Editorials

Occupational Respiratory Disease

The respiratory tract is, more than any other organ system with the exception of the skin, an important target for hazardous agents encountered in the work environment. Thus, respiratory conditions traditionally feature very prominently among the occupational diseases.[1,2] One of these is byssinosis, a fairly distinctive condition found in textile workers exposed to the dust of cotton, flax or hemp.[3] The study entitled 'Byssinosis in a Bombay textile mill' by Murlidhar *et al.*[4] in the present issue of this journal provides an excellent opportunity to emphasize a number of important features regarding present-day occupational respiratory diseases.

First of all, the study shows that disabling respiratory disease due to occupational exposure does occur!

In industrially advanced countries, many laymen and even physicians tend to consider that occupational lung diseases are a thing of the past, because they believe that working conditions have improved so much that no serious occupational diseases can occur in modern industry. In less industrially advanced and poorer countries, the burden of infectious respiratory diseases, including tuberculosis, is such that occupational causes of lung disease are often not taken into consideration.

The reasons for this 'ignorance' are many. One reason is of a sociological nature: generally, physicians have little first-hand experience of manual work in mines, factories, workshops or farms. Another reason, which is undoubtedly linked to the first one, is that medical schools traditionally devote little time to the teaching of occupational determinants of disease.[5] Finally, and perhaps most importantly, occupational diseases, including respiratory diseases, are rarely so specific that their occupational aetiology is immediately obvious to the clinician. Thus, as shown in the study of Murlidhar *et al.*,[4] and also in other published studies on byssinosis, e.g. from China,[6] Cameroon[7] and Belgium in the late fifties,[8] patients with byssinosis are often thought to have tuberculosis. Similarly, there is anecdotal evidence of a high incidence of 'tuberculosis' among diamond workers in India, but this may, in fact, be due to the use of cobalt-containing polishing disks which have been shown to cause interstitial lung disease.[9,10] Another example of missed occupational aetiology is that of occupational asthma, which in western countries has become the most frequent occupational respiratory disease,[11] and is clinically indistinguishable from 'ordinary', i.e. non-occupational asthma, at least if the physician fails to ask appropriate questions about the nature of the patient's work and working conditions.[12] The need for a proper occupational history was emphasized more than two centuries ago by Bernardino Ramazzini (1633–1714), an Italian physician, who is considered to be the founder of occupational medicine, since he was the first to describe systematically the association between occupation and disease, including respiratory disease.[13,14]

Another feature that emerges from Murlidhar *et al.*'s study[4] and the recent literature is that byssinosis,[15] at least in its epidemic form, has become a 'tropical' disease.

The situation of byssinosis is somewhat analogous to that of tuberculosis. By this, I do not mean that byssinosis and tuberculosis are no longer encountered

in temperate regions nor that their occurrence is determined by climate or geography, but that these diseases are 'caused' essentially by poverty and lack (or misuse) of resources. It is probably fair to generalize this concept and to state that there is a good epidemiological parallel between the distribution of the classical infectious diseases, such as tuberculosis, malaria or leprosy, and that of the classical occupational diseases, such as byssinosis, silicosis, asbestosis, and lead- or mercury-induced disease, even though accurate prevalence figures of occupational diseases are generally lacking in Third World countries. It seems, however, that public health specialists often ignore the burden of occupational disease on society.

The next fact that appears very clearly in Murlidhar *et al.*'s article[4] is the inherent conflictual context of the issue of occupational diseases.

Again, this is a constant throughout the history and practice of occupational health, particularly in the area of dust-induced lung disease.[16] Conflicts of interests are present, at least in a latent form, at various levels: the individual level, i.e. in the relation between the individual worker and his or her employer; the national level, i.e. in the tripartite relation between labour, capital and government; and the transnational level, i.e. in the relation between 'developed' and 'developing' countries. Thus, in the Bombay study,[4] only a small proportion of the target population (273 out of 1075 workers) could be persuaded to attend the camp for examination; the others were apparently afraid to do so. Of course, there are cultural factors (lack of education, resignation to fate, ignorance about long term health effects) which may explain why individuals appear to be unconcerned about occupational health risks. The main reason is that people often have to make a choice between an unhealthy job and no work at all. That they usually choose the former is understandable. One of the consequences of selective participation in occupational health studies is that the true extent of the ill effects may be considerably underestimated, thus contributing (together with other selection factors) to the famous 'healthy worker effect'.[17]

The study of Murlidhar *et al.*[4] also illustrates that the status of occupational health is determined by political choices and the balance of power at the national level. Here too, considerations about economic growth and competitiveness often take precedence over considerations about health. Thus, even when a legal framework and occupational hygiene standards exist, the legislation is not fully implemented or enforced. In this respect, the sectors that receive the least attention and, consequently, are at a particularly high risk of work-related injuries and disease, are those with the lowest degrees of organization of the workforce, such as farm labourers and workers in the 'cottage' industry.

However, with the increased globalization of the economy through the international trade of equipment and plants and the transboundary migration of hazardous products and waste, occupational health problems such as those related to the environment, must no longer be viewed solely in a national context.[18] Several recent disasters,[19] including the Bhopal catastrophe,[20] underscore their international dimension.

