is also related to exposure to various mineral and other dusts, and although their prevalence increases with cumulative dust exposure, in general the type of dust, whether fibrogenic or relatively inert, seems to be of little moment. The presence of irregular opacities remains a troublesome confounding factor in epidemiologic studies of both dust-exposed and nonexposed populations. The morbid anatomic changes that occur in the lungs of nondust-exposed workers and which are responsible for the development of irregular opacities in the chest roentgenogram remain unknown. (Chest 1992; 102:251-60)

The inclusion of irregular opacities in the International Labour Office’s (ILO) classification of the pneumoconioses was undoubtedly a step forward. As a result, it became possible to grade the severity of asbestosis in a manner similar to silicosis and coal workers’ pneumoconiosis (CWP). While there is general acceptance that irregular opacities are usually present in the interstitial fibroses, whatever the etiology, the occurrence of such opacities in other conditions is often not acknowledged. It is evident that the presence of irregular opacities in the chest roentgenogram may be associated with a number of conditions and factors, thereby leading to incorrect inferences being drawn. Some have suggested that the presence of such opacities in a coal miner denotes CWP, while others have stated that when irregular opacities are seen on the chest roentgenograms of an asbestosis-exposed population, their prevalence is unaffected by smoking and implies asbestosis. This view was subsequently modified; and in a further study, it was concluded that in those heavily exposed to asbestos, cigarette smoking conferred an added risk for the development of irregular small opacities.

For many years the chest roentgenograms of certain subjects with chronic airflow limitation and emphysema have been described as showing “dirty lungs” or increased bronchovascular markings. The term, “bronchovascular markings,” is for the most part a misnomer; nonetheless, the term is recognized by many experienced clinicians as an indication of the presence of certain appearances that may be present in the chest roentgenogram of cigarette smokers who have airways obstruction.

A number of years ago, the Montreal group, in a carefully performed series of studies, coined the term, “increased markings emphysema.” This phrase was applied to a minority of subjects who on pathologic examination were found to have extensive emphysema, but whose roentgenograms did not show the characteristic obvious overdistention that characterizes emphysema. Most of their patients were fat and had cor pulmonale. Their chest roentgenograms had shown what was variously referred to as small irregular opacities, “dirty lungs,” or increased bronchovascular markings; and at autopsy, extensive lower lobe emphysema was present.

Among the factors known to influence the prevalence of small irregular opacities on the chest roentgenogram are radiographic technique, age, obesity, cigarette smoking, and exposure to various dusts encountered in the workplace. Small irregular opacities may be seen in asbestos workers in the absence of asbestosis, besides being present in various interstitial fibroses, the etiology of which are nonoccupational.

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tionally related. The contributory roles of these various factors in the perceived prevalence of small irregular opacities will be reviewed.

Radiographic Technique

Commonplace defects in the chest roentgenogram may cause overreading or underreading.\(^1^\)\(^2\) Also influencing the interpretation of roentgenograms for pneumoconiosis are an inadequate inspiration, bodily habitus, in particular excessive weight or height, and sundry technical factors. In obese subjects and in those who have not taken a full breath, the basal markings are often accentuated due to crowding together of the airways and vessels and appear as scanty irregular opacities. Furthermore, the basal markings of a normal nonobese subject are often exaggerated by an inadequate inspiration. This is well demonstrated in Figure 1, which shows the lower zones of the chest roentgenogram of a nondust-exposed laborer on a fruit farm who had been a lifelong heavy smoker. He had moderate airways obstruction and a reduced diffusing capacity due to emphysema. The roentgenogram taken at about two thirds of his vital capacity was interpreted by several experienced B-readers as showing interstitial fibrosis. Subsequently, when a second film was taken on the same day at full inspiration and in the absence of knowledge of the prior film, two of the three readers interpreted the repeat film as normal apart from overdistention, while the third awarded the film a significantly lower category. Similarly, when serial films are compared to assess progression, the assumed chronologic sequence has a profound influence on interpretation of roentgenograms for pneumoconiosis and also other chest diseases.\(^13^\)\(^14\) When a grid is used and is placed transversely, the x-ray beam may be deflected so as to produce transverse lines at the bases. Fogged films and poor screen contact are also a source of misinterpretation, especially in roentgenograms showing scanty irregular opacities.

