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THE PREVENTION OF THE DUST DISEASES*

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Sir Malcolm Morris, who was on the staff of St. Mary's Hospital for over 20 years, was a pioneer in public health as well as being a great dermatologist. In the galaxy of great names for which the hospital is notable, he holds a high place and his contributions both to dermatology and public health are of enduring value. It is common knowledge that dermatitis causes more cases of ill health and lost time in industry than any of the other diseases of occupation. But it can be said that the diseases of the lungs caused by dust are more dangerous than the industrial dermatoses. The pneumoconioses often cause death, whereas skin lesions, though they are disabling, are rarely fatal. Again, the lung diseases caused by dust are harder to diagnose, and in the early stages at least they give no outward sign of the pathological process going on inside the chest. A disease of the skin has this advantage, that it can be seen, even by the novice, as soon as it appears. But the pneumoconioses come on like a thief in the night, or rather, like a thief who plies his trade for days, nights, and years before he is finally caught. And by that time the damage is done. It is like closing the stable door after the horse is stolen. In trying to prevent the dust diseases we aim to close the door while the horse is still in the stable; or while the man is still in good health. And in this discourse, I shall describe some of the ways in which we are trying to close the door.

Historical

Before dealing with the prevention of the pneumoconioses, I ought to say something about them. For, in order to prevent a disease, it is obvious that one must first know that it exists, and the causes of it.

To us in the 20th century it may seem strange that at any time it was not known that dust, when inhaled into the lungs, could damage them. But it took a long time before this fact was established, and though the history of the pneumoconioses goes back as far as Hippocrates (about 400 B.C.) and perhaps earlier, they were not charted with accuracy until about 30 years ago. Even now there is a great deal to be learned about them.

Progress in knowledge is usually associated with great names; but one should not forget those workers who made no great splash in history. The building-up of knowledge has been likened to a coral island, into the formation of which go the lives of many individual organisms. So it is with our knowledge of the occupational diseases. One of the great names in occupational medicine, after Hippocrates, was Paracelsus, also called Bombast. He, in 1530, published a book on industrial diseases in which he described the chronic lung diseases of miners as pulmonary consumption and asthma. He thought that these diseases were due mainly to the influence of the astral bodies, but he added that the "climate" of the mines might have something to do with them. I daresay that in writing about the climate he included dust as one of the possible causes, but he did not say so. The next great name was Georgius Agricola who, in a book called *De Re Metallica* (1556), said that the inhalation of "corrosive" dusts predisposed a worker to a disease characterised by exhaustion, coughing, and "that difficulty of breathing which the Greeks called asthma."

In 1912 an excellent translation of this book was made by Herbert Hoover, a mining engineer, and his wife. This was the same Herbert Hoover, who later became President of the United States.

But the outstanding name among the early doctors was Bernardino Ramazzini, an Italian who in 1700 published the first textbook (*De Morbis Artificum*) on occupational diseases, based on first-hand observations of varied occupations and the maladies which arise from them. Ramazzini is justly called the father of occupational medicine; and it was he who taught that when seeing a patient for the first time one should not only ask him where his pain is, but what his job is.† Even today there are doctors who do not get details of their patients' occupations. Ramazzini quotes the account of the necropsies carried out in 1649 by Diemerbroeck on stonecutters, "in whose lungs he found such heaps of sand, that in running the knife through the pulmonary vesicles he thought he was cutting some sandy body." This is the first account of the pathology of silicosis, as it is now called. In spite of this, the earlier doctors confused the dust fibroses of the lungs with tuberculosis. But we have to bear in mind that the tubercle bacillus was not discovered until 1882, and that the study of pathology was not put on a firm basis until the early part of the 19th century. As a point of historical interest, it might be added that Sir Malcolm Morris was present at the first demonstration of the tubercle bacillus in Koch's laboratory in Berlin in 1882 (*Lancet* 1924).

