history in English translation while commuting by car from the suburbs. Starting in Egypt with the second book of Herodotus, we covered the Persian and Peloponesian wars, the decline of the City States, the Anabasis of the Ten Thousand and that of Alexander, the quarrels of his successors, and then, skipping from one author to another to maintain the chronology, right through the history of the Roman Republic and Empire.

Somewhere around 350 AD my wife become sick of the whole thing, leaving me to wander on my own through the darkness of the Middle Ages, but not before we reached certain conclusions about which authors we would like to invite for dinner. Herodotus, we thought, would be most entertaining with his stories, some true, some false, for it was not for nothing that he was called the father of lies as well as the father of history. Polybius would probably monopolise the entire conversation, talking in his pedantic manner about the shortcomings of earlier historians—in particular, about the errors of Phylarchus, who, "for the sake of making his narrative sensational composed a tissue of ... improbable falsehoods" as well as betraying his gross ignorance. Xenophon, we agreed, would be a most pleasing guest, jovial and entertaining, talking about hunting and fishing, not too intellectual, never playing "the old culture game." Indeed, we wondered why his *March of the Ten Thousand* was never made into a movie, with some glamorous film star taking the role (albeit expanded) of Queen Synnesis, and with some Kurdish or Armenian princesses added for good measure to increase box office appeal. But Thucydites, perhaps the greatest of historians, might be too serious as a guest; the atmosphere might be too constrained; and he might even refuse the wine and abstain from dessert as he would lapse into reminiscences about the fate of the Athenians in Sicily, who "having done what men could, suffered what men must."

Pollution and People

How much more can we do about air pollution?

DAPHNE GLOAG

With the reduction of smoke and to a lesser extent sulphur dioxide other components of air pollution have gained more prominence. The most important of these pollutants are carbon monoxide,[1] nitrogen dioxide,[2] ozone and other photochemical oxidants,[3] and polycyclic aromatic hydrocarbons.

Carbon monoxide

Haldane experimented on himself with carbon monoxide, reaching a carboxyhaemoglobin concentration as high as 49%—by which time his vision was dim and he had difficulty in moving.[4] Carbon monoxide owes its toxicity to the fact that it has about 240 times greater an affinity for haemoglobin than has oxygen and also impairs the dissociation of oxyhaemoglobin. The two current problems are the toxic concentrations of the gas that still build up occasionally indoors and the question of whether ill effects occur at low concentrations.

People assumed that the lethal effects of carbon monoxide would seldom be seen once natural gas replaced town gas. But even though natural gas contains only 1% of carbon monoxide, inadequate ventilation will lead to its production. Normally natural gas undergoes combustion with the formation of carbon dioxide and water vapour; but if too little oxygen is available combustion is incomplete and carbon monoxide is formed. Appreciable concentrations have been found in kitchens with normal gas cookers but inadequate combustion.[5] The old coal fires had extremely inefficient combustion but there was ample ventilation—and if a flue became blocked smoke acted as a warning. Smokeless fuels can give rise to fatal accidents if a flue becomes obstructed. Gas or oil appliances (often unflued) in confined spaces such as caravans and boats are an especial hazard. Paraffin stoves, moreover, are increasingly used in almost sealed rooms, causing a sudden increase in carbon monoxide concentrations, often while the occupants are asleep. Slow-moving traffic in a tunnel or at the entrance to an indoor car park is also potentially hazardous and the build-up of carbon monoxide has produced early symptoms of poisoning. In such circumstances engines should be switched off whenever possible and workmen should be exposed for only short periods.

In 1979 there appeared to be an increase in fatal cases of carbon monoxide poisoning, though a change in classification makes it difficult to be sure about this (DHSS, unpublished information). In any case the 158 largely avoidable deaths are too many. We need far more publicity about the importance of ventilation and of checking appliances, flues, etc. Equally doctors, especially casualty officers and general practitioners, need to be alert to the possibility of early carbon monoxide poisoning—symptoms such as nausea, headache, and dizziness have been disastrously ascribed to a "flu-like" illness or gastroenteritis. Three deaths in a caravan, for example, followed a visit from a GP who diagnosed gastroenteritis; and previous occupants had also suffered from early symptoms of poisoning.[6]