Overpenetrated films tend to be underread, while the converse situation applies to underpenetrated films.\(^12\) Poor quality films, whether too light or too dark, may cause underreading, presumably due to a reluctance to read pneumoconiosis in a technically poor roentgenogram. In a study designed to assess the effect of film quality on the reading of chest roentgenograms for pneumoconiosis, three NIOSH-approved B-readers were asked to interpret two roentgenograms from the same subject taken on the same day.\(^12\) The second film had been taken because the first roentgenogram had been found on inspection to be unsatisfactory, and in order to obtain a better quality film, a repeat examination was requested. Some 150 paired films were selected from a large series of 4,000 roentgenograms that were being interpreted in a study of CWP. The films were randomized and interpreted without any knowledge that paired films were included. The study showed that overpenetrated films were underread, and vice versa for underpenetrated films. Although the original study was carried out in coal miners, many of whom had the typical upper zone opacities of CWP, the effect of a submaximal inspiratory capacity (IC) was greater when the opacities were basal and irregular, as they are in asbestosis. This impression was subsequently confirmed by taking a chest roentgenogram at about two thirds of vital capacity in six subjects with established mild asbestosis ranging from category 1/0 to 1/2.

Obesity

The presence of large pectoral muscles, gynecomastia, or excessive chest wall fat often leads to the assignment of a higher category for pneumoconiosis than that which is present and in some instances leads to pneumoconiosis being diagnosed when it is ab-
This is especially true for persons who are markedly obese. The effect of obesity on the reading of films for pneumoconiosis was investigated by Musch et al. and was shown to cause increased interobserver variation and overreading. In this study, three physicians with extensive experience of reading roentgenograms for pneumoconiosis interpreted 743 roentgenograms from a coal mining community in West Virginia, of which 392 films were from miners and 351 from nonminers. The percentage of films categorized by each reader as simple CWP varied from 8.5 percent to 15.3 percent. A significant number of obese nonexposed subjects were read as 0/1 or 1/0. When film quality was corrected by stratification, the effect of obesity on interobserver profusion agreement was no longer present.

**AGE**

The older the subject, the more likely that irregular opacities will be present in the chest x-ray film. Unfortunately, the effect of age is difficult to quantify, because as a subject ages, so does dust response increase and, in a smoker, so also do pack-years. Increasing age is likewise often associated with a gain in weight, which has been demonstrated to have an effect on the interpretation of films for pneumoconiosis. While it is often maintained that the use of multiple regression analysis will separate the effects of dust, cigarette smoking, age, and other confounding factors, that is not necessarily the case, especially when there is collinearity, i.e., when each of these factors is closely related and increases in a collinear manner.

**Cigarette Smoking**

It has been established that cigarette smoking is associated with the presence of irregular opacities. These opacities for the most part are found in the lower zones of the lungs, are of scanty profusion, ranging mostly from 0/1 to 1/1, and when present are frequently associated with some degree of airways obstruction. Such opacities may be present in the absence of dust exposure and have been shown to be present in female smokers. In this study the chest roentgenograms were not categorized according to the ILO classification, but the interpreters were able, for the most part, to determine in a series of roentgenograms from a population of smoking and nonsmoking female subjects which subjects were smokers; however, most studies have been concerned with quantifying the effects of cigarette smoking vs dust exposure and other factors. Thus, the effects of smoking can be best assessed from a review of those studies that have been carried out on dust-exposed populations and in which the specific purpose was to apportion the contributions of dust, cigarette smoking, and other factors to changes in the chest roentgenogram and pulmonary function.

**Coal**

The hypothesis has been put forward that irregular opacities are related to the inhalation of coal dust and are a manifestation of CWP. It was further suggested that in coal miners the presence of such opacities is associated with the presence of emphysema and that the latter can be a consequence of exposure to coal dust.

Ryder and colleagues published a survey correlating the pathologic, physiologic, and radiologic findings of emphysema in 247 deceased miners and ex-miners. These had been referred to the Pneumoconiosis Medical Panel (PMP) in Cardiff, and most of them had been diagnosed as having CWP during life. The investigators were able to correlate the appearance of the chest roentgenogram and the ventilatory capacity as measured during life with the degree of emphysema found at autopsy. They went on to state that in their opinion, "there is no reason why these deaths should not provide a true sample of the experience of men with this disease." This would imply that disability claimants are representative of coal miners as a whole in South Wales and, if it comes to that, with coal miners throughout the rest of the world, a statement for which there is no justification. Ryder and colleagues maintained that the extent of the emphysema as quantified by point counting was remarkably closely related to ventilatory impairment. They also suggested that, similarly, the severity of emphysema found at autopsy appeared to be greater in those miners who had pneumoconiosis, whether simple or complicated. The data of Ryder et al do not support the conclusion that irregular shadows are a common result of coal dust exposure in miners in the absence of other evidence of pneumoconiosis.