The 19th century was notable for the rise of the idea that inhalation of large quantities of dust of any kind can damage the lungs but that some dusts are more harmful than others. It was our first great English writer on the occupational diseases who emphasised this. He was Charles Turner Thackrah, a doctor in Leeds who in 1831 published his book on the *Effects of Arts, Trades and Professions*. The title goes on—"and of civic states and habits of living on health and longevity, with suggestions for the removal of many of the agents which produce disease, and shorten the duration of life." He noted that bricklayers and limeworkers were long-lived and that sandstone masons usually died before they reached the age of 40. But he was not quite clear in his mind how

* Based on the Malcolm Morris memorial lecture given under the auspices of the Chadwick Trust at St. Mary's Hospital, London, on Dec. 9, 1952.

† Ramazzini's motto was "Medici munus plebeios curantis est interrogare quas artes exerceant."

TABLE I—DUST DISEASES OF THE LUNGS

Diseases	Dust or fume	Varieties
1. Chronic fibroses	Silica	Silicosis
	Asbestos	Asbestosis
	Coal	Coal pneumoconiosis
	Talc	Talc pneumoconiosis
	Bauxite and corundum	Shaver's disease
	Beryllium	Beryllium granulomatosis
2. Acute pneumonitis, œdema and/or bronchiolitis	Manganese, Beryllium, Cadmium, Vanadium and their oxides	Manganese pneumonia; Beryllium pneumonitis &c.
	Bagasse (sugar-cane)	Bagassosis
3. Asthma	Wood (e.g., Western red cedar)	
	Seeds	
	Grain	
	Feathers	
	Wool	
	Double salts of platinum	
	Gum acacia	
4. Cancer	Arsenic	
	Chromates	
	Nickel carbonyl (?)	
	Asbestos	
	Radioactive emanations	
5. Chronic bronchitis and emphysema	Cotton	Byssinosis
	Flax and other dusts	

much the taking of alcohol had to do with these pulmonary diseases. He was against alcohol (in the same way that President Coolidge was against sin) and no doubt he had good reason to be, because it was quite cheap in those days.

Two other great English names in the history of the pneumoconioses are T. B. Peacock and E. H. Greenhow. Peacock, a physician on the staff of St. Thomas's Hospital, first established between 1860 and 1866 the existence of miners' disease as an entity and distinguished it clinically from pulmonary tuberculosis. Greenhow (1860, 1861), who was attached to the Middlesex Hospital, carried out the first large field investigation into the dusty industries such as the potteries, metal trades, cutlery making, tin and copper mining, coal-mining, lead mining, cotton, flax, silk and woollen manufacture, hosiery and lace making, glove-making, and agriculture. In the *Transactions* of the Pathological Society of London (1860–66) are to be found excellent clinical and pathological descriptions by both these doctors of the disease which was later to be called silicosis by Visconti in 1870. They even found the dust of free silica in the lungs and examined it under polarised light.

For about 40 years after this excellent work nothing much was done about the dust diseases. But with the turn of the century a new interest began to be taken in

Fig. 1—Dust of quartz or free silica showing crystalline structure (× 650).

the problem, not only in this country, but also in other parts of the world. It is significant that about the same time there was a quickening of the tempo of life in general. The horse began to give way to the motor-car, and soon there came the first aeroplane. Then too there was the telegraph and the telephone and all those "comforts" of present-day life which give us little or no time to think. In the factories and in the mines, there was a speeding-up of output. Hand labour began to be replaced by the machine. In the cotton industry the change had come a little earlier. But the air-hammer or the pneumatic tool began to be used more extensively in the early part of the century for such jobs as mining, quarrying, and the cleaning of castings in foundries. There is also the spray gun, which is used to spray paint, glazes, metals, and asbestos, and was introduced with the idea of speeding up the work. This, from a health point of view, is a dangerous instrument, because it is very difficult to control the spray and to prevent its inhalation by the workers.

