Byssinosis in a Nontextile Worker[1,2]

JOHN D. HAMILTON, VICTOR H. GERMINO, JR., JAMES A. MERCHANT, JOHN C. LUMSDEN, and KAYE H. KILBURN

SUMMARY

A nontextile worker was observed on 4 successive days in a cotton textile mill for the development of respiratory systems and changes in pulmonary flows and arterial oxygenation. The subject had a history of childhood asthma.

Acute, profound decreases in forced expiratory volume in 1 second and arterial oxygen tension were noted within 15 minutes of entering the mill and were correlated with the development of severe shortness of breath. On successive days, comparable objective changes were noted but without as severe shortness of breath.

These findings suggested that a history of asthma may increase the likelihood of reaction to cotton dust, that a prior period of sensitization to cotton dust is probably not essential, that no objective improvement was noted on successive days of exposure, and that very severe changes in pulmonary flow and arterial oxygenation may occur in a nontextile worker.

Introduction

Among the unanswered questions concerning byssinosis are the role of atopy in susceptibility, the nature of tolerance to systemic effects of cotton dust, and whether blood gas abnormalities accompany the characteristic bronchoconstriction of byssinosis.

Study of a subject with a history of childhood asthma but without occupational exposure to a cotton textile mill environment offered an opportunity to make observations about these questions. The subject, one of the present investigators (JDH), visited several textile mills during a 2-year period for brief intervals to study textile workers. Symptoms and objective measurements were made on these occasions.

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1 From the Division of Environmental Medicine, Department of Internal Medicine, Duke University Medical Center, Durham, North Carolina 27710.

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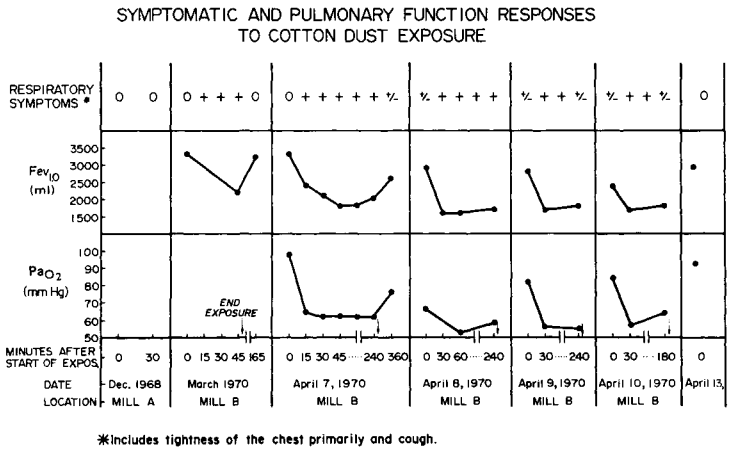
Case Report

The subject was a 31-year-old, pipe-smoking male with a history of childhood asthma and hay fever who had had no exposure to the cotton mill environment before December 1968. That exposure for 30 minutes was unaccompanied by subjective complaints (figure 1). No objective measurements of pulmonary function were made. The exposure was in a different cotton mill (Mill A) than were later exposures. Total dust and respirable dust concentrations were both less in this mill (table 1) (1).

Re-exposure in March 1970 for 45 minutes reduced vital capacity (VC) from 4,400 to 3,800 and forced expiratory volume in one second (FEV_1) from 3,300 ml to 2,200 ml (figure 1). The onset of respiratory symptoms was apparent within 15 minutes of entry into the mill and symptoms were intensified with movement into "dustier" portions of the mill. Complete recovery occurred 1.5 to 2 hours after exposure ceased.



*Includes tightness of the chest primarily and cough.

Fig. 1. Symptomatic and pulmonary function responses to cotton dust exposure.

No symptoms suggestive of "mill fever" were noted despite exposure in nearly all parts of the mill.

Several weeks later (April 7 to 10), the subject entered the same mill and remained adjacent to the operating carding machines for 4 consecutive hours on 4 consecutive days. Measurements made before entry into the mill and at 15- to 30-minute intervals thereafter included temperature, leukocyte count, eosinophil count, arterial blood gases in the sitting or standing posture, i.e., pH, arterial carbon dioxide and arterial oxygen tensions ($Paco_2$ and Pao_2), FEV_1, and VC, using the best of 3 trials with a Jones Pulmonor (waterless wedge spirometer).