In conclusion, this article[4] illustrates that the medical profession has a role to play in the elimination of occupational diseases. We owe it to our patients that occupational factors should always be considered as possible causal or contributory factors to their illness. In some instances, the identification of such factors will permit a more appropriate treatment than symptomatic therapy, which we can often offer only to some patients; the discovery of an occupational cause should allow them to obtain compensation. Moreover, and perhaps more importantly, the medical profession owes it to society to recognize occupational causes of disease, in order to help prevent their occurrence. At least in theory, occupational diseases are among those which should be most amenable to prevention.

REFERENCES

1 Rom WN. *Environmental and occupational medicine.* Boston:Little Brown, 1992.
2 Rosenstock L, Cullen MR (eds). *Textbook of clinical, occupational and environmental medicine.* Philadelphia: W.B. Saunders, 1994.
3 Pickering CAC. Byssinosis. In: Parkes WR (ed). *Occupational lung disorders.* New Delhi:Oxford, Butterworth-Heinemann, 1994:729–54.
4 Murlidhar V, Murlidhar VJ, Kanhere V. Byssinosis in a Bombay textile mill. *Natl Med J India* 1995;**8:**204–7.
5 Cullen MR, Rosenstock L. The challenge of teaching occupational and environmental medicine in internal medicine residencies. *Arch Intern Med* 1988;**148:**2401–4.
6 Christiani DC, Eisen EA, Wegman DK, Ye TT, Lu PL, Gong ZC, Dai HL. Respiratory disease in cotton textile workers in the People's Republic of China. I: Respiratory symptoms. *Scand J Work Environ Health* 1986;**12:**40–5.
7 Takam J, Nemery B. Byssinosis in a textile factory in Cameroon: A preliminary study. *Br J Ind Med* 1988; **45:**803–9.
8 Tuypens E. Byssinosis among cotton workers in Belgium. *Br J Ind Med* 1961;**18:**117–19.
9 Demedts M, Gheysens B, Nagels J, Verbeken E, Lauweryns J, van den Eeckhout A, *et al.* Cobalt lung in diamond polishers. *Am Rev Respir Dis* 1984;**130:**130–5.
10 Nemery B, Nagels J, Verbeken E, Dinsdale D, Demedts M. Rapidly fatal progression of cobalt lung in a diamond polisher. *Am Rev Respir Dis* 1990;**141:**1373–8.
11 Beckett WS. The epidemiology of occupational asthma. *Eur Respir J* 1994;**7:**161–4.
12 Burge PS. Problems in the diagnosis of occupational asthma. *Br J Dis Chest* 1987;**81:**105–15.
13 Sakula A. Ramazzini's *de morbis artificum* and occupational lung disease. *Br J Dis Chest* 1983;**77:**349–61.
14 Bisetti AA. Bernardino Ramazzini and occupational lung medicine. *Ann NY Acad Sci* 1988;**534:**1029–37.
15 Parikh JR. Byssinosis in developing countries. *Br J Ind Med* 1992;**49:**217–19.
16 Markowitz G, Rosner D. The limits of thresholds: Silica and the politics of science, 1935 to 1990. *Am J Public Health* 1995;**85:**253–62.
17 Choi BCK. Definition, sources, magnitude, effect modifiers, and strategies of reduction of the healthy worker effect. *J Occup Med* 1992;**34:**979–88.
18 Sekimpi DK, Testa Tambellini A, Van Trung L. The global economy and occupational health. *Int J Occup Environ Health* 1995;**2:**76–9.
19 Ould Kadi F, Mohammed-Brahim B, Fyad A, Lellou S, Nemery B. Outbreak of pulmonary disease in textile dye sprayers in Algeria. *Lancet* 1994;**344:**962–3.
20 Dhara R, Dhara VR. Bhopal: A case study of international disaster. *Int J Occup Environ Med* 1995;**1:**58–69.

B. NEMERY
Department of Occupational Medicine and
Laboratory of Pneumology (Unit of Lung Toxicology)
K.U. Leuven
Belgium

Pre-eclampsia

Pre-eclampsia is a peculiar disorder characterized by the development of proteinuric hypertension during pregnancy. It is an important cause of maternal and perinatal death. Pre-eclampsia is unpredictable in onset and variable in its progression; the only known cure is termination of pregnancy.

The exact cause of pre-eclampsia is still a mystery but it appears to be associated with abnormal trophoblastic implantation. In normal pregnancies, trophoblast invasion into the uterus produces extensive changes in the spiral arteries that supply the intervillous space. The endothelium and the internal elastic lamina are replaced by trophoblast and amorphous matrix containing fibrin. These vascular changes extend to the inner third of the myometrium and increase the diameter of the spiral arteries up to four to six times of that in the non-pregnant state. These large channels do not respond to circulating vasopressors, thus ensuring adequate blood flow to the placenta. In contrast, such changes either do not occur in pre-eclamptic women or are limited to the decidual portion of the vessels, with the myometrial segments retaining smooth muscle and the blood vessels supplying the intervillous space remaining narrow.[1] The changes during normal and deficient implantation are complete by mid-pregnancy; yet the disorder does not manifest itself until late pregnancy.