If there is one thing certain about increased speed of production in industry, it is that a dusty process will become more dusty; and that there will be a greater incidence of the dust diseases, if attention is not given at the same time to increased control of dust. But quite often the efforts at dust control are inadequate and do not keep pace with the increased speed of production.

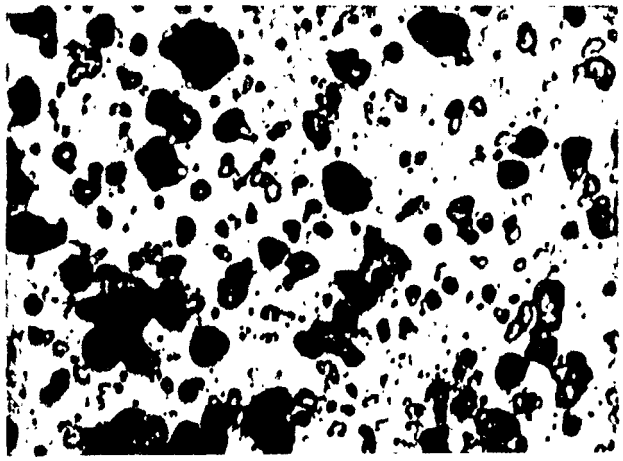

Fig. 2—Quartz crystals and aggregates of iron oxide fume (large black areas) in a foundry dust cloud (× 1000).

For instance, silicosis and silico-tuberculosis did not become a problem in hæmatite mining until the introduction of the pneumatic drill. Stewart and Faulds (1934) say that hæmatite mining has been carried on in Cumberland since the time of the Roman occupation. "It was formerly considered a healthy trade, but evidence is accumulating to show that in this respect a definite change for the worse has taken place. The miners themselves believe that the trouble started with the introduction of the dry mechanical drill in 1913." Previously the ore had been obtained by the old "hammer and jumper" method.

Meiklejohn (1951, 1952a and b), in his history of lung diseases of coalminers in Great Britain, points out that mechanical cutting and conveying of coal was introduced early in the present century. The change from hand-cutting of coal led to an increased dustiness of the air in the mines, not only at the coal face but generally throughout the workings. He quotes H.M. Chief Inspector of Mines (Bryan 1950) as saying: "There is no doubt that one result of the adoption of many of the present methods of machine mining is that the production of dust in mines has increased in recent years and is still increasing. If we are to get rid of the scourge of pneumoconiosis, this process must be reversed." McCallum (1952) also thinks that mechanised coal-getting has increased the prevalence of pneumoconiosis in the Durham coalfield and "that future developments in the coalfield will intensify dust production and the risks of pneumoconiosis unless dust suppression methods are considerably extended."

Fig. 3—Dust of diatomaceous earth or kieselguhr; a non-crystalline form of free silica (× 300).

...e use of the pneumatic tool instead of hand methods ... the fettling of steel castings has also increased the ...sk of silicosis in this occupation (McLaughlin et al. 1950).

Dust and Dust Diseases

It is commonly thought that the dust diseases are confined to a small group of fibroses of the lung caused by the inhalation of inorganic dusts such as silica, asbestos, and coal. That this is not so was clearly shown by Collis in his Milroy lectures in 1915. He pointed out that an excessive mortality from all respiratory diseases was experienced by dwellers in dusty atmospheres, an excess which increases with age and with the amount of dust present. In London at that time 650 tons of dust a year came down over a square mile, whereas in an agricultural district it was 195 tons, a figure which is big enough in all conscience. It is interesting to note also, that the inorganic content of the lungs, which largely comes from inhaled dust, also increases with age. Radiologists sometimes have difficulty in distinguishing in chest films the shadows thrown by dust deposits from the abnormal shadows seen in films of old people. It does not often occur to them that the changes associated with age might in fact be partly or even mainly caused by dust deposits in the lungs.

