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Pointers

Combined Immunization: Addition of aluminium hydroxide to triple antigen vaccines used in primary immunization at 3 to 6 months of age both increased potency and reduced tendency to cause reactions (p. 663).

Nephrotic Syndrome in Childhood: Cyclophosphamide proved effective therapy in 46 children whose renal biopsy showed minimal changes but who had developed dependence, resistance, or toxic effects to steroid therapy (p. 666); leader at p. 660.

Rosacea: Treatment by prolonged topical applications of fluorinated steroids in 14 patients produced aggravation and extension of telangiectasia, and marked rebound phenomena. Hydrocortisone and oral tetracycline were substituted satisfactorily three months later (p. 671).

Iron in Food: Extraction of iron in food, and study of effects of cooking, indicate that only about half the total iron is available for absorption (p. 673).

Diabetes in Remission: Apparent relationship between degree of remission and magnitude of insulin response to glucose demonstrated in three juvenile patients (p. 676).

Self-poisoning: Intensive supportive and conservative care, occasionally supplemented by forced diuresis, resulted in a very low mortality in severely self-poisoned patients (p. 679).

Estimating Ovarian Function: Gynaecography considered a simple, safe, and valuable adjunct to clinical and laboratory findings for diagnosis and management of infertility and menstrual irregularity (p. 682).

Myenteric Plexus Lesion: Two cases of bowel obstruction associated with a destructive lesion of the myenteric plexus (p. 686).

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Absence from Work Attributed to Sickness

In 1967 about 2·5 million man-days were lost owing to industrial disputes and about 300 million man-days owing to sickness absence. The last 40 years have seen a substantial fall in death rates at all ages under about 55, but there has been no reduction in sickness absence,[1] though such absences have been reduced in schoolchildren.[2] Sickness absence, both certified and uncertified, is not distributed in a random fashion, for some persons consistently incur more than others,[3–5] but in any year the majority of persons entitled to claim do not do so.

Since the operation of the National Insurance Act, 1946, began in 1948 the flat rate of sickness benefit for a single person has increased by nearly 250% (from 26s. per week to £4 10s.), whereas over the same period wage rates have increased by about 150%.[6] When an employer supplements National Insurance benefits up to full pay, the employee off sick may be better off financially through tax relief and not having to pay contributions than when he is at work. Absence attributed to sickness is generally considered to be a poor index of physical illness.[1 7 8] Nevertheless, this means of withdrawing from work is of sufficient importance to warrant an examination of it and of the doctor's role.

A symposium on absence from work attributed to sickness held by the Society of Occupational Medicine has brought together the contributions of a number of experts.[9] Of the episodes of sickness absence among the male patients in a general practice 51% were found to last for seven days or less.[5] It was generally held that absences of one to three days are not primarily a medical problem. Indeed, they are a long-established socio-economic fact stemming from the days when men could pick their own time for work, and they were one of the perennial problems of the early industrialists.[10] The introduction into one organization of a sick-pay scheme on the basis of full pay from the first day of absence (with certification not being required for short absences) increased the number of 1–3-day absences by nearly five times.[11] On the other hand at an oil refinery where payment from the first day of sickness absence was already made the introduction of self-certification for short-term absences did not encourage serious abuse[12] but merely accelerated a trend, already present, towards the taking of more frequent but briefer spells.[13]

The control of sickness absence remains a compromise between reasonable action and unreasonable prying. The Department of Health and Social Security requires doctors' certificates, but at the start of an illness, in the absence of physical signs, it may be argued that the patient determines whether he should stay off work, though it is the doctor who signs the note.[5 14] In the more obvious cases with physical signs, such as a fracture or high fever with prostration, it hardly needs a medical man to decide that the patient is unfit for work. Similar considerations may be applied to the decision to return to work. The family doctor has a

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.

