confused role ; he is a personal medical adviser to his patient in the Hippocratic tradition and at the same time a guardian of the sick-pay funds. It is little wonder that he is sometimes acutely conscious of a dilemma.

The industrial medical officer can also feel himself to be in a difficulty. He claims, with reason, to be part of the management team, but at the same time to regard his relationship with an employee of the firm as being one between doctor and patient, with the consequent ethical obligations. He is anxious to avoid the image of the former Workmen's Compensation doctors. But these practitioners disappeared not because industrial medicine changed its standpoint but because payment for injury at work (common law claims apart) was transferred in 1948 from the employer, or his insurer, to the State. Nowadays many firms pay large sums in sickness benefit, and it is understandable that they should again look to medicine for help, just as the Department of Health and Social Security looks to its regional medical officers.

As the State gradually increases its share of sick pay, can we foresee an increase in the State control of payment during sickness absence as happened when it took over payment for injury at work and for the prescribed diseases in 1948 ? Is the present system of regional medical officers adequate, or should it have closer ties with industry so that more attention may be given to resettlement ? The Department of Employment and Productivity has recently proposed alterations in the duties of appointed factory doctors,[15] and in their widened role (enigmatically called the " A " doctor service) they might form an industrial link with the regional medical officers. This would be easier if the two operated under one Ministry. In many instances resettlement would be easier if the present binary " fit/unfit " used by the State and by most of industry were to be altered to a more flexible system. A. W. Gardner[16] has suggested that less rigidity in this sphere might result in the 2% increase in productivity which Mr. Aubrey Jones has calculated to be required for national solvency.

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Viruses of Birds, Mice, and Men

One of the fascinations of scientific work is when patiently accumulated facts suddenly " fit together " in a new and interesting way. This has recently occurred with three apparently unrelated viruses which, known to cause diseases of man and animals, have now been shown to belong to one large biological group.

Several years ago some new viruses were isolated from laboratory mice.[1] They were found under various circumstances, often being recovered from the alimentary tract, liver, and spleen of apparently normal animals. Nevertheless, when they were grown in preparations of mouse brain, and reinjected into young animals or into those infected with the parasite *Eperythrozoon coccoides*, these viruses produced a severe and fatal hepatitis. Since then it has been found that they are not related to the virus responsible for infectious hepatitis in man, though some specimens of human sera have been found to contain antibodies against these particular mouse viruses.

The second finding was that flocks of poultry reared in broiler houses may become infected with avian infectious bronchitis virus.[1] The latter may cause an acute infection of the respiratory tract, and—of greater economic importance —may also damage the genital tract, causing a serious decline in egg laying. The virus exists in several serotypes, and vaccines against them have been used to try to control the disease. Antibodies against this virus have also been found in man, and are probably commoner in men who come into contact with poultry.[2] Nevertheless, so far there is no direct clinical evidence of human infection with the virus.

The third apparently unrelated finding was the cultivation by workers using organ cultures of human trachea at the Common Cold Research Unit at Salisbury of a " new " virus.[3] Though this agent caused streaming colds, it was not a rhinovirus, for it was ether-labile ; neither was it one of the ether-labile myxoviruses, which are also known to cause colds. Almost simultaneously D. Hamre and J. J. Procknow,[4] in the United States, described another " new " respiratory pathogen which grew in tissue cultures but was an ether-labile non-myxovirus. It was then shown by June Almeida[5] that

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both this and the virus discovered at Salisbury had identical appearances under the electron microscope. Moreover, many of their features had never been seen before in a virus recovered from man, but they were indistinguishable from the viruses causing mouse hepatitis[6] and avian infectious bronchitis.[7-11] A further stage in the story was provided by K. McIntosh and his co-workers, who used both organ culture and electron microscopy to study the properties of six more viruses and successfully adapted two of them to suckling mice.[12 13] They compared these and other viruses by complement-fixation tests and found that there were cross reactions between mouse hepatitis viruses and the human strains.

Because of all these findings it has now been suggested that, in spite of their different origins and pathogenicity, these viruses belong to a single biological group. The group name suggested is coronaviruses—to recall the crownlike outline of some particles in electron micrographs, and their resemblance to the solar corona. The name is much less of a mouthful than avian-infectious-bronchitis-like viruses, but, though it has so far been well received, it is still unofficial.

