Taylor further noted that in London in the epidemic of 1946–47 there was a transposition of what customarily would have been the successive minor and major phases. This, together with the epidemic of 1948–49 just terminated (or terminating), may lend some support to the view that at least transiently annual rather than biennial epidemicity has become a feature of London measles. It was anticipated that "the minor phases as such may cease and, if enlarged and separated by definite sub-epidemicity from what follows, be regarded justly as completed epidemics. In that case, of course, biennial periodicity disappears and is replaced by annual recurrences." [2] The two—or, so regarded, four—most recent epidemics in London (1946–47 and 1948–49) may be instanced as illustrative of this anticipation, but it must not be overlooked that anomalies of incidence in London are to be expected.

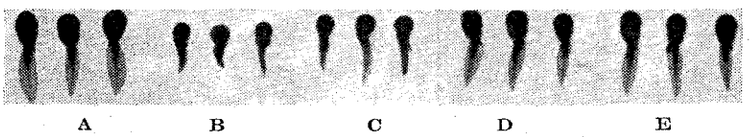
The exodus of children at the beginning of the war could not fail profoundly to modify the course of measles prevalence in the metropolis. That for 1940–41, for instance, was diminutive; nevertheless, in attenuated form the features dominantly defined in composite graphs of the three epidemics of 1940–41, 1942–43, and 1944–45 are clearly to be traced ab origine. The epidemics of 1946–47 and 1948–49, while retaining anomalously, if regarded as biennial epidemics, the features of such, would if they were characteristically recurrent over a prolonged period be seen, I think, as annual epidemic recurrences. Extensively, during the period of evacuation, children who normally would have contracted measles in London escaped, and with their return have added to the numbers of susceptibles in the metropolis. This change in the immunity constitution of the population, the raising of the fee for notification which has made it worth while for the practitioner to seek out family contact sufferers from the disease, and other circumstances, contribute to such anomalies as those we are now witnessing.

Ministry of Health,
London, S.W.1. WILLIAM BUTLER.

ÆTIOLOGY OF GRAVES'S DISEASE

SIR,—In his most lucid and stimulating lecture, published in your issue of Sept. 24, Professor Means asks "How actually does thyroid hormone act on cells, and what potentiates or inhibits such action?" and he continues: "To these questions we badly need answers."

The problem of how the action of the thyroid hormone and of other hormones may be modified once they have been released by the glands, was studied many years ago by H. Zondek and myself.[4] The results of these old



A B C D E

Tadpoles on 4th day of experiment: A, controls; B, thyroid action at pH 6·5; C, thyroid action on addition of 66 mg. calcium chloride per 100 ml. water; D, thyroid action on addition of 200 mg. calcium chloride per 100 ml. water; E, thyroid action at pH 7·4.

experiments are, I believe, interesting enough to be briefly recalled.

Using the metamorphosis of tadpoles as a test for thyroid hormone, we found that comparatively slight changes of the pH of the water in which the tadpoles were kept modifies thyroid action. Metamorphosis took place much more rapidly at a pH of 6·5–7 and of 7·7–8·5, than at a pH of 7–8 and below 6·5; and we concluded that the changes in hydrogen-ion concentration, as they occur in the body and between blood and tissues, may perhaps cause variation in hormonal response. Previously, Zondek and Reiter [5] had observed that addition of small quantities of potassium and calcium salts enhanced and inhibited, respectively, the influence of thyroid hormone on the metamorphosis of tadpoles. The accompanying figure, taken from our own experiments, illustrates the action of thyroid hormone on the metamorphosis of tadpoles when calcium chloride is added to the water and when this is buffered at a pH of 6·5 and 7·4.

Similar observations were made with respect to other hormones. Zondek and I [6] found that addition of small quantities of calcium chloride to insulin diminished and sometimes even reversed the action of insulin on the blood-sugar. McCarrison [7] described modification of the action of adrenaline by alteration of the pH, and Kylin [8] observed similar changes on addition of potassium and calcium salts.

Thus it appeared that variations in the concentration of hydrogen ions and of certain electrolytes might modify hormonal action.

London, W.1. H. UCKO.

4. Zondek, H., Ucko, H. *Klin. Wschr.* 1924, no. 29, p. 1752.

INTRAVENOUS IRON IN RHEUMATOID ARTHRITIS

SIR,—I was interested to read the results reported by Dr. Sinclair and Dr. Duthie in your issue of Oct. 8.

Early this year I gave several patients with long-standing active rheumatoid arthritis a course of intravenous iron ('Ferrivenin,' Benger). The general régime was much the same as that of Sinclair and Duthie.