At lower carbon monoxide concentrations, the point at which the reduction in oxygen transport becomes important clearly differs for different people and circumstances. The breakdown of blood pigments produces enough carbon monoxide to give a carboxyhaemoglobin concentration of 0·1-1·0%; there is then equilibrium between blood and air if the carbon monoxide concentration in the air is about 5 mg/m^3.[1] Smoking usually provides the most important source of carbon monoxide, commonly giving the smoker a carboxyhaemoglobin concentration of 5-15% or even more, whereas non-smokers even when exposed to street air rarely have a carboxyhaemoglobin saturation over 3%.[7] The carboxyhaemoglobin concentrations at which various impairments—notably of the central nervous system and the cardiovascular system—have been found in controlled studies are summarised in the WHO report (table 10).[1]

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DAPHNE GLOAG, MA, staff editor

More about air pollution

Carbon monoxide is produced mainly by incomplete combustion of carbon-containing fuels. Out of doors the most important source is the exhaust of petrol-fuelled vehicles, indoor sources are cooking and heating appliances, particularly if they are faulty or ventilation is inadequate, and tobacco smoke. In busy traffic maximum eight-hour concentrations may be 20-60 mg/m³ (1 mg/m³=0·9 ppm)[1] and peaks of over 340 mg/m³ have been recorded.[7] The relation between ambient carbon monoxide and carboxyhaemoglobin concentration depends on several factors, including length of exposure; in a non-smoker it might be 2·5% after one hour at 70 mg/m³.

Of the oxides of nitrogen in the general atmosphere, only *nitrogen dioxide* is known to have biological effects. Worldwide, by far the largest quantities are produced by the action of bacteria, volcanoes, and lightning; but man is exposed to much greater amounts from burning fossil fuels. Urban values vary widely, with highest hourly values in the range 240-850 μg/m³ (100 μg/m³=0·05 ppm). Indoor concentrations may be much higher, especially near unflued gas appliances.

Ozone and other *photochemical oxidants* result from the interaction between reactive hydrocarbons and nitrogen oxides in strong sunlight. In some large cities the maximum one-hour oxidant (mainly ozone) concentrations often exceed 200 μg/m³ (0·1 ppm)—in Los Angeles they do so on most days from May to October.[3]

Studies on vigilance, discrimination, and the ability to perform fine tasks, clearly relevant to the safety of driving in heavy traffic, have given conflicting results.[7 8] The lowest level at which an effect has been reported is 2-3% carboxyhaemoglobin saturation: impairment of time discrimination at this level appeared in one study[9]; and though this result was not later confirmed it has been influential and has been incorporated in some legislation. Impaired vigilance or reaction time has been found at concentrations of about 3-7%.[1] But some studies have failed to show any effect at these and higher levels. For instance, in double-blind experiments 573 mg/m³ of carboxyhaemoglobin for one hour, giving a mean carboxyhaemoglobin saturation of 8%, had no effect on perceptual discriminations—unlike 100 mg of phenobarbitone—nor did a 5% saturation affect performance in a battery of tests.[7 8] The balance of the evidence suggests that effects are unlikely below 3% saturation. One of the chief requirements is that experiments should be carried out double blind; but this precaution has often been omitted.[7] One difficulty in such work is that the results may be affected by many other factors, including alcohol and other drugs, food, and fatigue. This also means that carboxyhaemoglobin in the environment should be low enough to allow for interacting factors: it may in some cases act synergistically.[8] On the other hand, setting a very low environmental standard, on the grounds that driving and other such activities must not be impaired, would be bizarre when smokers (though they are probably rather less sensitive[1]) are free to do these things with much higher carboxyhaemoglobin concentrations—to say nothing of alcohol concentrations.

More realistically, perhaps, people with cardiovascular or pulmonary disease may be threatened by any extra hypoxia—in other words, by quite low concentrations of carbon monoxide. Some evidence supports this.[3] For instance, breathing 57 mg/m³ carbon monoxide for two hours was detrimental to patients with angina; their carboxyhaemoglobin saturation was 2·5-3·0%.[10] Presumably iller people would be even more vulnerable. Moreover, if a series of animal studies (mainly in rabbits) is relevant to man, prolonged moderate carbon monoxide exposure could encourage atherosclerosis, at least when there is a high cholesterol diet[11]; but the value of these studies is limited by their not having been carried out blind.