There were a number of anomalies present in the report of Ryder et al. Thus, their data indicated that the level of FEV, had little if any relationship to radiographic category, and moreover, the men with clear chest x-ray films had worse function than those with categories 1, 2, and 3 combined, and with complicated A. This would suggest that in simple CWP the more dust present in their lungs, the better the function, an observation that is clearly not tenable. Figure 3 of their report, which showed the relationship between the emphysema count and the final FEV, included subjects with both simple and complicated pneumoconiosis. Since complicated CWP is known to lead to the development of emphysema, the inclusion of miners with this condition is inappropriate. Finally, Ryder et al added gratuitously that the smoking history of the reference population was not available, but "that there should be no reason why
they should have a lighter smoking experience in life than have or had coal miners." Since no smoking history was available from the miners, this seems a dangerous assumption, in particular because in several subsequent studies that are referred to later, the percentage of smokers in those applying to the PMP for compensation was generally significantly higher than that in the general coal mining population. At the start of the Pneumoconiosis Field Research (PFR) in 1953, some 84 percent of British coal miners were smokers, but there has been a gradual decline in smoking in both Britain and the United States. In the United States, most coal miners who show significant lung impairment or who are claiming compensation are or have been smokers. This also seems to be the case in Britain, at least as far as significant impairment of ventilatory capacity is concerned.

A further report by Lyons et al concluded that impairment of ventilation was not related to radiologic category of small rounded opacities; however, these investigators found that impairment of ventilation was related to pathologic evidence of emphysema. There they suggested that the importance of round opacities had been overemphasized in the past and that more attention should be given to irregular or linear opacities, since these could be evidence of emphysema, which itself might be a feature of pneumoconiosis. Lyons et al did not present direct evidence that the emphysema was caused by exposure to coal dust.

Subsequently, a later report by Lyons and colleagues selected 95 deceased subjects from the original group of 247 miners whom they had studied earlier. This group consisted of all subjects who had simple CWP in the absence of progressive massive fibrosis (PMF) and had had a chest roentgenogram within four years preceding death. Lyons et al commented that although irregular opacities occasionally occurred alone, they were usually admixed with rounded opacities, and while there was a relationship between the prevalence of irregular opacities, the extent of emphysema, and the level of the FEV₁, this was not true for rounded opacities. The crucial question as to whether their data indicate that dust exposure is an important cause of irregular opacities and of emphysema remained unanswered. Unfortunately, dust exposures were not available, the smoking histories may have been biased because they were recorded during evaluation for compensation purposes, and inconsistencies such as frequent radiographic regression were noted. Moreover, there did not appear to be any direct evidence of a causal relationship between irregular opacities and dust exposure.

Cockcroft et al reported a pilot study of 46 men who had been referred for pulmonary function tests during life. The miners had been selected from subjects referred to the Pneumoconiosis Research Unit. Most were selected because they were short of breath, had p-type opacities, or had other symptoms. Thus, they were likely to be impaired and may well have been claiming compensation. Of this group of 46 men, there were two nonsmokers. There were 13 subjects in whom the predominant type of opacity was irregular. Cockcroft et al claimed that the presence of irregular opacities was associated with a reduced carbon monoxide diffusing capacity (Dco), a small decrease in TLC, an increase in the emphysema score, and, to a lesser but not significant extent, with the interstitial disease score. The higher emphysema scores were associated with a decreased Dco and an increased TLC; however, the mean TLC was only 103 percent of the predicted value in those subjects with the worst emphysema scores. Cockcroft et al concluded that the irregular opacities in coal workers represent a combination of emphysema and interstitial fibrosis. These investigators state that in their cohort, smoking was not associated with any particular pulmonary function abnormality, an observation that goes against all the available evidence. The association of irregular opacities with a low Dco in this report can easily be explained by the fact that the irregular opacities are associated with smoking and that smoking coal miners are more likely to have emphysema and a significantly reduced Dco. The TLC and RV also tended to decrease slightly as the frequency of irregular opacities increased and is best explained by the fact that the lung volumes were determined with helium.

A further report by Cockcroft and colleagues in 1983 studied 124 subjects, in 20 of whom irregular opacities predominated. All of the cohort were disability claimants and comprised 36 lifelong nonsmokers and 88 current smokers or ex-smokers. The proportion of nonsmokers in this cohort (29 percent) was exceptionally high because of their method of selection. The percentage of smokers and ex-smokers in British coal miners at that time was around 86 percent and for nonsmokers around 14 percent, respectively. Smoking habits were closely correlated to irregularity scores, and of the 20 subjects with higher irregularity scores, only one was a nonsmoker.

