LUNG FUNCTION WITH ASBESTOS-RELATED CIRCUMSCRIBED PLAQUES

EDWARD A. GAENSLER, M.D. • Peter J. Jederlinic, M.D. • Theresa C. McLoud, M.D.

Departments of Medicine and Surgery, Boston University School of Medicine and
Department of Radiology, Harvard Medical School, Boston, MA, USA

Early studies of asbestosis made no mention of plaques, and pleural calcifications were not identified with asbestos exposure until 1955.[10,12,22] In 1965 Selikoff[36] found a long latent period of 20 years and a high prevalence of 44%. Thereafter, pathologic studies showed no difference between plaques with and without calcification, and often plaques were found without microscopic asbestosis.[20,23] Radiographs showed plaques in only a small proportion of cases who had such lesion at autopsy.[20,37] Epidemiologically, it emerged that plaques can be caused by relatively slight household and neighborhood exposure.[8] Clinically, plaques early on were described as "harmlos-skurriler Schönheitsfehler," that is, a harmless beauty mark,[5] since they were neither precancerous lesions nor caused symptoms or loss of function.[5,7,23]

A number of studies since 1968[24] have dealt with the functional consequences of asbestos-related pleural disease but most often no clear distinction was made between circumscribed plaques and diffuse pleural thickening. Because fibrothorax of whatever cause may have serious physiologic consequences,[13,16,27,29,38] in this study we made a strict distinction between plaques and diffuse thickening. Also, we addressed the confounding effect of smoking; and for controls we used both normal subjects studied by the same protocol and employees matched for age and years of employment but without plaques.

METHODS

Clinical Material

We studied 1,764 persons during annual industrial surveys between 1966 and 1988 at two large shipyards, three papermills and one asbestos plant. Details of employment and type of exposure have been described elsewhere.[9,15,27]

Control subjects included two groups: *100 Normal unexposed males (group I)* 40 years or older without discernable lung disease and without prior asbestos exposure, who presented for pre-employment examination (Table I). The second group consisted of *154 persons exposed for 15 or more years* (group II) who had normal roentgenograms and who were selected from our survey group by matching for age and years since first exposure with group III which had plaques only and no diffuse thickening or asbestosis.

Survey Studies

On-site examination included a medical and detailed occupational history, a physician-administered respiratory questionnaire and chest physical examination.

Lung function studies included forced vital capacity (FVC), the forced expiratory volume in one second (FEV_1) and other flow derivatives, and the single breath diffusing capacity (D_L) with alveolar volume (VA) calculated from single-breath helium dilution. Instrumentation, unchanged over the years, has been described in detail.[17] FVC and FEV_1 were selected from the best of 3 efforts[2] and DL values were accepted if VA (BTPS) was at least 90% of FVC (BTPS). Predicted values were calculated from Morris et al.,[31] and for D_L from our own data.[18]

Chest roentgenograms, PA, lateral, and on at least one occasion oblique views, were obtained within one week of examination. Reading was according to the 1980 ILO scheme[21] by two "B" readers, one of whom was unaware of the nature and type of exposure. Films were read prospectively without recourse to other films ("apart reading"), and were reviewed later by display of the entire series of each case in order of date ("side-by-side" reading).

Definitions for this Study

Excluded were persons with significant non-asbestos intrathoracic disease, most often chest surgery, trauma, extensive pleural and parenchymal scarring from tuberculosis or residuals from infarction or pneumonia. Persons with heart disease we excluded only with marked cardiomegaly and/or evidence of chronic passive congestion.

Chronic obstructive lung disease (COLD) was diagnosed for this study only when the ratio of FEV_1 /FVC was more than 2 SD below the normal predicted.[1] We ignored lesser degrees of COLD and evidence of "small airways disease" which was observed in virtually all smokers and ex-smokers.