The types of pulmonary disease caused by dust are shown in table I.

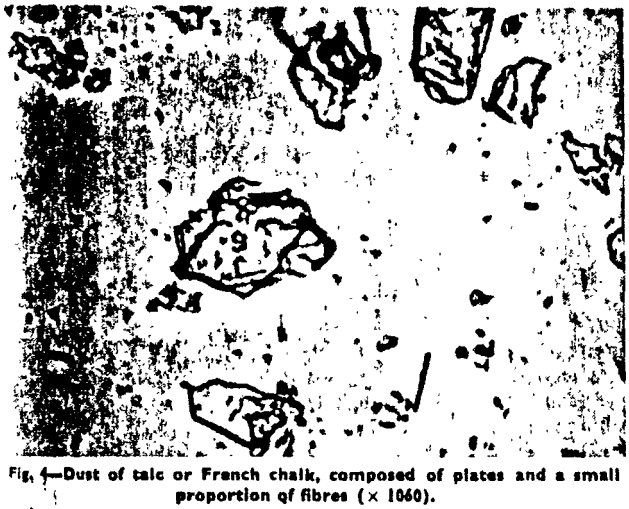

Fig. 4—Dust of talc or French chalk, composed of plates and a small proportion of fibres (× 1060).

It will be seen that these diseases fall into five broad groups: chronic fibrosis; acute pneumonia, œdema, and bronchiolitis; asthma; cancer; and chronic bronchitis and emphysema. There is also a sixth group which hardly comes under the heading of disease—namely, the abnormal X-ray appearances seen in those workers who have been inhaling the radio-opaque dusts such as iron or its oxides (siderosis), barium (baritosis), tin (stannosis), and emery. Such cases usually present an abnormal X-ray picture without clinical evidence of disease or disability. Even these six groups do not complete the list, but it will be enough to show that a wide range of pulmonary diseases can be caused by the inhalation of dust, which may be both organic and inorganic. It should be remembered, too, that dusts can convey infections such as anthrax and tuberculosis, and indeed pulmonary anthrax (wool-sorters' disease) is a well-defined occupational malady, now fortunately rare in this country.

Dusts and Fumes

To understand the dust diseases it is necessary to know a good deal about the chemical and physical properties of dusts and fumes, the behaviour of dust clouds, and the reaction between them and the tissues of the respiratory tract. Intensive research during the past 30 years has given us a great deal of information on these matters, and a few points may be mentioned here.

Fig. 5—Dust cloud from asbestos boarding showing asbestos (fibres) and amorphous cement dust (× 150).

Both dusts and fumes are composed of particles which, when airborne, can be inhaled into the lungs. Dusts, which may be organic and inorganic (or animal, vegetable, and mineral) are mechanically formed by vigorous action such as grinding, rubbing, crushing, drilling, hammering, and sawing, and in general are of the same chemical composition as the substances from which they come. Fumes (the term is often used incorrectly) are the result of condensation of particles from the gaseous state, and in industry are usually the oxides formed from hot or boiling metals. Iron oxide, for instance, when it comes hot off the welding arc is a fume, but when it is in the form of rust knocked off an iron girder it is a dust. In a dust cloud (fig. 1) the particles remain separate, but in a fume they tend to flocculate and form large masses composed of very small particles.

It has been shown that most of the particles of dust or fume which get into the lung tissue are about 3 μ or less in diameter, and it is often assumed that anything larger cannot get into the alveoli. But these structures measure up to 100 μ across and there seems to be no reason why particles much larger than 3 μ cannot get into them. The factor which determines the size of dust particles found in the lungs is the diameter of the lymphatic channels, through which they are taken by phagocytes. It is true, however, that many of the larger particles are trapped in the nose and the upper respiratory passages.