Should we then aim at a carbon monoxide standard that will protect even the most fragile? Or should we merely urge that these people keep away from the most polluted places? The WHO report settles for a compromise; it tentatively recommends that levels should be such that carboxyhaemoglobin concentrations do not exceed 2·5-3·0% in non-smokers (agreement on a single value could not be reached), and that both environmental and biological monitoring should be carried out.[1] An earlier WHO report had specified 4%.[12]

Nitrogen dioxide

Nitrogen dioxide is an ingredient in photochemical pollution and also a pollutant in its own right. Urban levels vary widely (see box) and indoor exposure is more important for some people using gas in their homes—concentrations of up to 2000 μg/m³ may occur at breathing level directly above gas cookers,[2] and even 3000 μg/m³ has been recorded.[13] This is far below the estimated occupational exposures reported to have caused bronchitis and pneumonia (estimated at 47 mg/m³ upwards). Inhaled tobacco smoke, however, may contain as much as 226 mg/m³.[2]

Experiments on volunteers exposed to nitrogen dioxide have shown impaired ventilatory function[2]—in some asthmatic patients a slight but significant worsening appeared with one hour's exposure to only 190 μg/m³,[14] but this result needs confirmation. In healthy volunteers respiratory effects have been seen after 10 minutes at 1300-3800 μg/m³. Epidemiological studies cannot usually separate any effects of nitrogen dioxide from those of other pollutants but some have suggested an increase in respiratory illness and reduced lung function, especially in schoolchildren exposed to appreciable levels of nitrogen dioxide combined with other pollutants.[2] Animals have been made more susceptible to infection by being continuously exposed to nitrogen dioxide at levels found intermittently indoors. For example, mice exposed to 940 μg/m³ had significantly more infections from 90 days; later they were less able to clear inhaled organisms from their lungs.[15]

Studies on people using gas in their homes have given varying results. The US Environmental Protection Agency found no more acute respiratory illness in housewives with gas cookers (half- to one-hour peak nitrogen dioxide concentrations ⩾ 940 μg/m³) than in those using electricity[2]; and several studies on the children of families using gas for cooking have shown no evidence of increased infections—for example, an American study of 7000 adults and children aged 7 and over found no more respiratory symptoms or worse lung function in those with gas cookers in their homes, even though nitrogen dioxide concentrations were appreciable.[13] But a large retrospective study found a small but significant excess of respiratory illness under the age of 2 years.[16] Among families in Middlesbrough, after earlier suggestive findings, only a weak association was found between respiratory illness in children and gas cooking. There was some suggestion that illness was related to the (quite low) bedroom but not kitchen levels of nitrogen dioxide; but the nitrogen dioxide concentration could conceivably have been a marker for some other factor such as humidity or temperature.[17]

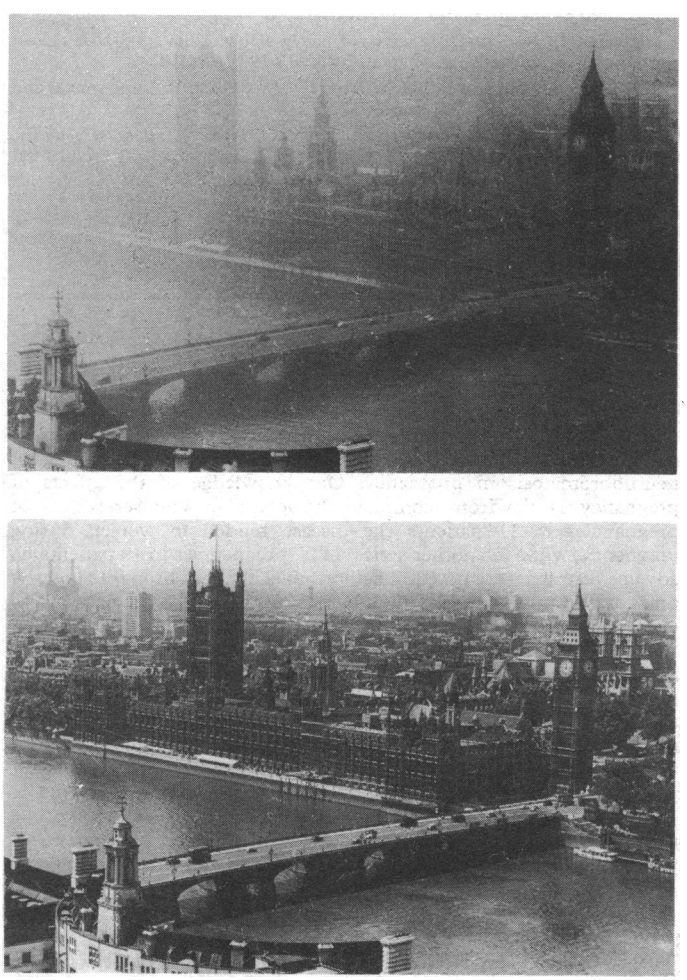
The latest WHO report,[2] unlike an earlier one,[12] proposes guidelines on the strength of experimental data from animals and man—the task group thought it prudent not to wait for more conclusive epidemiological evidence. It selected 940 μg/m³ as the estimated short-term threshold for effects; to allow for a safety factor (especially as those with asthma may be sensitive well below that level) it proposed 140-320 μg/m³ for the maximum one-hour exposure level (not to be exceeded more than once a month). But the report points out that a larger safety