Circumscribed plagues were distinguished from *diffuse thickening* by detailed study of routine and oblique films aided by review of history, outside records, and CT scans in some cases.[27] Descriptions of Fletcher and Edge[11] were useful, but the ILO Film was of no help because we believe that the single example of "diffuse thickening" also represents a circumscribed plaque.[21] *Large plaques* were bilateral with width "b" and extent "2" or larger. *Large diffuse thickening* could be unilateral but had to be of width and extent 2b or larger. Asbestosis was graded according ILO major categories 0,1,2 and 3.[21] *Years since first exposure* included prior asbestos exposure. *Nonsmokers* had

Table I

Clinical Material for Plaque Study

Group	Definition	No COLD	Also COLD	Also Diffuse
I	Normal Unexposed, age over 40	100	0	0
II	Exposed > 15 Yrs, Normal X-ray (Matched For Age & Exposure With Group III)	129	25	0
III	Circumscribed Plaques Only	197	21	12
IV-VI	Plaques and Asbestosis 1,2 or 3	151	25	50

smoked less than 2 pack-years and had stopped at least 20 years earlier.

RESULTS

Among 1,764 persons in the survey we found 218 (12.3%) with circumscribed pleural plaques as the only abnormality (Group III), and there were 176 (10.0%) who had plaques and asbestosis (Groups IV–VI) (Table I). Additionally, 158 exposed persons with normal roentgenograms were matched for age and years since first exposure with the group with plaques (Tables I, II). COLD was found in 16.2% of those with normal X-rays, 9.6% with plaques only, and 14.2% among those with plaques and asbestosis. For some comparisons with normal unexposed persons the COLD group was excluded (Tables I–III, Figure 2). Mean age was similar for all exposed groups, but the 100 unexposed controls were 3 years younger. First exposure was 27 years ago for groups II–VI, and in this study this figure was virtually the same as total years exposed.

Lung function tests for the 100 controls were about 4% lower than predicted (Table II) because the Morris equations are for nonsmokers.[31] For the 40 nonsmokers in this group all function tests averaged almost exactly 100% (Table III). Mean values for the exposed controls (Group II) were slightly lower ($p < .05$).

With circumscribed plaques as the only abnormality (Group III) all three screening tests actually were slightly higher than the normal subjects ($p < .05$) or exposed persons without plaques ($p < .01$) (Table II). With progressing asbestosis there was the predictable precipitous decline, with D_L most severely affected. Inclusion of persons with COLD reduced mean function by only 2%–3% (Table II) because there were few such persons (Table I) and usually their obstructive disease was slight. COLD was no more common among persons with plaques than those without them (Table I). However, among all smokers and ex-smokers, including "normal" controls, both FEV_1 and D_L were significantly worse ($p < .01$) than among nonsmokers (Table III).

The fact that circumscribed plaques have no measurable effect on function was further documented by separation according to width and extent. Table IV and Figure 2 indicate no functional difference between large and small plaques.

Calcified plaques were seen in 80 persons (17.5%). This group was older and had longer employment by about 4 years. Nevertheless, the screening tests were virtually identical compared to the group with uncalcified plaques.

Diffuse pleural thickening initially was recorded from apart readings in 158 cases (Table V). Subsequent detailed study led to exclusion of 96 cases. Among these, subpleural fat pads were recognized more often following publication of the beautiful illustrations by Sargent et al.[35] Among the included 62 cases (3.5%) diffuse thickening was most often the residue of a benign effusion (Table V).

Diffuse thickening, unlike plaques, caused a significant loss of lung function and, unlike plaques, this loss was strongly related to extent and thickness, with bilateral cases most markedly impaired (Figure 2). Lung volume (FVC) was most severely affected, and D_L/VA often was larger than predicted as has been noted by others.[26,29,38]

DISCUSSION

The prevalence of asbestosis is declining rapidly, and most of the 254 cases (14.4%) in this series were the result of first exposure more than 38 years ago.[15] Circumscribed pleural plaques were more frequent (23.6%), and many were recognized among persons first exposed less than 38 years ago. This was, in part, because plaques can arise from lesser exposure and, in part, because of improved recognition of early lesions. Therefore, clinical and functional implications of plaques have become of increasing interest.

Published material does not provide a good overview of the physiologic effects of circumscribed plaques because, initially, attention was focused on calcifications,[24] and later the effect of plaques was obscured by inclusion of diffuse pleural thickening under the general term of "pleural changes."[3,6] This is not surprising because neither the 1958 nor the 1971 ILO schemes provided for separate quantification of these pleural reactions.[27].