Cockcroft and Andersson in 1987 described a case control study involving 515 men who had been referred to chest clinics in various parts of England and Wales. Subsequently, the chest roentgenograms of 489 of the men were interpreted according to the ILO Classification. Of the 489 subjects, 189 were coal miners. It may be assumed that since the coal miners had been referred to chest clinics, virtually all of them had symptoms, and some were referred to establish whether CWP was present. No less than 14 percent
of the subjects had lung cancer. Since there was a higher percentage of lung cancer in the case controls, it might be inferred that the reasons for referral may have differed for the coal miners and the controls. Of the nonsmoking coal miners, one out of 12 subjects had irregular opacities, while in the noncoal mining nonsmokers, zero out of 37 had irregular opacities. This difference was not statistically significant. In the case of the smoking coal miners, ten out of 143 subjects had irregular opacities, while seven out of 239 smoking noncoal miners had irregular opacities. This study would suggest that irregular opacities are more commonly seen in coal miners than in the general population which is not exposed to coal dust, but that such opacities also occur, but less frequently, in the non-smoking coal mining population. Of great import is the fact that the smoking histories in 40 men were unknown. Of the 40 men, 23 were coal miners (12 percent of the total coal miners), and 17 were noncoal miners (5.6 percent of the total nonmining reference population).

Collins et al. studied the occurrence of irregular opacities in 895 coal miners. The subjects were chosen from a population of 3,600 coal miners from ten different coal mines who had been included in the PFR. The subjects included in the study were those who had remained in the industry for 15 years. Subgroups of subjects were selected according to whether they had high, medium, or low dust exposures. Unfortunately, data concerning lifelong smoking habits were not recorded, and the only smoking information available was the smoking habits at the third survey, i.e., 10 years after the study began. Of the 895 subjects, 39 (4 percent) were found to have predominant irregular opacities, of whom 35 were smokers or ex-smokers, and four were nonsmokers. Of the smokers, 5 percent had irregular opacities, as compared to 3 percent among the nonsmokers. The roentgenograms of 138 miners were excluded from the analysis, since there was no agreement as to the type of opacity. More importantly, it was shown that 15 so-called nonsmokers had been smokers according to prior questionnaires, and the authors make the point that the smoking histories were inconsistent. The presence of irregular opacities, but not rounded opacities, was associated with respiratory impairment and a reduction of ventilatory capacity averaging about 190 ml for both the FEV1 and the FVC; however, only four subjects with small irregular opacities were recorded as nonsmokers. As such, an alternative explanation for the presence of irregular opacities in one or more of these four subjects, for example, interstitial fibrosis or bronchiectasis was not sought. The rate of increase of pneumoconiotic response with respect to dust exposure was more severe in those subjects with only rounded shadows. Nonetheless, in the nonsmoking miners who were stratified by cumulative dust exposure, it was apparent that the profusion of small irregular opacities was related to dust exposure.

Only one study used data collected prospectively from a cohort of over 6,000 working US coal miners without any exclusions except for those subjects with PMF. Of the group, 85 percent had satisfactory chest roentgenograms and pulmonary function tests. The films were read by one observer only, and in the group, there were 59 nonsmokers with irregular opacities, of whom 31 had irregular opacities only, and 28 had an admixture. They constituted 3.4 percent of a total of 1,687 nonsmokers. In contrast, there were 287 smokers (6.4 percent of smokers) with evidence of irregular opacities, of whom 191 had irregular opacities only, and 96 had mixed opacities. In smokers the prevalence of irregular opacities was about twice as frequent as mixed opacities, while in the nonsmokers the proportion was roughly the same. The investigators also related the type of opacity, i.e., whether rounded or irregular, to pulmonary function tests, including static and dynamic lung volumes, to years spent underground (used in this instance as a surrogate index of dust exposure), and to cigarette smoking, among other factors. Small rounded opacities were strongly related to years spent underground, and to a much lesser extent to smoking, but were unrelated to bronchitis and age. Irregular opacities were strongly associated with the presence of bronchitis, age, and smoking and only slightly to years underground. When it came to pulmonary function, rounded opacities appeared to be unrelated to the FEV1; however, those with predominantly irregular opacities had a significantly lower FEV1. In smokers the FVC did not appear to be affected by the presence of irregular opacities, while in contrast the FEV1/FVC ratio was decreased and the RV significantly increased. In the nonsmokers with irregular opacities, neither the FEV1 nor the FVC was affected, but the RV and TLC were minimally, but not significantly, increased. The FEV1/FVC% did not appear to be affected by the presence of irregular opacities. In nonsmokers, there was a negligible association between irregular opacities and bronchitis and a weaker association between years spent underground and the frequency of irregular opacities.

