“Inhalation of cotton dust may also result in chronic lung disease.”

In his discussion of prospective studies of cotton dust exposure, incomplete or misleading information is again brought forward. Berry et al. highlighted through the use of figures the problems presented by selection, even in a careful prospective study. One figure demonstrated a clear increase in decline in FEV₁ among employees exposed for less than five years, which then declined with longer exposure only to increase among those with the longest exposure. The second figure pointed out a rather greater decline among cardroom workers, even though they found no significant association between decline in FEV₁ and dust or whether workers had symptoms of byssinosis. These findings are consistent with the frequent reports of selection of affected workers away from cotton dust exposure—which tends to make Berry’s significant finding of a 54 ml/annual decline in FEV₁ among their cotton mill workers, compared with a 32 ml decline among synthetic mill workers, all the more significant. Jones goes on to dismiss observations we made in a ten-month cotton steaming intervention study as “clearly too short to allow much confidence in the estimates of lung function change.” Yet he failed to note the most relevant data in that study which demonstrated a dose-response relationship between dust concentration (measured monthly) and decline in FEV₁ (measured monthly in a panel of workers and quarterly in the remainder). These data are provided to illustrate this point:

<table>
<thead>
<tr>
<th>Work Area</th>
<th>Vertical Elutriator median dust level—mg/m³</th>
<th>Decline in FEV₁ over 10 month study—ml</th>
</tr>
</thead>
<tbody>
<tr>
<td>Opening/Picking</td>
<td>13/31 2.65</td>
<td>−292</td>
</tr>
<tr>
<td>Carding</td>
<td>45/63 0.65</td>
<td>−145</td>
</tr>
<tr>
<td>Spinning</td>
<td>70/106 0.48</td>
<td>−88</td>
</tr>
<tr>
<td>Winding/Twisting</td>
<td>65/108 0.52</td>
<td>−67</td>
</tr>
</tbody>
</table>

*(n)* provides a denominator of those who began the study and a numerator of those completing the study providing a measure of outward migration by work area.

These data also provide evidence of increased selection away from the dustiest work area in which a 10-fold increase in expected annual decline in FEV₁ was observed. Finally, Jones attempts to discredit the studies of Beck et al. as being biased due to “uncontrolled referral and/or self-selection” without recognition of recent discussion of that point by Beck et al that supports their earlier original conclusions.

Jones further suggests that I “seek to favor a Brown Lung Law drawn on lines similar to the existing Black Lung Law” which misrepresents what was written in my editorial for the Journal of the American Public Health Association. What I believe epidemiologic data support is compensation for textile workers with cotton dust exposure prior to the introduction of adequate dust control. Remarkable progress has been made in the American textile industry in the control of cotton dust. Whether workers exposed in work areas compliant with all aspects of the Cotton Dust Standard, which includes systematic medical surveillance, will develop progression in decline in lung function is doubtful, but presently unknown. This is the subject of a study of Burlington Industry Plants (known as a progressive company with a corporate medical and industrial hygiene program for 14 years) by Drs. Weill, Jones and colleagues. While this is an important study which will address the question of whether the Cotton Dust Standard is doing its job as applied in one progressive company, it will not address the question of the cotton dust exposures which existed in the 1960s and 1970s, the time of the several quoted epidemiologic studies. Hence, my concern is principally for those workers exposed to higher dust levels during this period, but who still remain in the industry or have recently left the industry. I have recommended two approaches to compensation for such workers: (1) through work with the North Carolina Industrial Commission to establish cotton dust exposure criteria and a medical panel, and (2) through Congressional testimony on comprehensive minimum federal compensation standards for diseases found to be occupationally related. In 1969 we developed cotton dust exposure criteria and a Byssinosis Medical Panel to work with the North Carolina Industrial Commission. This arrangement continues today and has resulted in compensation for several hundred North Carolina textile workers. Unfortunately, such a process has not been adopted by other states where very few workers’ compensation claims have been awarded. Hence, the need for minimum federal compensation standards for exposure to cotton dust, asbestos, silica and many other occupational hazards.

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REFERENCES


To the Editor:

Authors, on the subject of their own work, sound much like doting parents on the subject of their own offspring; rather more attentive to merits, and less sensitive to faults, than is the general observer. It thus comes as no surprise that Schachter and colleagues hold their study in the highest regard, and give it the greatest possible evidentiary weight. Conversely, I and other writers have given the work less weight. This reflects only a difference of opinion, not a misinterpretation of facts.

More interestingly, Schachter and colleagues label my gibe at the Black Lung Law as haughty and superficial. My own view is that the Black Lung Law casts its sooty pall over all better efforts to find solutions to problems of workers’ compensation. This may not be entirely bad, because disease-specific compensation laws are urged upon us under two assumptions, both dubious. The first is that workers in a particular industry, when they fall ill, are especially deserving of compensation, in comparison to sick workers in less favored industries. The second is that the villainous employer who caused the illness can be made to bear the entire financial burden resulting from his mischief. The Black Lung Law is a standing refutation of the latter, raising energy costs that all must bear, and siphoning billions more from tax revenues. It is also a massive proof of how quickly and completely the medical bases for diagnosis and assessment of an occupational illness can be abandoned, in favor of the legislative ukase and the administrative fiat. It is therefore a source of constant amusement that proponents of a Brown Lung Law speak highly of the Black Lung Law, failing to recognize it as the chief obstacle to the realization of their own wishes.