factor could be needed since nitrogen dioxide may interact with other air pollutants. City concentrations are likely to rise, it adds, if effective measures are not taken. British authorities are considering whether the values should be lower. Even so, a more pressing problem may be to reduce indoor concentrations in some homes.

Photochemical pollutants and hydrocarbons

Cities combining plentiful sunshine with heavy traffic may have pollution from ozone and other photochemical oxidants[3] (box). This happens only occasionally in Britain. The worst occasion was in the summer of 1976 in south-east England, when ozone concentrations rose above 500 μg/m³. The death rate increased sharply, but this appeared to be due rather to the high temperatures.[18]

In volunteers and in community studies, adverse effects, including impaired respiratory function, have been found at oxidant concentrations of from 200 to 500 μg/m³.[3] Coughs, chest and eye discomfort, and headaches (unaccompanied by fever, etc) were significantly more common when the maximum hourly concentrations in Los Angeles reached 200-580 μg/m³,[19] and there is a possible association between asthma attacks and high oxidant levels, though seasonal variations in the former were not taken into account in the study concerned.[3] An adverse effect on runners' performance has been reported, with an estimated threshold of 240 μg/m³ oxidant concentration.[3] Photochemical pollution has several components but ozone concentration seems to show the strongest correlations with ill effects.



Haze in London: though elimination of coal smoke has greatly improved visibility aerosols of other pollutants lead to hazy conditions at times.

Animal studies support an association with respiratory effects[3]; and mice have been made more susceptible to bacterial infection by being exposed to ozone concentrations as low as 160 μg/m³.[20] Subcellular effects, chromosome aberrations, and suppression of interferon production have also been produced experimentally, but mainly by high ozone concentrations. The WHO report recommends more experimental work on the possible roles of ozone in mutagenicity, carcinogenicity, and depression of immunity. It proposes 100-200 μg/m³ for hourly ozone concentrations as a public health guideline not to be exceeded more than once a month.

As carcinogenic polycyclic aromatic hydrocarbons are components of smoke, epidemiological studies have looked at lung cancer, which has shown higher rates in urban than rural areas, in relation to air pollution. Studies on immigrants—for example, from Britain to less polluted countries—have tended to suggest an environmental factor beyond the possible differences in smoking habits[21]; but immigrant populations are likely to be unrepresentative in some respects. Mortality from lung cancer has also been studied in relation to the falling smoke concentrations in Britain.[22] Benz(*a*)pyrene concentrations were some 10 times higher in London around 1950 than in the early 1970s. Differences between the death rates from lung cancer in London and those in rural districts diminished over 20 years. At present, however, the effects of reduced air pollution cannot easily be separated from those of changes in smoking habits, with the possibility also of "selective migration" effects between town and country. In any case smoking would be a much more important cause of lung cancer. A recent American study, starting from the premise that air pollution could be a weak carcinogen in combination with other factors, failed to find any evidence of this—with allowance made for smoking habits, non-occupationally exposed men showed no difference in lung cancer rates between urban and rural areas with high, medium, and low concentrations of benzene-soluble organic matter or particulate air pollution.[23]

Indoor pollution

Outdoor pollutants can all penetrate indoors; but they tend to be less important since most are absorbed to some extent by furnishings. Nitrogen dioxide, as we have seen, may be more important indoors. The most obvious indoor pollutant is tobacco smoke, and recent evidence suggests that it may cause lung cancer in non-smokers constantly exposed to it.[24,25] A smoky atmosphere containing 44 mg/m³ carbon monoxide raises non-smokers' mean carboxyhaemoglobin concentration from 1·6% to 2·6% (compared with 5·9% to 9·6% in the smokers).[7]