All studies that have examined the prevalence of irregular opacities in a working population of miners have shown that they occur relatively infrequently in working coal miners. That such opacities were present in only 4.3 percent of the cohort examined by Collins et al. may be due to the fact that the sample was stratified by dust exposure and may not have been representative of British coal miners as a whole. Of the 39 subjects who had irregular opacities, 35 were smokers or ex-smokers, and four were nonsmokers.
Similarly, of the 6,166 miners studied by Amandus et al., 5.6 percent had irregular opacities either alone (3.6 percent) or mixed with rounded opacities (2 percent). The percentage of smokers with only irregular opacities was 4.2 percent, while the corresponding figure for nonsmokers was 1.8 percent.

It is thus clear that most studies of disability claimants in which the relative frequency of rounded and irregular opacities has been ascertained do not reflect frequency of occurrence of each type of opacity in a working population of miners. The investigations of Amandus et al. and of Collins and colleagues were the only studies carried out on working miners. In working miners the prevalence of irregular opacities is influenced by both cumulative dust exposure and by pack-years, with the evidence suggesting that the higher dust levels in the early British studies may have had a greater influence. In contrast, the relatively lower dust exposure in the US miners tended to emphasize the effects of cigarette smoking. It is also abundantly clear that in general, the predominant cause of such opacities in coal miners is cigarette smoking (Table 1).

In coal miners, the weight of the evidence suggests that irregular opacities are associated with age, cigarette smoking, bronchitis, and dust exposure, with by far the strongest influence being cigarette smoking except in the most heavily dust-exposed miners. The effects of dust exposure were evident whether cumulative dust exposure or a surrogate measure of exposure such as years underground was used. The effect of dust exposure is in the induction of irregular opacities is in general more tenuous than it is with small rounded opacities, and the former are rarely associated with a profusion of more than category I. In addition, there is little doubt that among coal miners with irregular opacities, the FEV₁ tends to be lower and the RV higher than in those miners with small rounded opacities or with a clear chest roentgenogram. In nonsmoking coal miners, irregular opacities are associated with a slight increase in the RV but no obvious effect on the FEV₁, the FVC, or the FEV₁/FVC. And in this, they resemble miners who have a clear roentgenogram. This is not to deny that nonsmoking coal miners may show a minor reduction in FEV₁ similar to that which is found in coal miners when they are compared to a reference population.

### Table 1—Irregular Opacities in Coal Workers

<table>
<thead>
<tr>
<th>Study</th>
<th>Group</th>
<th>Total Subjects (n)</th>
<th>Nonokers</th>
<th>Smokers and Ex-Smokers</th>
<th>Total with Rounded Opacities†</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Total Subjects (n)</td>
<td>n</td>
<td>Irregular Opacities</td>
<td>n</td>
<td>Irregular Opacities</td>
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<td></td>
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<td>No. with</td>
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<td></td>
<td></td>
<td></td>
<td>8 mixed</td>
<td></td>
<td>28 (2) mixed</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>31 (2)</td>
<td></td>
<td>191 (4) irregular</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>124</td>
<td>1 (3) predominantly</td>
<td>88 (71)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>46④</td>
<td>irregular; 21 (58) mixed</td>
<td>39 (85)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>758</td>
<td>4 (4) predominantly</td>
<td>653 (86)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>95</td>
<td>12 (13) No irregular</td>
<td>83 (87)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>6,166</td>
<td>1,687 (27) 8 mixed</td>
<td>4,479 (73)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>46④</td>
<td>Unknown</td>
<td>39 (85) 11 (25) predominantly irregular</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>758</td>
<td>105 (14) 4 (4) predominantly irregular</td>
<td></td>
</tr>
</tbody>
</table>

*Table data are numbers of subjects; numbers within parentheses are percents of group.
†Smokers, ex-smokers, and nonsmokers.
④Smoking history unknown in five subjects.

Similarly, of the 6,166 miners studied by Amandus et al., 5.6 percent had irregular opacities either alone (3.6 percent) or mixed with rounded opacities (2 percent). The percentage of smokers with only irregular opacities was 4.2 percent, while the corresponding figure for nonsmokers was 1.8 percent.

