the disease itself may change. They rightly suggest that a faulty mental "data base"—a term used by Weed[6] in his crusade for better case records—is a common cause for erroneous diagnosis. Further studies of this type would also provide evidence to grade the discriminating value of different symptoms—a useful addition for any textbook. The rating for night pain in detecting duodenal ulcer or postural heartburn for diagnosing hiatus hernia would be high. A low value might be given to nausea, aerophagy, or even loss of weight—which may not occur in cancer of the stomach but does happen in those with nervous dyspepsia.[1] The clinician's problem is how to keep his mental "data base" up to date and so be ready to alter cherished notions about the clinical pictures of common disorders.

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Humidifier fever: a disease to look out for

Man's ability to pollute his environment may take extremely subtle forms, and occasionally the first evidence of atmospheric contamination comes from patients with symptoms due to inhaling microscopic particles. In 1970 Banaszak *et al*[1] described four patients with cough and shortness of breath apparently related to working in an office. Investigation showed them to be suffering from a form of allergic alveolitis indistinguishable from farmer's lung.[2] The cause was eventually found to be the humidifier of an air-conditioner, which contained thermophilic actinomycetes whose spores were being liberated as an aerosol into the atmosphere of the office.

Subsequent reports[3–5] from the United States have defined humidifier fever in greater detail. The usual presenting symptoms are cough, dyspnoea, and fever related to the season in which the air-conditioner is in use. Often—and not surprisingly—the illness may be misdiagnosed for some years. In common with allergic alveolitis from other causes it may be relatively acute or slowly progressive and chronic. This latter type is potentially serious; it is particularly difficult to recognise, and there is a real danger of progression to irreversible pulmonary fibrosis.

Air-conditioning systems are not used so widely in Britain as in the United States, but they are becoming popular in hospitals, schools, and factories. In addition to circulating air at a controlled temperature they also provide the right humidification, and to do so they require a source of water. If this water is recirculated it is often collected in a reservoir, which may provide a suitable environment for the growth of a food-chain of micro-organisms. The variety depends on factors such as the substrate available and the temperature of the water. In the warmed water typically found in American air-conditioners, thermophilic actinomycetes seem to have been the main pathogen isolated, though a host of other organisms may usually be found wherever water is allowed to stagnate. When this water is sprayed the organisms will become airborne, and those of a certain size may be inhaled.

Humidifier fever as described in Britain[6 7] seems to differ slightly from that seen in North America. The American disease is altogether similar to farmer's lung, and the patients' blood contains the corresponding precipitating antibodies (but also rheumatoid factor, often in high titre—an unexplained finding). In the British outbreaks[8] patients affected were workers in a rayon factory and in two separate printing and stationery works. Their cough, dyspnoea, fever, and malaise were worse on Mondays, improving as the week progressed. In all three factories stagnant water was used in systems that produced an aerosol. Cellulose was abundantly available as a raw material and may have produced a suitable substrate for micro-organisms. The most striking difference between the British and American outbreaks was that thermophilic actinomycetes were not being incriminated here. While the patients' sera contained precipitating antibodies to extracts of water or sludge these could not be shown to correspond to any of the bacteria or fungi cultured from it. The mystery has probably been solved by Edwards,[9] who showed that the water from one outbreak was rich in protozoa and that the antibody response of the patients was to an amoeba, *Naegleria gruberi*, present in the water and in settled factory dust. Subsequent investigation of the other British outbreaks has confirmed that precipitin responses to various amoebae were also present in these patients.[8]

Once humidifier fever is diagnosed treatment depends on avoiding exposure to the organisms. Usually changes in the humidification system, such as steam injection or avoidance of recirculation, are sufficient. The possibility of producing such diseases should be considered in the design of humidifying systems. The physician needs to add a new question to the history taken from patients with unexplained fever, cough, or breathlessness: "Do you have an air-conditioner or humidifier at home or work?"

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Risks of environmental exposure to asbestos

Asbestos-related cancers are well established as an occupational hazard, but what risk is there from pollution of the general environment by asbestos fibres? As these may be both inhaled and ingested, clearly there are several possible sources of pollution.

Environmental pollution by asbestos fibres has been recognised for over 15 years. Wagner and his colleagues,[1] who originally linked exposure to crocidolite asbestos with mesothelial tumours, found several patients who had been exposed to it only by living near the mines or playing on the mine dumps as children. In a series[2] of 76 patients who died with a mesothelial tumour at the London Hospital 11 had had no occupational or domestic exposure but had lived within half a mile of a large asbestos factory. A high incidence of pleural

plaques has been found in people who have lived near asbestos mines; and many postmortem investigations have detected asbestos bodies, and more recently asbestos fibres, in the lungs of people who have had no obvious exposure to asbestos. Asbestos body counts in the lung are higher in men than women and in urban than rural areas; in London[3] these were higher in people living in the industrial and dockyard areas near the Thames than in more peripheral areas—but no higher in those with cancer of the lung than in those dying of other diseases.