Other pollutants released inside buildings include radon, formaldehyde, and asbestos (21 February, p 623). Radon, emitted by certain building materials and soils, can easily produce "non-negligible" irradiation of the lungs, concludes a WHO report; while formaldehyde, which may irritate eyes and respiratory tract, is released by ubiquitous materials such as particle boards; glued wooden products; paints, carpets, etc; textiles; and insulating foam.[26]

The simplest and most effective way of restricting indoor pollution is to increase ventilation. Here, however, engineers tend to work in opposition to health authorities, as the WHO report points out,[26] now that energy conservation dictates lower ventilation rates. This makes it all the more important to keep indoor pollutants, especially tobacco smoke, to a minimum—and to obtain more information about this somewhat neglected subject. After all, most urban adults spend most of their time indoors, at least in the colder climates; and the most vulnerable people—the old and frail and small babies—are likely to leave their houses least. Moreover, indoor exposure needs to be taken into account when outdoor standards are being considered.

* * *

"There are quality levels with different price tags," it has been said of air pollution: "It is the prerogative of the people to decide what quality they will pay for. It is up to the scientific community to define what the people can get for their money."[9] But once levels are fairly low such definition is difficult—certainly the WHO guidelines are often speculative. Moreover, further reduction not only becomes expensive but gives slight additional health benefits, as a WHO report (fig 2) illustrates.[12] Smoking, moreover, is undoubtedly a more important influence than air pollution. Resources being finite, should we aim at protecting everyone all the time? We still do not know enough about the extent to which present levels of air pollution do contribute to illness. Even so, with the growth of traffic and industry, such measures as reducing emission of pollutants and improving traffic management are needed anyhow: they still need to be controlled by the "best practicable means." But indoor pollution could be more important for many people; while the interaction of pollutants presents one of the main problems for the future.

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Where could I obtain a half or a whole skeleton, and what is the approximate cost?

Skeletal material may be bought from either Adam Rouilly (London) Ltd, Crown Quay Lane, Sittingbourne, Kent, or from Gerard Biological Centre, Worthing Road, East Preston, West Sussex BN16 1AS. Most of this material comes from the Indian subcontinent, and for various periods during the 1970s the Indian Government restricted its export: the resulting sharp rise in prices is still in evidence, despite the recent lifting of this ban. A whole articulated skeleton in good condition costs between £240 and £255, and a "half" disarticulated skeleton (including skull) from £60 to £95. A first-class skull for dental studies cannot be obtained for under £65. Plastic replica material is an acceptable substitute, although it precludes first-hand experience of variability. A whole, articulated plastic model (full size) is relatively good value at about £133; the half skeleton rather less so at about £70. The "real thing" is to be recommended whenever possible—purchasers of real bones may be asked to verify professional status.

A woman of 26 with severe diffuse neurofibromatosis has had numerous operations for removal of these tumours. Is it safe for her to start oral contraception?

There is no definite information on the effect of oral contraceptives on neurofibromatosis.[1] There has been a reluctance to prescribe the pill to such patients for fear that it may mimic the effects of pregnancy: a few isolated case reports have described worsening of neurofibromatosis in pregnancy. Our knowledge of the effects of pregnancy is far from complete, however: in one series[2] of 24 pregnancies in 11 patients the disease tended to worsen during pregnancy, while in another series[3] of 27 pregnancies in 10 patients no adverse features were noted. The hormonal changes brought about by oral contraceptives are far from identical to those of pregnancy and so one cannot extrapolate with any certainty from one to the other. Because of the doubt, most doctors would probably prefer to play safe and avoid prescribing the pill if an alternative method of contraception is suitable. Nevertheless, if the pill is the only suitable method it is not contraindicated, provided that the patient understands the uncertainty over its effect on her condition. Exacerbations of neurofibromatosis during pregnancy are said to improve once pregnancy is over,[2] and the pill may be immediately discontinued if the condition seems to deteriorate. Naturally the patient should be supervised closely, and the blood pressure should be checked carefully in view of reports[2] that pregnancy hypertension is particularly common in women with neurofibromatosis.

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