It is thus clear that most studies of disability claimants in which the relative frequency of rounded and irregular opacities has been ascertained do not reflect frequency of occurrence of each type of opacity in a working population of miners. The investigations of Amandus et al. and of Collins and colleagues were the only studies carried out on working miners. In working miners the prevalence of irregular opacities is influenced by both cumulative dust exposure and by pack-years, with the evidence suggesting that the higher dust levels in the early British studies may have had a greater influence. In contrast, the relatively lower dust exposure in the US miners tended to emphasize the effects of cigarette smoking. It is also abundantly clear that in general, the predominant cause of such opacities in coal miners is cigarette smoking (Table 1).

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### Silica

A number of studies have shown that workers exposed to silica may develop irregular opacities. Theriault et al. in their study of Vermont granite-shed workers, showed a relationship between rounded opacities and dust exposure, but such a relationship was not evident for irregular opacities. The presence of irregular opacities was strongly related to the presence of smoking, with 29 percent of their cohort showing irregular opacities. The increased RV noted in some of the granite-shed workers likewise was related to cigarette smoking, but not to the presence of rounded opacities. A repeat study carried out by Graham et al. using three different readers in the same group of subjects, confirmed that irregular opacities were likewise associated with cigarette smoking and only weakly, if at all, associated with other factors. Glover and colleagues have shown a similar relationship in the slate miners of North Wales. Here again, irregular opacities were more frequently found in older smokers, and this was true not only for the exposed group, but also for the reference group.

### Cigarette Smoking and Asbestos Exposure

There have been a number of reports which have suggested that smoking is associated with a greater prevalence of irregular opacities than is present in nonsmokers. This problem was thoroughly reviewed.
by Weiss. He pointed out that in 1963, Auerbach and colleagues carried out a large study of the histologic appearances of pulmonary tissue from over 1,000 autopsies in a VA hospital. The smoking, occupational, and residence histories were obtained from relatives, but the specimens were examined without any knowledge of these factors. Auerbach et al were interested in relating the frequency of ruptured alveolar septa, alveolar and peribronchial fibrosis, and thickening of the pulmonary arterioles to various exposures and, in particular, smoking. These investigators demonstrated a strong correlation between alveolar fibrosis, age, and smoking. The frequency of fibrosis in men who never smoked was negligible, and moreover, there was a dose-response relationship to cumulative cigarette smoking. Auerbach et al also noted bronchiolar wall thickening but was unable to show a relationship between the duration and number of cigarettes smoked, although bronchiolar wall thickening and changes were only present in smokers. Auerbach et al maintained that in those with substantial alveolar fibrosis, cigarette smoking was the prime etiologic agent.

Boucot et al subsequently compared 76 subjects with lung cancer, all of whom were smokers, with a matched sample of smokers without lung cancer and a further matched sample of nonsmokers. Matching was effected by age, race, date of entry into the project, and by pack-years in the smokers with and without cancer. The roentgenograms of all of the subjects were compared without any knowledge of their smoking habits or occupation. A further survey was conducted of 999 men and women for evidence of small irregular opacities. Some of these had minifilms carried out which were examined subsequently under magnification. While the percentage of nonsmokers who had evidence of radiographic changes suggesting fibrosis was around 1.5 percent, in the cigarette smokers as a whole, it was 4.4 percent. In the sample of those who smoked more than 20 cigarettes per day, the prevalence of irregular opacities was 14.3 percent. Weiss then compared the prevalence of asbestosis radiographically in 11 prevalence studies. He calculated a prevalence ratio of smokers to nonsmokers and showed that in all but two studies, there was an increased prevalence of "radiographic asbestosis" in the smokers. One study, that of Liddell et al was carried out in asbestos miners, and in it the matched response relationship differed significantly from that usually found in users of the end product. Weiss in addition reviewed seven cohort studies, and here again, there appeared to be a positive interaction between cigarette smoke and asbestos exposure. He concluded that smoking certainly may mimic the effects of asbestosis and that it may increase the fibrogenic effect of fibers that are retained in the lungs through interference with clearance.