Initial physiologic and pathologic studies of benign asbestos effusion showed that these often bilateral and often recurring bloody effusions frequently result in marked functional impairment and fibrothorax, sometimes so severe as to require decortication.[16,29] An epidemiologic study of effusions showed persisting radiographic changes: Among 34 persons there remained a blunted costophrenic angle in 91.4% and measurable diffuse thickening in 54.3%.[9] The serious con-

sequences of imprisoned lung, so well described in the days of tuberculosis and empyema,[13] have now been rediscovered in the asbestos-exposed under such fancy terms as "lung en cuirasse," "lung entrapment,"[29] "pleural hyalinosis complicata,"[32] or "squashed lung."[38] However, unlike pleural thickening after empyema or trauma, in asbestos cases the cortex may increase over the years,[28,38] probably from recurring subclinical effusions. Rounded atelectasis from effusion is also described in the asbestos exposed[4,30] and also may be associated with functional impairment. In all of these cases there was marked dyspnea, severe reduction of all lung volumes and D_L, and sometimes ventilatory failure. However, in contrast to pulmonary fibrosis, D_L/VA (sometimes called KCO) was normal, indi-

Table II

Pleural Plaques: Age, Years Since First Employment and Lung Function

Group	No.	Age	Years Since First Empl.	FVC % Predicted	FEV_1 % Predicted	D_L % Predicted
Excluding Obstruction, and Excluding Diffuse Thickening						
I	100	51.8 ± 10.7	0	94.5 ± 12.6	96.9 ± 13.3	95.3 ± 17.4
II	129	55.3 ± 8.1	27.9 ± 7.5	91.0 ± 13.6	97.3 ± 13.3	93.8 ± 17.9
III	197	53.8 ± 7.6	27.4 ± 5.9	96.1 ± 11.4	101.8 ± 13.6	101.6 ± 13.9
IV	112	56.1 ± 7.6	27.4 ± 6.1	82.3 ± 11.3	87.5 ± 12.5	80.2 ± 16.5
V	31	59.5 ± 8.1	29.0 ± 7.0	73.6 ± 16.7	77.6 ± 17.7	58.1 ± 13.6
VI	8	56.8 ± 6.6	23.4 ± 7.9	53.9 ± 16.8	58.7 ± 20.1	45.8 ± 11.1
Including Obstruction, Excluding Diffuse Thickening						
I	100	51.8 ± 10.7	0	94.5 ± 12.6	96.9 ± 13.3	95.3 ± 17.4
II	154	55.5 ± 7.7	28.3 ± 7.4	90.5 ± 14.3	93.6 ± 17.2	91.0 ± 19.1
III	218	54.0 ± 7.5	27.4 ± 6.3	94.5 ± 12.7	98.7 ± 16.8	100.4 ± 14.7
IV	128	56.3 ± 7.3	27.4 ± 6.8	81.6 ± 12.5	84.9 ± 15.4	78.4 ± 17.0
V	40	59.5 ± 7.3	28.9 ± 7.1	74.4 ± 17.1	75.2 ± 19.1	58.4 ± 14.0
VI	8	56.8 ± 6.6	23.4 ± 7.9	53.9 ± 16.8	58.7 ± 16.8	45.8 ± 11.1

Table III

Pleural Plaques: Effect of Smoking

Excluding Diffuse Thickening, Including Obstruction

Group		No.	Smokers	No.	Nonsmokers
I	FVC %	60	92.4	40	97.8
II		110	90.5	44	90.5
III		163	93.8	55	96.8
IV		107	82.4	20	77.6
V		36	75.6	4	63.8
VI		7	55.3	1	44.0
I	FEV_1 %	60	93.4	39	102.4
II		110	91.1	44	99.7
III		163	97.4	55	102.6
IV		107	84.9	20	84.6
V		36	75.4	4	73.0
VI		7	60.1	1	49.0
I	D_L %	60	91.6	39	101.0
II		110	86.5	44	102.2
III		163	98.8	55	105.0
IV		107	77.3	20	86.3
V		36	57.8	4	64.1
VI		7	47.7	1	32.4