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Carboxyhemoglobin Levels in Banked Blood

To the Editor:

I am concerned that Aronow et al (Carboxyhemoglobin levels in banked blood. Chest 1984; 85:694-695) have “identified” and proposed a solution to a problem which does not exist. The authors have confirmed the work of Stewart et al and Spieb et al who also found banked blood to have elevated carboxyhemoglobin (CHB) levels with a frequency similar to that of the general population. Dr. Aronow concludes that the CHB content of banked blood should be determined and that banked blood containing relatively high levels is hazardous. Implicit in the authors’ concern, and that of many of the papers they cite is the mistaken notion that organ dysfunction associated with exposure to carbon monoxide (CO) is a result of elevated levels of CHB. This traditional view was laid to rest by Goldbaum et al in an elegantly simple study published in 1975.

Goldbaum’s group were stimulated by the wide variation in CHB levels reported to the Armed Forces Institute of Pathology in association with fatal aircraft accidents. They observed that some patients died as a result of CO poisoning with carboxyhemoglobin levels of 50 percent, while other persons were virtually asymptomatic at the same level. Conversely, severe toxicity occasionally occurred at levels of only 10 percent to 20 percent CHB. This suggested that the mechanism of CO toxicity might not primarily involve hemoglobin. Their investigation demonstrated in dogs that exchange transfusion using autologous blood containing 80 percent carboxyhemoglobin to achieve a level of 60 percent CHB in the recipient animal resulted in no clinical illness.

Supporting work by Ball et al and Chance et al demonstrated earlier that carbon monoxide interferes with the activity of cytochrome oxidases. There is a specific competition of CO with oxyhemoglobin to form the cytochrome a, an essential link in the cellular electron transport chain. It appears that this causes clinical illness.

Goldbaum and associates also demonstrated that although CO does dissociate from hemoglobin approximately 210 times less readily than does oxygen, the initial combination of carbon monoxide with hemoglobin is very slow. Complete saturation of hemoglobin by CO in vitro requires well over 20 minutes. Thus, plasma can carry large quantities of CO to the tissues without necessarily causing a striking increase in CHB levels.

The studies “documenting” adverse effects of even relatively low CHB levels in patients with significant pulmonary and cardiac disease are flawed by the assumption that it is the CHB per se that aggravates cardiopulmonary dysfunction. On the contrary, the evidence from Goldbaum’s work strongly suggests that relatively high levels of carboxyhemoglobin cause little or no decrement in cardiopulmonary performance. It is the tissue levels of carbon monoxide which produce clinical illness. Indeed, it is intuitively unattractive to imagine that a change in carboxyhemoglobin level from 1 to 2 percent could cause a significant decrease in oxygen transport. (That such a level might alter the ease with which hemoglobin unloads oxygen to the tissues has been quoted repeatedly in the literature dealing with CO. That speculation seems to be based on an early in vitro study by Boughton and Darling which, in fact, suggested minimal if any effects at low levels of CHB).

Finally, the authors refer to “air quality standards” for CHB levels in blood. This writer is not aware of “air quality standards” as such dealing with CHB levels. The United States Department of Labor (Occupational Safety and Health Administration, OSHA) and the US Department of Health and Human Services (National Institute for Occupational Safety and Health, NIOSH) do offer guidelines for permissible exposure limits (PEL) for CO. The current OSHA standard for carbon monoxide is 50 parts per million (ppm) of carbon monoxide averaged over an eight-hour work shift. NIOSH has recommended that the permissible exposure limit be reduced to 35 ppm. These levels can result in blood carboxyhemoglobin levels of up to 10 percent after an eight-hour exposure. The United States Environmental Protection Agency has identified “danger levels” for CO. These specify a one-hour mean maximum of 35 ppm and an eight-hour mean not to exceed 9 ppm. They are intended to keep the CHB levels of the nonsmoking population at large less than 2 percent. All such guidelines tend to ignore our improved understanding of the mechanism of carbon monoxide toxicity. Nonetheless, the dependence on CHB levels as a crude index of the extent of exposure to carbon monoxide is a pragmatic concession to available technology.

In summary, plasma is a transport medium which can carry a great deal of carbon monoxide to the tissues before it has a chance to bind to hemoglobin. As a result, the blood carboxyhemoglobin level is probably of very minimal importance in CO poisoning.

Ten million units of blood are prepared for transfusion in this country each year. Determination of a CHB level on each unit would increase the cost of medical care in this country by a minimum of $50 million annually. I urge the authors to reconsider their recommendation that such determinations be done. This writer believes there is no demonstrable benefit in performing such determinations, even when the intended recipient is severely compromised.

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