The importance of the human viruses belonging to this group is still being studied, and will be helped by the recent discovery of a human cell line (L132) which may bring isolation of these organisms within the scope of any virus laboratory.[16] There is also a need to find out exactly how many human serotypes there are—at present three are distinguished with certainty.[14 17] Since some of the human strains are related to the mouse hepatitis virus it is not surprising that some human sera contain antibodies against this and that the titre of these antibodies may rise in the course of a respiratory infection. Of 31 specimens, mostly from adults with colds, four yielded coronaviruses,[14 18] while serological studies have shown that children can be infected as well and that antibodies against some strains are common in normal subjects. In adults about 7% of minor respiratory disease may be due to one serotype, or to one related to it antigenically. In children admitted to hospital, on the other hand, only one out of nearly 1,000 cases of respiratory diseases were due to this serotype.[20] Hamre and Procknow detected small epidemics of common colds due to the virus in students,[4] and similar epidemics have been detected in staff working in a laboratory near Washington. The infections seem to occur between December and April and to vary in frequency from year to year. Clearly a new chapter has been started in the story of the causes of the common cold—and it is headed "coronaviruses."

Anaemia in Rheumatoid Arthritis

Some degree of anaemia is present in about two-thirds of patients with active rheumatoid arthritis, and an understanding of its causes is necessary for successful treatment.

The commonest form of anaemia is the type which has been labelled the anaemia of chronic disorders. It has been reviewed by G. F. Cartwright.[1] Erythropoiesis is depressed in consequence of the primary disease state. The severity of the anaemia is related to the activity of the rheumatoid process.[2] The red cells may be normal in appearance or they may be hypochromic. If the latter, the mean corpuscular haemoglobin concentration is low. The resemblance to true iron deficiency anaemia is heightened by the presence of a low serum level of iron. But the anaemia differs from iron deficiency anaemia in that the serum-iron-binding capacity is reduced, whereas it is raised in true iron deficiency. In the anaemia of chronic disorders adequate iron stores are generally to be found in appropriately stained marrow spreads, and, more important, there is no response to iron therapy. Indeed, failure to respond to iron therapy is often an indication of the nature of the anaemic process.

Sometimes this type of anaemia is combined with true iron deficiency. In rheumatoid arthritis this is often due to gastrointestinal haemorrhage resulting from salicylate treatment, but other causes of blood loss leading to true iron deficiency should not be overlooked. Under these circumstances there is some response to treatment with iron, and the level of haemoglobin attained is related to the activity of the rheumatoid process.

Less frequently the anaemia is megaloblastic in character. R. E. H. Partridge and J. J. R. Duthie[3] have reported that 27 out of 2,544 patients with rheumatoid arthritis had pernicious anaemia, whereas only some 15 cases were encountered among 5,515 controls. Thus there was a five-fold higher frequency of pernicious anaemia among the rheumatoid group. Nevertheless, this should not be interpreted as conclusive evidence of an association between pernicious anaemia and rheumatoid arthritis.

The frequency of pernicious anaemia recorded by Partridge and Duthie[3] (10.5 per 1,000 patients with rheumatoid arthritis) is about the same as the expected frequency of pernicious anaemia in an elderly predominantly female Scottish population.[4 5] Nor do data on the frequency of parietal-cell antibodies in rheumatoid arthritis lend support to an association between those disorders, and a recent study[6] failed to find any difference between patients with rheumatoid arthritis and matched controls. It may be that the apparently higher frequency of pernicious anaemia in rheumatoid arthritis[3] is due to missed cases in the large control group comprising patients with degenerative joint disease, who may not have been seen as frequently by physicians as patients with rheumatoid arthritis.

It has also been suggested that there is an increased frequency of folate deficiency, and of megaloblastic anaemia due to folate deficiency, among patients with rheumatoid arthritis.[7] A detailed study of the folate status of such patients has been reported by A. Omer and A. G. Mowat.[8] They found that two-thirds of patients with rheumatoid arthritis had both "low" serum folate levels and an increased excretion of formiminoglutamic acid (figlu) in the urine, one-third had a "low" level of folate in red cells, and one-fifth (8 out of 37 rheumatoid patients) were thought to have early megaloblastic changes in cells obtained by marrow aspiration.

How should these data be interpreted? Firstly, it is worth remembering that biochemical evidence of folate

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