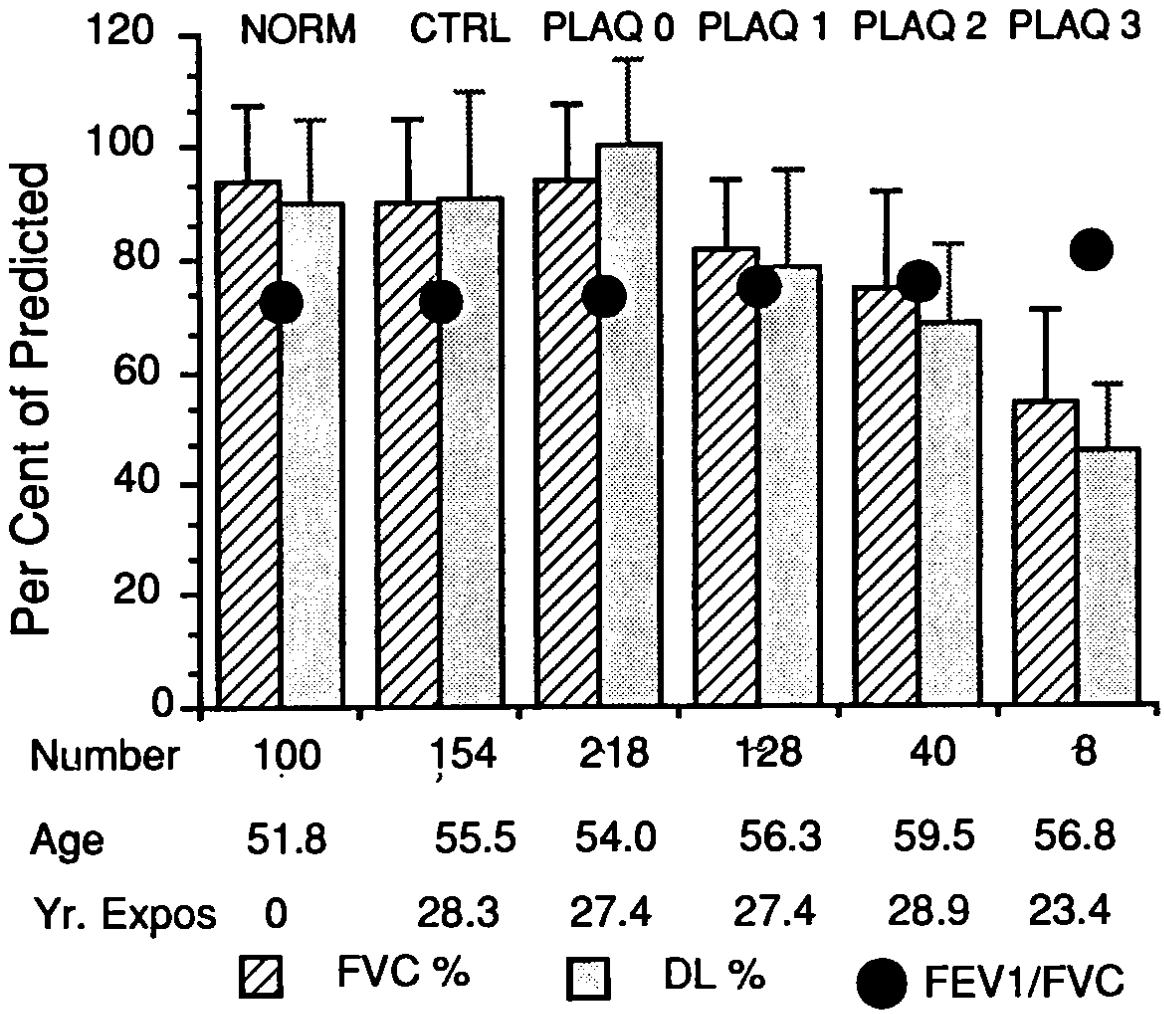

Figure 1. Lung function with plaques and asbestosis. Forced vital capacity (FVC) and diffusing capacity (D_L) as percent of predicted, and the FEV_1/FVC ratio of 100 normal unexposed males over age 40 (NORM), exposed males with normal roentgenograms matched for age and years since first exposure (CTRL), 218 males with circumscribed hyaline pleural plaques only (PLAQ 0), and 176 persons with plaques and varying degrees of asbestosis (PLAQ 1,2,3). This representation includes persons with chronic obstructive lung disease while persons with diffuse pleural thickening have been excluded.

cating that reduced D_L was the result of reduced lung volume and not of impaired respiratory gas exchange. From the foregoing it is evident that inclusion of but a single case of this nature in a group largely with circumscribed plaques would have a significant effect on average values of lung function.