More recently workers have studied the possible effects of ingesting asbestos. In occupational groups, particularly where exposure may have been heavy and amphibole asbestos has been used, peritoneal mesotheliomas may be as common as pleural tumours, and deaths from gastrointestinal tumours may be two or three times more frequent than would be expected in the general population. Asbestos filters are commonly used to clarify beer and other alcoholic and non-alcoholic beverages and asbestos fibres have been identified in these.[4] Town water supplies may also be contaminated—there may be asbestos deposits in the supply area, for example, or fibres may be leached from asbestos cement pipes. Fibres have even been found in fluids used for parenteral injections. Asbestos fibres instilled into the stomach of rats will penetrate the intestinal mucosa and appear in the blood stream, and when injected into the femoral vein of pregnant rats can be recovered from the fetuses removed before parturition.[5]

In 1973 the United States Environmental Projection Agency reported the discovery of large amounts of asbestos in Lake Superior, the source of the municipal water supply for the city of Duluth in Minnesota. Electron microscope studies showed 1-30 million amphibole asbestiform fibres per litre of Duluth tap water—far more than in water from the Canadian asbestos mining areas. The source was the taconite tailing waste from an iron ore processing plant, which began to be dumped in the lake in 1955. Epidemiological investigations followed in both the United States and Canada.

Levy and his colleagues[6] investigated the incidence of gastrointestinal cancer in Duluth from 1969 to 1972, obtaining information from the city's hospitals and comparing the incidence in Duluth with that in Minneapolis and St Paul, two cities with similar demography. There was no detectable excess of cancer in Duluth, but contamination of the water supply had been present for only 14-17 years. Wigle[7] compared the mortality in 22 towns in Quebec with known high, possibly high, and low exposure to asbestos. Although he found an excess mortality rate from cancer of the lung and stomach in men and of the pancreas in women in the two towns with high exposure, the excess in men may have been due to occupational exposure, as these were asbestos mining towns, and the higher cancer mortality could not therefore be definitely attributed to asbestos in drinking water. Harrington *et al*[8] studied the incidence of gastrointestinal cancer in Connecticut using the records of the cancer registry. Calculating exposure and risk factors based on the age of the cement pipes, the ability of the water to leach asbestos fibre, and the length of pipe used by the population, they found no association between the various risk factors and the incidence of gastrointestinal tumours.

Recently the Health and Safety Directorate of the European Communities has reviewed the risks of environmental pollution by asbestos.[9] Its report argues that true ambient exposure through air, water, beverages, food, or drugs carries no definite risk, but that too many uncertainties exist to deny such a risk; nevertheless, any substantial hazard, it concludes, is likely to have been detected by now.

In view of the increasing evidence of a dose-response relationship between asbestos exposure and mortality from asbestos-related tumours this report may be a little too tentative in its conclusions. People developing cancer now would have been exposed to the conditions of 20-50 years ago, and special areas such as Duluth must obviously remain under surveillance. Nevertheless, with increasing knowledge of the hazard and increasingly strict control of industrial use and disposal of asbestos the community at large seems most unlikely to be exposed to a perceptible risk.

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Non-paralytic motor dysfunction after strokes

The treatment of hemiplegia is based on two criteria, one diagnostic, the other an estimate of capacity.[1] The prognosis clearly depends on the nature of the lesion, and care must be taken to exclude other possibilities before the cause is assumed to be a cerebral thrombosis. Paralysis alone seldom accounts for a patient's incapacity and may contribute little to it. The overriding defect may be a change in intellect or some potentially reversible neurophysiological disturbance.[2] These mental barriers to recovery[3] have a disparate effect on the two hemispheres: patients with lesions of the left hemisphere make better recoveries than those with right-sided lesions,[2] and each hemisphere provides a different challenge.

The non-dominant hemisphere is concerned with man's perception of his environment.[4] Parietal dysfunction causes neglect of the affected limb, denial of the motor deficit, and bizarre conceptual defects. These defects may be overlooked and—tragically—active treatment stopped because the patient is thought to be confused, uncooperative, or lacking motivation.[3] By contrast, left hemisphere damage may be obvious and be interpreted, incorrectly, as giving a poor prognosis. Disturbances of language are readily apparent. Far from denial of illness, the patient may be depressed, and absence of movement may be added to the paralysis. There is a danger that comprehension defects, depression, and clumsiness may be labelled as dementia.[3]

The perceptual impairment seen with non-dominant hemisphere lesions affects the restoration of motor skills. Similar disorders are common in children with cerebral palsy.[5] Denial of disease (anosognosia) may lead to neglect, lack of recognition, and even delusions as to the nature of the affected limb.[6] Patients underestimate, deny, or are unaware of the extent of their disabilities.[7 8] They tend to set themselves unrealistic goals and fail to appreciate their mistakes[9]; often they are unable to profit from experience and so are unlikely to improve.[10 11] When depression occurs, it arises as an indirect