The findings of Weiss were subsequently criticized by Kilburn and others on the grounds that Weiss had used the term, diffuse pulmonary fibrosis, as indicating accentuated bronchovascular markings that extended to the periphery of the lung. There seems little doubt that there is a distinction between the appearances created by extension of bronchovascular markings to the lung periphery and those seen in the roentgenograms that have been used to illustrate the ILO classification. Kilburn also suggested that the histologic changes caused by smoking, as identified by Auerbach and associates, would not be visible using standard radiographic techniques, especially in 70-mm roentgenograms. This criticism is not entirely convincing in that the average coal macule is not large enough to be seen on the chest roentgenogram, and, indeed, coal itself is not radiopaque; however, there is a summation and superimposition of macules which are almost certainly responsible for the production of the rounded shadows in CWP. Kilburn concluded that there was no evidence that smoking produces pulmonary fibrosis recognizable on the chest x-ray film. Kilburn was subsequently taken to task by Weiss who called attention to the fact that he had been criticized by Kilburn for not using the 1980 ILO classification in 1970! Nevertheless, in one group of asbestos-exposed workers whom Kilburn et al studied, an increased prevalence of parenchymal asbestosis was noted in the cigarette smokers when compared to the nonsmokers. These investigators proffered the explanation that smoking impaired the clearance of dust from the lung.

Subsequently, Hnizdo and Sluis-Cremer examined the relationship between cigarette smoking and irregular opacities in a case control study of 430 South African asbestos miners. These investigators concluded that their studies did not support the hypothesis that smoking is associated with the presence of asbestosis at autopsy or that smoking and asbestos dust act synergistically in producing an increased response to asbestos and hence an increased frequency of asbestosis at autopsy. They believed that cigarette smoking produced some change in lung structure that was sometimes visible on the chest x-ray film and that could be incorrectly and fallaciously interpreted as irregular opacities caused by asbestos-induced fibrosis. Their conclusions were subsequently disputed by Kraut and associates who criticized Hnizdo and Sluis-Cremer for presenting radiographic data from one group of asbestos miners and pathologic data from a different group. Kraut et al also suggested that other potential sources of bias existed and suggested that smoking asbestos miners may die before they develop asbestosis. Subsequently, each criticism, most of which involved selection bias, that had been brought
up by Kraut and colleagues was systematically and fairly convincingly rebutted. A recent report by Ducatman and colleagues emphasizes the difficulty of distinguishing the small opacities found in asbestosis from those found in nonexposed smokers. A more recent investigation concluded that in an asbestos-exposed population, cigarette smoking confers added risk for the development of irregular small opacities.

It therefore seems that the weight of the evidence indicates that cigarette-smoking asbestos workers can develop small irregular opacities in their lungs which closely mimic the changes of asbestosis. Such opacities may be present in the lungs of those exposed to asbestos but who have no evidence of asbestosis. These observations argue compellingly against the hypothesis that cigarette smoking delays clearance of asbestos fibers from the lung and that the hypothetical delay is solely responsible for the increased prevalence of irregular opacities in smokers.

**ALUMINUM**

A cross-sectional study of 788 male employees from an aluminum production company examined the relationship of radiographic abnormalities to smoking and dust exposure from the refining of bauxite to alumina. All of the roentgenograms were read by two B-readers without any knowledge of the smoking history or dust exposure. The predominant radiographic abnormality noted was the presence of small irregular opacities situated mainly in the lower zones and of limited profusion. The presence of irregular opacities was predominantly related to cigarette smoking and to a much lesser extent to cumulative dust exposure. Up to 7 percent to 8 percent of aluminum workers had radiographic findings of scanty irregular opacities.

**MAN-MADE MINERAL FIBERS**

Weill and colleagues described a study of the respiratory health of workers exposed to man-made mineral fibers. Of the 94 x-ray films that were categorized as having a profusion greater than 0/1, some 55 were read by at least two interpreters as having irregular opacities and 15 as having rounded as the primary opacity. Of the remaining 24, the two readers disagreed according to shape. Among current cigarette smokers the prevalence of small irregular opacities increased with length of employment. This was not evident among the nonsmokers. The investigators believed that it was unlikely the radiographic changes represented fibrosis.

**KAOLIN**

An increased frequency of small irregular opacities has been noted among china clay workers in Cornwall. This was noted by Oldham and more recently by Ogle and co-workers. The prevalence of irregular opacities was increased in cigarette smokers and ex-smokers, although dust may have also contributed. Similar findings have been described in kaolin workers from east central Georgia. In this instance, dust exposure could not be shown to be related to the occurrence of small irregular opacities; however, there was a relationship between cigarette smoking and the prevalence of such opacities.