A larger series of diffuse thickening was reported by McGavin and Sheers.[26] They, like Britton[6] before them, devised a grading scheme to measure radiographic extent of diffuse thickening and found impairment of FVC and D_L closely related to severity score. Calcifications alone had no significant effect on function.[24,25]

SUMMARY AND CONCLUSIONS

In 218 persons with circumscribed pleural plaques, but without diffuse thickening or apparent asbestosis, we found lung function with respect to volume, flow and gas exchange no different than that of 154 persons matched for age and years since first exposure who had no visible plaques, and no different than that of 100 unexposed normal subjects. Inclusion of persons with chronic obstructive lung disease in the exposed groups did not alter results significantly. There was no difference among persons with and without calcification of plaques, and no difference between large and small plaques. With plaques and increasingly severe asbestosis there was the predictable progressive functional impairment.

Diffuse pleural thickening, unlike plaques, caused significant functional loss, especially with regard to lung volumes. This is because pleural plaques involve only the parietal pleura, do not cause adhesive pleuritis, and are patchy and interrupted structures that do not interfere with thoracic motion. Diffuse thickening, on the contrary, involves both visceral and parietal pleurae forming an uninterrupted fibrous peal with granulation tissue that extends to involve cortical interlobar septa, and seriously interferes with motion of both

Table IV

Circumscribed Plaques: Effect of Size and Extent (Cases with Diffuse Thickening or Obstruction Excluded)

Group	No.	Age	Years Since First Empl.	FVC % Predicted	FEV_1 % Predicted	D_L % Predicted
Small Plaques (< 2b)						
III	100	53.6 ± 6.9	27.5 ± 5.5	96.0 ± 11.6	102.5 ± 13.1	101.0 ± 14.4
IV	27	55.9 ± 7.5	28.5 ± 7.2	81.3 ± 9.0	89.4 ± 8.8	75.1 ± 17.8
V	7	62.4 ± 6.8	31.4 ± 6.9	74.7 ± 23.2	74.3 ± 16.7	58.8 ± 9.5
VI	4	59.3 ± 5.1	26.5 ± 8.5	58.8 ± 18.7	65.8 ± 22.6	47.5 ± 10.7
Large Plaques (> 2b)						
III	97	54.0 ± 8.3	27.4 ± 6.4	96.3 ± 11.1	101.2 ± 14.1	102.2 ± 13.4
IV	35	56.4 ± 5.9	27.9 ± 5.3	80.3 ± 10.6	86.5 ± 12.0	84.7 ± 17.2
V	11	58.4 ± 9.5	29.5 ± 7.0	71.4 ± 19.3	76.0 ± 19.3	49.2 ± 12.1
VI	4	54.3 ± 7.7	20.2 ± 6.9	49.0 ± 15.7	51.8 ± 17.2	44.0 ± 13.0