1 Morris, J. N., *Proc. roy. Soc. Med.*, 1965, **58**, 821.
2 Morris, J. N., *Uses of Epidemiology*, 1964, 2nd ed. Edinburgh and London.
3 Taylor, P. J., *Brit. J. industr. Med.*, 1967, **24**, 169.
4 Froggatt, P., *Trans. Soc. occup. Med.*, 1968, **18**, 89.
5 Carne, S., *Brit. med. J.*, 1969, **1**, 147.
6 Ager, J. E., in *Proceedings of a Symposium on Absence from Work Attributed to Sickness*, 1968, p. 38, ed. A. W. Gardner. Society of Occupational Medicine, London.
7 Ashworth, H. W., *Proc. roy. Soc. Med.*, 1965, **58**, 825.
8 Howell, M. W., in *Proceedings of a Symposium on Absence from Work Attributed to Sickness*, 1968, p. 38, ed. A. W. Gardner. Society of Occupational Medicine, London.
9 *Proceedings of a Symposium on Absence from Work Attributed to Sickness*, 1968, ed. A. W. Gardner. Society of Occupational Medicine, London.
10 Froggatt, P., in *Proceedings of a Symposium on Absence from Work Attributed to Sickness*, 1968, p. 114, ed. A. W. Gardner. Society of Occupational Medicine, London.
11 Ager, J. E., in *Proceedings of a Symposium on Absence from Work Attributed to Sickness*, 1968, p. 42, ed. A. W. Gardner. Society of Occupational Medicine, London.
12 Taylor, P. J., *Brit. med. J.*, 1969, **1**, 144.
13 Taylor, P. J., in *Proceedings of a Symposium on Absence from Work Attributed to Sickness*, 1968, p. 77, ed. A. W. Gardner. Society of Occupational Medicine, London.
14 Handfield-Jones, R. P. C., *Lancet*, 1964, **2**, 1173.
15 *The Appointed Factory Doctor Service*, Ministry of Labour, Report by a Subcommittee of the Industrial Health Advisory Committee, 1966. H.M.S.O., London.
16 Gardner, A. W., in *Proceedings of a Symposium on Absence from Work Attributed to Sickness*, 1968, p. 51, ed. A. W. Gardner. Society of Occupational Medicine, London.

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

1 Andrewes, Sir C., and Pereira, H. G., *Viruses of Vertebrates*, 2nd ed., 1967. London, Baillière, Tindall and Cassell.
2 Miller, L. T., and Yates, V. J., *Amer. J. Epidemiol.*, 1968, **88**, 406.
3 Tyrrell, D. A. J., and Bynoe, M. L., *Brit. med. J.*, 1965, **1**, 1467.
4 Hamre, D., and Procknow, J. J., *Proc. Soc. exp. Biol. (N.Y.)*, 1966, **121**, 190.
5 Tyrrell, D. A. J., and Almeida, J. D., *Arch. ges. Virusforsch.*, 1967, **22**, 417.
6 Almeida, J. D., and Mallucci, L., unpublished findings.
7 Berry, D. M., Cruickshank, J. G., Chu, H. P., and Wells, R. J. H., *Virology*, 1964, **23**, 403.
8 David-Ferreira, J. F., and Manaker, R. A., *J. cell Biol.*, 1965, **24**, 57.
9 Becker, W. B., McIntosh, K., Dees, J. H., and Chanock, R. M., *J. Virol.*, 1967, **1**, 1019.
10 Hamre, D., Kindig, D. A., and Hann, J., *J. Virol.*, 1967, **1**, 810.
11 Nazerian, K., and Cunningham, C. H., *J. gen. Virol.*, 1968, **3**, 469.
12 McIntosh, K., Dees, J. H., Becker, W. B., Kapikian, A. Z., and Chanock, R. M., *Proc. Nat. Acad. Sci. (Wash.)*, 1967, **57**, 933.
13 McIntosh, K., Becker, W. B., and Chanock, R. M., *Proc. nat. Acad. Sci. (Wash.)*, 1967, **58**, 2268.
14 Bradburne, A. F., unpublished findings.
15 *Nature (Lond.)*, 1968, **220**, 650.
16 Bradburne, A. F., *Nature (Lond.)*, 1969, **221**, 85.
17 McIntosh, K., Kapikian, A. Z., Hardison, H. A., and Chanock, R. M., *J. Immunol.*, 1969, in press.
18 Tyrrell, D. A. J., Bynoe, M. L., and Hoorn, B., *Brit. med. J.*, 1968, **1**, 606.
19 Bradburne, A. F., Bynoe, M. L., and Tyrrell, D. A. J., *Brit. med. J.*, 1967, **3**, 767.
20 Kapikian, A. Z., James, H. D., Kelly, S. J., Dees, J. H., McIntosh, K., Parrott, R. J., and Chanock, R. M., *J. inf. Dis.*, 1969, in press.