**DISCUSSION**

The presence of small irregular opacities on the chest roentgenogram is associated with a number of factors, including age, cigarette smoking, and dust exposure. The majority of the evidence suggests that exposure to dust is associated with the development in low profusion of small irregular opacities. This appears to be a nonspecific effect in that it has been associated with exposure to a number of dusts, including coal, kaolin, bauxite, silica, and that generated during the production of man-made mineral fibers. In the case of asbestos, this mineral in sufficient quantities induces a specific response characterized by the presence of irregular opacities, but such opacities may be seen in asbestos workers in the absence of the interstitial fibrosis that characterizes asbestosis. With the exception of asbestos, any effects from dust-induced irregular opacities on pulmonary function are slight, and there is little to indicate that the presence of scanty irregular opacities in a nonsmoking dust-exposed subject ever leads to disabling impairment of pulmonary function. The suggestion that the occurrence of such opacities constitutes a form of pneumoconiosis and that in coal miners, their presence should be accepted as a criterion for compensation is based on tenuous evidence.

The recorded prevalence of irregular opacities is also associated with age and obesity, both of which are usually collinearly related to dust exposure and smoking. The most consistent relationship of such opacities in low profusion is to cigarette smoking, which is evident both in nondust-exposed and dust-exposed populations. In this regard, in those claiming disability, it must be borne in mind that smoking histories tend to be less reliable, and inferences drawn from data collected from such groups must be treated with caution.

A relatively low prevalence of irregular opacities has been found in nondust-exposed blue-collar workers in the South of the United States, and this observation has been used to argue against the proposition that such opacities may confound the interpretation of x-ray films for pneumoconiosis; however, the average age of the workers in this study was only 34 years. Thus, the fact that the population selected excluded those exposed to dust precludes any dust.
effect, and their relative youth precludes or obviates to a large extent any effect from cigarette smoking. Even so, those few subjects in the study who were noted to have irregular opacities were older, and almost all were cigarette smokers.

The precise pathologic changes leading to the development and presence of irregular opacities are unknown. Alveolar or peribronchial fibrosis and emphysema have been suggested as possible explanations for their occurrence. Weiss suggested that irregular opacities could be explained by the presence of alveolar fibrosis, although bronchiolar thickening may also be involved, and he based his opinion on the studies of Auerbach et al. Furthermore, Weiss suggested that when emphysema is present, it may accentuate the opacities, making them more obvious than they would be in its absence. Militating somewhat against this is the observation that irregular opacities may be seen in nonsmokers as an effect of dust and in the presence of normal ventilatory capacity and normal lung volumes, findings that make the presence of significant emphysema unlikely.

Centrilobular emphysema is the characteristic lesion seen in subjects with chronic airflow limitation due to cigarette smoking and usually affects the upper lobes, while irregular opacities are predominantly located at the bases. Nevertheless, emphysema may preferentially involve the bases, and this is particularly true in increased markings of emphysema. In this condition, irregular opacities are frequently present on the chest roentgenogram and are predominantly located at the bases. It has also been suggested that the opacities arise from increased "bronchovascular markings." In reality, this term is a misnomer, in that most increased markings are vascular in origin; and moreover, there is little or no pathologic evidence that the vascular changes found in emphysema provide an explanation for the occurrence of the opacities. Other explanations that have been put forward include the suggestion that pulmonary venous hypertension or, in certain instances, interstitial edema may explain the presence of irregular opacities.

The suggestion that cigarette smoking induces a delay in the clearance of fibrogenic particles, thereby permitting such particles to cause a greater fibrogenic effect, with the resultant development of irregular opacities, is likewise far from convincing. This hypothesis is discussed by Hnizdo and Sluis-Cremer in their report and in the subsequent correspondence. Moreover, irregular opacities occur in nondust-exposed populations, especially in smokers. By the same token, the presence of irregular opacities in nonsmoking dust-exposed subjects is associated with an altogether different effect on pulmonary function than that which is present in cigarette smokers. This was particularly evident in a study on working US coal miners in which not only dynamic lung volumes, but also static lung volumes were measured. The relative paucity of irregular opacities in dust-exposed nonsmokers is a consistent finding, and when present, any associated effect on pulmonary function has been either minimal or not demonstrable. Clearly, several factors are involved in the pathogenesis of irregular opacities, but there is no justification for assuming that their presence in an asbestos-exposed population is always related to asbestosis in the absence of the other criteria listed by the American Thoracic Society as necessary for diagnosing this condition. The same caveat must also be applied to their occurrence in the chest roentgenograms of coal miners, those exposed to silica, and workers in other dusty occupations.

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