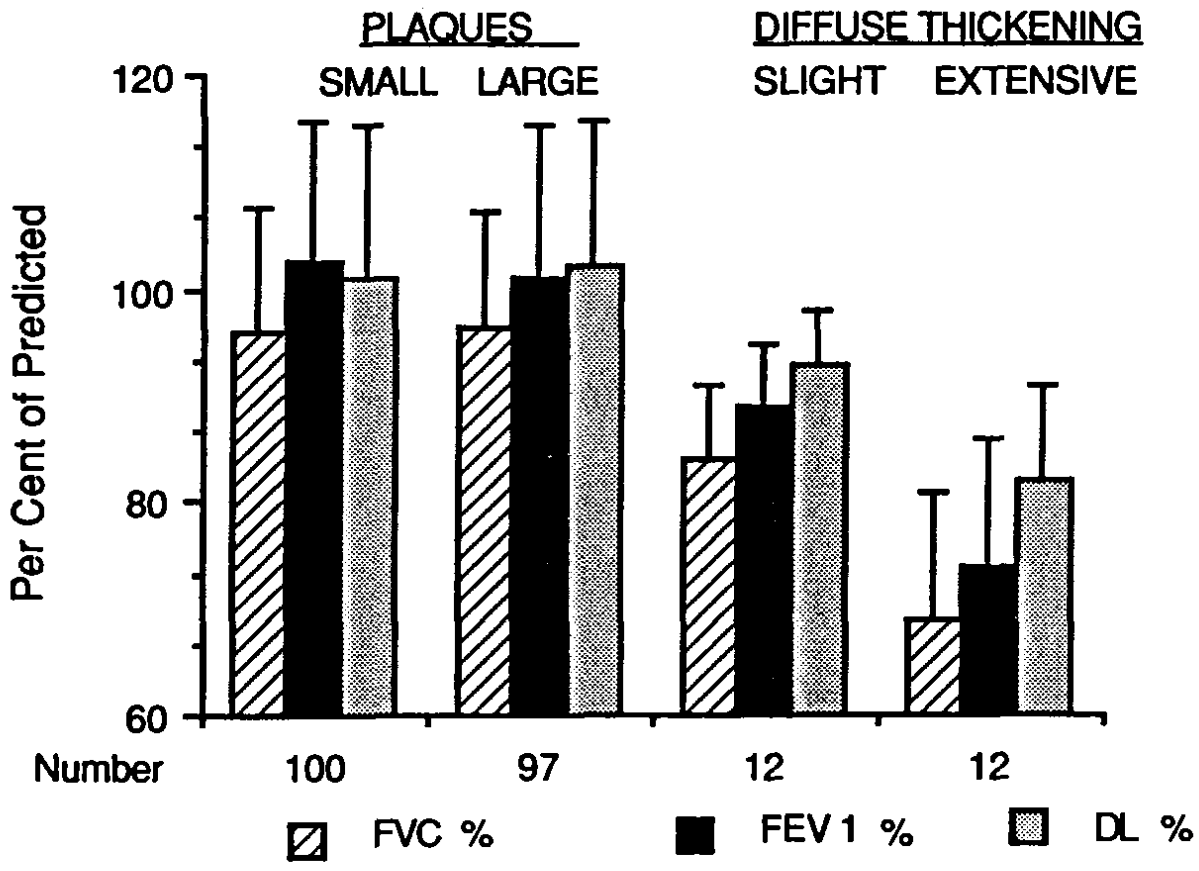

Figure 2. Lung function: plaques and diffuse thickening. The effect on lung function of asbestos-related pleural disease. Forced vital capacity (FVC), forced expiratory volume in one second (FEV_1) and diffusing capacity (D_L) were all in the normal range for persons with circumscribed plaques, and there was no difference between small plaques (<2b) and large plaques (>2b). Persons with diffuse pleural thickening had a significant functional deficit, and this was greater with extensive diffuse pleural disease.

Table V
Causes of Diffuse Thickening

Initial Coding from Apart Readings	158
Excluded After Further Study	96
Actually Confluent Plaques	40
Malignancy	5
Infection, Trauma, Surgery	22
Subpleural Fat Pads	18
Included as Diffuse Thickening	62
After Benign Effusion	41
Related to Asbestosis	15
Unexplained	6

lungs and thorax. The cause of diffuse thickening in most cases could be traced to one or more episodes of benign asbestos effusion. Our studies confirm the old dictum of Bohlig et al[5] that plaques are an epidemiologic leading fossil for asbestos exposure, but otherwise are merely a beauty mark and without functional consequences. We also agree with McGavin and Sheers[26] that diffuse thickening at times may lead to significant pulmonary insufficiency and then represents an industrial injury even when there is no asbestosis.

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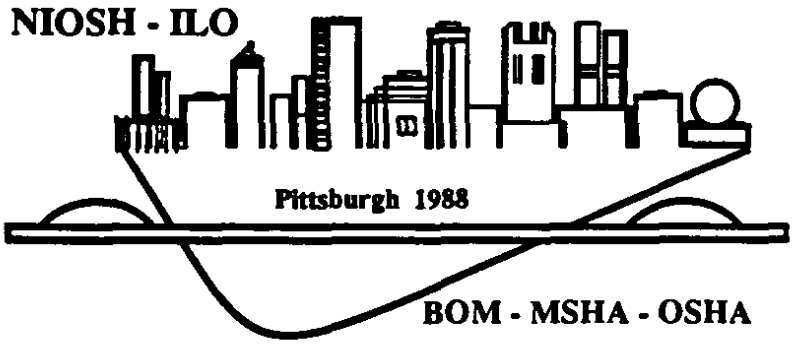

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