The symptomatic course, FEV_1 and Pao_2 measurements made during the course of the 4-day exposure are summarized in figure 1. Pre-exposure measurements on the first day revealed a normal temperature, leukocyte, and eosinophil count; VC was 5,200 ml, FEV_1, 3,300 ml, and Pao_2, 99 mm Hg; approximately 30 minutes after exposure began, FEV_1 had decreased by 1,100 ml (42 per cent of baseline) and Pao_2, by 37 mm Hg (40 per cent of baseline). These low levels were maintained during continued exposure. Shortness of breath with tightness in the chest and minimal cough characterized the symptomatic response to exposure. No significant changes in temperature, leukocyte count, or eosinophil count were detected. After 4 hours, exposure was discontinued, and during the next hour FEV_1, VC, and Pao_2 improved, as did the symptomatic response, although not to pre-exposure levels.

By the next morning (April 8), after a restless night with some continued mild tightness of the chest, FEV_1 had further improved, although not to baseline, and Pao_2 was lower than postexposure on the previous day. On this second day, exposure produced smaller absolute changes from baseline but further decreases below the lowest measurement on the previous day. Changes from

TABLE 1

DUST LEVELS IN COTTON MILLS A & B

	Opening Picking (mg/m^3)	Carding (mg/m^3)	Spinning (mg/m^3)	Winding (mg/m^3)
Mill A*				
Total	5.14†	0.23–0.35	1.84	–
Respirable ($\leqslant 10\ \mu$)	0.13–0.24	0.11–0.12	0.02	–
Mill B*				
Total	8.2	3.2	7.4	14.3
Respirable ($\leqslant 10\ \mu$)	1.0	0.3	0.3	0.3

*Mill A: One sample in each room by a cyclone fractionator. Mill B: Average of multiple samples in each room by cyclone fractionator.

†Picking room only.

baseline on this second day were comparable to the first day when expressed as per cent change in FEV_1 (47 per cent), and somewhat less for per cent change in Pa_{O_2} (25 per cent).

Similar findings were noted on each day, but symptomatic responses were milder later in the week despite reproduction of similar abnormalities of pulmonary function. After a 3-day rest period, all of the parameters had returned to pre-exposure levels.

Discussion

McKerrow and Molyneux reported a study of 12 medical students exposed for the first time to cotton dust (2). Symptomatic and objective (fever or decreased $FEV_{0.75}$, increased airway resistance, or both) responses were found in all but 2 subjects. They concluded that on first exposure to cotton dust, both subjective and objective evidence of byssinosis can develop. A history of atopy among these subjects was not reported.

It is not possible from the present study to conclude that a prior history of atopy confers a sensitivity to cotton dust. There is no doubt, however, that this subject with a history of atopy but no dust exposure did exhibit the criteria for classification as Grade II byssinosis according to the criteria of Schilling (3), as well as remarkable changes in FEV_1 and Pa_{O_2}.

There were several features of this response pattern that are atypical of other textile workers surveyed (4). In addition to the uncommonly large change in FEV_1 after exposure to cotton dust, the rapidity of the change would also be considered unusual when compared with the expected changes in textile workers. This may indicate a different mechanism in this subject from that occurring in workers with byssinosis, or it may represent selective migration of symptomatic workers out of dusty textile mills. It is unlikely that many textile workers with an initial response to cotton dust such as the one described here would remain working in dusty areas.

This is the first measurement of a reactive subject's arterial blood gases after exposure to cotton textile dust in a manufacturing plant. The blood oxygen tension decreased precipitously to levels below optimum for this subject. Probably such dramatic reductions would not be tolerated by subjects with less reserve than the subject in this report. If such a reduction in gas transfer occurs in other subjects who react to dust, it might result in reduced work capacity. Further, oxygenation of the heart may be decreased, and angina or arrhythmias induced.

Subjective but not objective tolerance developed during subsequent days of exposure. The magnitude of changes was less on successive days, although the low level end-point remained essentially the same throughout the week. This finding would seem best explained by continued activity of the provocative agent each day, incomplete recovery during the night, smaller reductions in pulmonary function on successive days, and, perhaps to a rather large extent, psychologic adaptation to adverse conditions with subconscious adjustment of activity level and work expenditure.

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