Fig. 6—Leather dust composed mainly of non-striped muscle-fibres (×1240)

TABLE II—DEATH FROM ALL TYPES OF PNEUMOCONIOSIS IN ENGLAND AND WALES 1940–51

Industry	1940	1941	1942	1943	1944	1945	1946	1947	1948	1949	1950	1951	Totals
Potteries	53	45	47	41	32	41	49	54	48	63	73	62	608
Sandstone	109	82	54	77	57	65	61	55	68	51	29	71	779
Grinding of metals, &c.	41	26	26	21	24	22	32	27	26	37	22	21	325
Refractories	16	12	7	7	6	9	8	10	13	13	8	8	117
Miscellaneous	7	5	7	5	13	19	14	26	27	25	70	129	347
Coalmining	232	196	230	276	311	387	421	577	639	756	846	937	5808
Other mining	64	40	46	43	39	40	51	50	49	64	42	51	579
Asbestos	11	17	11	8	10	11	16	15	15	17	12	18	161
Cotton (byssinosis)	..	..	6	7	1	10	3	4	8	7	11	8	65
	533	423	434	485	493	604	655	818	893	1033	1113	1305	8789
Non-occupational	650	461	443	493	531	529	568	616	651	554	679	732	6907

Each dust cloud has its own characteristic appearance under the microscope. In fig. 1 is shown the dust of quartz or free silica. It is composed of small crystals which remain separate in the dust cloud. By contrast, the appearance of iron oxide fume is shown in fig. 2. The large black masses are made up of hundreds of small particles. Fig. 3 shows the microscopic appearances of kieselguhr or diatomite, a non-crystalline form of free silica. Fig. 4 shows the dust of talc or French chalk, which is composed of plates and a few fibres, while in fig. 5 asbestos dust is seen to be made up mainly of fibres. Leather dust is shown in fig. 6 to be made up of non-striped muscle-fibres; wood dust (fig. 7) has characteristic transverse striations.

The Body's Defence Mechanisms Against Dust

If a man's lungs are healthy they can deal with a good deal of dust or fume without becoming damaged. The body's defence mechanisms against dust are briefly: (*a*) the vibrissæ of the nose, which act as a partial filter for the larger particles; (*b*) the mucous secretions of the nose and the upper respiratory passages, in which a large proportion of the particles is trapped and then expelled by (*c*) the wave-like action of the cilia of the nasal and bronchial epithelium. Below the respiratory bronchioles, where there are no cilia, (*d*) the phagocytes come into play. They engulf the dust particles and take them up to the area of ciliary action, or into the lung lymphatics. Recent work has cast doubt on the hypothesis that dust is taken into the lung parenchyma by phagocytic action, but the fact remains that dust does get into the lungs, whatever may be the exact method of locomotion.

The concentration of dust which can be inhaled without danger varies according to the nature of the dust and also to the length of time that a man is breathing it. Again, the intermittent exposure to high concentrations of dust may be more dangerous than exposure to lower concentrations over a longer period. The harder the job is, the more deeply will a man have to breathe, and in consequence he will breathe more dust. Individuals, too, vary greatly in their capacity to deal with dusts, and of two men who have been working at the same job for the same length of time one may get a disease of the lungs and the other may be unaffected. This is one reason why I am not greatly impressed by the validity of what are known as the maximum allowable concentrations of dusts (M.A.C.), of which lists have been drawn up in various countries. The M.A.C.s seem to be based on the assumption that man is a standardised machine, which clearly he is not. The reasons for the differences in individual reaction to dust are not accurately known, but it is likely that they depend on anatomical, physiological and biochemical variations from one person to another.

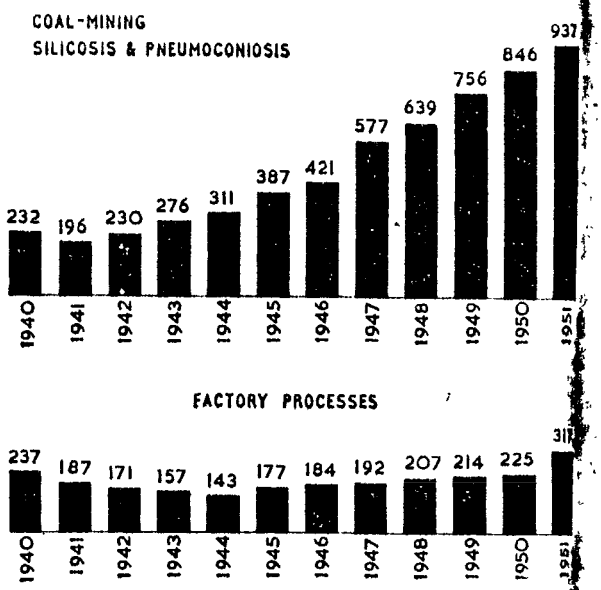

Fig. 8—Chart showing trends of deaths from fibrosis of the lung coalminers and factory workers during the period 1940-51 inclusive.

Fig. 7—Dust of wood-pulp (× 300).

is known, however, that previous damage to the lungs is a factor which leads to the retention of dust in them. In any case, there are instances where people have spent long years in the dusty trades and have died from causes other than the dust diseases. On the other hand, many thousands have died as a direct result of the inhalation of dust.

The Size of the Problem

How many deaths have occurred from the dust diseases? Our information is incomplete in many respects but table II gives at least some idea of the size of the problem. It is based on figures supplied to the Factory Department by the Registrar-General, and it shows the number of deaths from fibrosis of the lung from 1940 to 1951 inclusive.

In all industries there were 8789 deaths from occupational fibrosis of the lung in the 12-year period and it will be seen that the total yearly figures are going up. Over the same period there were 6907 deaths from non-

occupational fibrosis of the lung. It should be emphasised that the deaths occurring each year in the occupational group are the result of conditions which obtained in industry some years previously, possibly 10–20 years or even longer. About two-thirds of the total number of deaths occurred in coalminers, and the figures rose steeply from 232 in 1940 to 937 in 1951. It is likely that part of the increase is due to more accurate diagnosis or at least to greater interest in the pneumoconiosis problem amongst coalminers. In fig. 8 is shown a comparison, based on the crude figures from table II between the deaths in coalminers and factory workers. In factory processes the yearly number of deaths was going down until 1943 but there has been a slight rise in the later years.

(To be concluded)

A FOLIC-ACID EXCRETION TEST IN THE INVESTIGATION OF INTESTINAL MALABSORPTION

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IN 1880 Manson, having anglicised a similar Dutch word, described "sprue" as a disease occurring in the tropics or subtropics or among persons who had previously resided in warm climates; the disease was characterised by glossitis and stomatitis, by the passage of pale copious loose fermenting stools, and by flatulence, wasting, and anæmia. The phrase "the sprue syndrome" has been used in recent years to include the features found in idiopathic steatorrhœa of non-tropical origin and in cœliac disease; since the introduction of the fat-balance test of Cooke et al. (1946) it has been extended to include cases where there is megaloblastic anæmia and deficient absorption of fat but little other clinical evidence of intestinal malabsorption, and the term "malabsorption syndrome" has come into use.

In the diagnosis of sprue the fat-balance test is most useful, but it suffers from the fact that the patient has to be in a hospital where a suitable diet can be given, that all the food given must be consumed (or necessary corrections made), and that the stools must be collected for several days, always a difficult matter in general hospital wards.

It has usually been considered rather surprising that the megaloblastic anæmia of sprue can be treated with folic acid administered *by mouth*. The seeming paradox of successfully treating a condition believed to be due to malabsorption of hæmopoietic factors by giving these same substances orally has led to the hypothesis that the fault in sprue is malabsorption of naturally occurring folic-acid conjugates rather than of folic acid itself. (In some instances it appears that there is malabsorption of vitamin B_{12}, since therapy with parenterally administered vitamin B_{12} has been effective in a few patients.)

The present investigation indicates that there is, in fact, considerable deficiency of absorption of folic acid in sprue, and that advantage can be taken of this to devise a test of folic-acid absorption and excretion for the diagnosis of malabsorption by the small intestine.

The estimation of the folic-acid content of the urine or other body-fluids is usually done microbiologically. The growth of a certain strain of a streptococcus or lactobacillus in a suitable medium depends on the amount of folic acid present in that medium. Thus it is possible to set up tubes containing the medium with measured amounts of folic acid and others containing the medium with various dilutions of urine. In both sets of tubes the amount of growth depends on the concentration of folic acid. The amount of growth is measured photo-electrically after a suitable period of incubation, by estimation of turbidity in the medium, and the amount of folic acid in the urine may easily be calculated. The test has many difficulties but when it has been successfully initiated it is satisfactory, giving reproducible results. If further work confirms the value of the folic-acid excretion test, the diagnosis of sprue should, at least in some instances, be rendered easier in that special diet is not required, and specimens of urine can be sent for assay from other hospitals to laboratories in which folic-acid tests are being done. It is not yet possible to say whether a positive fat-balance test may be associated with a negative folic-acid excretion test in the "malabsorption syndrome." No cases of chronic pancreatic disease have been available for study.

METHODS AND MATERIALS

The folic acid used in these tests, whether it was given by injection or by mouth, was derived from ampoules of 'Folvite' (Lederle Laboratories Ltd.). The ampoules were taken from batches tested by us and found to contain 15 mg. of folic acid per ml. Certain batches were rejected because their content proved to be higher than that stated on the label. Most of the ampoules were, in fact, taken from one batch, supplied by Dr. A. T. Mennie, of Lederle Laboratories Ltd., London. In any one patient the same batch was used for all the tests.

On two occasions, where the intention was to load the tissues by large doses (cases 33 and 37, table III) hospital stock ampoules were used.

The dose was accurately measured in a tuberculin syringe. For tests of excretion following oral therapy this test dose was diluted with a small quantity of water.

24-hour collections of urine were made in brown bottles containing toluene and a phosphate buffer of pH 6·8. The greatest possible care was taken to ensure that the urines were total 24-hour specimens. Recovery experiments in which folic acid was added to urine gave completely satisfactory results. The urines were kept in a refrigerator at 4°C, and readings were usually made within 3 days. All the readings were made at least in duplicate.

Folic acid was estimated by the method of Teply and Elvehjem (1945), *Streptococcus fæcalis* R being used as the test organism. The tubes were incubated at 37°C for about 16 hours, and readings were made by turbidity estimations in a Spekker photo-electric absorptiometer. No correction need be made for the resting urinary content of folic acid, since in ten normal persons the mean urinary folic-acid content corrected for citrovorum factor was only 1·7 µg. (range 0·12–3·6 µg.) per 24 hours.

Urinary citrovorum factor was estimated in most instances, since citrovorum factor is also a growth factor for *Strep. fæcalis*, the organism used in the folic-acid assay. Citrovorum factor was estimated by a modification of the method of Sauberlich and Baumann (1948), with *Leuconostoc citrovorum* as the test organism. Here, too, turbidity readings were made after incubation at 37°C for about 16 hours. The mean resting urinary content of citrovorum factor in ten normal persons was 0·84 µg. (range: negligible amount—3·3 µg.) per 24 hours. No correction has been made for this in the citrovorum-factor readings, which are included only for biochemical interest.

It will be seen that a small proportion of the folic acid administered in these investigations was excreted as citrovorum factor or as a substance with similar microbiological properties, but the figures given for folic-acid excretion have not been corrected for this, because such a correction would in no way alter the conclusions.

Several of the patients were in hospitals in outlying districts where it was impossible to make as full investigations as would have been preferred. In some instances it was necessary to collect the urine at the patients' homes.