So far I have detailed results in only 3 cases. Each of these patients had been under observation for at least a year, during which they had been taking iron regularly by mouth. In no case was the anæmia very pronounced, and the intravenous iron was given in the hope that, apart from raising the hæmoglobin level to normal, it would produce more general improvement which could be measured by means of the erythrocyte-sedimentation rate (E.S.R.). Hæmoglobin was estimated by the Haldane method; and the E.S.R. at the end of one hour by the Westergren method.

CASE 1.—A man, aged 40 years, who had had psoriasis arthropathica for the past 6 years.

Date	*Hb, %*	*E.S.R., mm.*	*Intravenous iron, mg.*
Nov. 17, 1948	92	39	—
Jan. 10, 1949	82	40	—
Jan. 10–29	—	—	1370
Feb. 7	90	43	—
Feb. 25	94	46	—
June 2	78	64	—
July 14	80	60	—

CASE 2.—A man, aged 31 years, with a history of rheumatoid arthritis for the past 3 years.

Date	*Hb, %*	*E.S.R., mm.*	*Intravenous iron, mg.*
July 5, 1948	102	86	—
Dec. 31	80	71	—
Jan. 10, 1949	88	64	—
Jan. 10–31	—	—	1470
Jan. 25	88	62	—
Feb. 7	92	52	—
Feb. 16	98	66	—
May 26	86	71	—
Sept. 16	92	89	—

A total of 1 g. of 'Myocrisin' was administered between July and October, 1948, and a further 1 g. between May and July, 1949.

CASE 3.—A man, aged 33 years, who had had rheumatoid arthritis, associated with fairly severe bronchiectasis, for 7 years.

Date	*Hb, %*	*E.S.R., mm.*	*Treatment*
March 25, 1948	76	50	—
March 26	—	—	Blood-transfusion, 600 ml.
March 30	82	36	—
April 15	92	22	—
July 29	98	49	—
Jan. 20, 1949	76	55	—
Jan. 29 to Feb. 14	—	—	Intravenous iron, 1280 mg.
Feb. 16	82	55	—
March 30	80	52	—

The bronchiectasis was treated during this time with postural drainage and aerosol penicillin inhalations.

5. Zondek, H., Reiter, T. *Z. klin. Med.* 1923, **99**, 139.
6. Zondek, H., Ucko, H. *Klin. Wschr.* 1925, no. 1, p. 6.
7. McCarrison, R. *Brit. med. J.* 1923, i, 101.
8. Kylin, E. *Klin. Wschr.* 1925, no. 11, p. 501.

Thus in these 3 cases the disease became no less active, as judged by changes in the E.S.R. All the patients, including these 3, said that they felt much better and had less pain in the joints while treatment with intravenous iron was being continued and for about a fortnight afterwards. It is difficult to say how much of this symptomatic improvement was due to the psychological effect of a new treatment. On no occasion were any toxic effects noted.

I wish to thank the Director-General of Medical Services, Ministry of Pensions, for permission to publish these cases.

Ministry of Pensions Hospital, Ronkswood, Worcester. A. M. CLARK Medical Officer.

CONTROL OF TUBERCULOSIS

SIR,—I doubt whether, under the existing circumstances, many chest physicians will feel able to accede to Dr. Temple Clive's request, in his letter of Oct. 22, that their patients should be admitted to sanatoria "preconditioned" by bed rest only.

Many patients have to wait up to six or nine months before being admitted to a sanatorium, and during this period not a few progressively deteriorate despite bed rest, which in many cases cannot be properly applied in the patient's own home. Does Dr. Temple Clive seriously suggest that these cases should not receive the benefit of collapse therapy or of treatment with streptomycin or para-aminosalicylic acid when they are in the earliest, and therefore most treatable, stage? Dr. Temple Clive states that the results of domiciliary collapse therapy have not been uniformly successful, but collapse therapy instituted in a sanatorium is also unsuccessful in many instances; success in this field depends more on selection of the right type of case than on whether treatment is instituted in the home or in a sanatorium.

So long as there are not sufficient sanatorium beds for the treatment of the tuberculous it is up to the chest physician to treat his patients as best he can as soon as he can, and to this end the use of domiciliary collapse therapy appears to be entirely justified. Moreover, the suggestion that it is dangerous for the chest physician to treat his cases in the patients' homes, whether by collapse therapy or with streptomycin or para-aminosalicylic acid, is unfair to the ability of the chest physician, who is quite as able to assess his patients and to plan their treatment as is the sanatorium physician.

Frodsham, Cheshire. M. B. PAUL.

SIR,—In his letter of Oct. 22, Dr. Temple Clive warns us that domiciliary collapse treatment in pulmonary tuberculosis should not be undertaken lightly. I should like to support his warning most strongly.

Domiciliary collapse treatment, like any other treatment, can only give good results in experienced hands and if all the necessary facilities are available. In my opinion this treatment should only be undertaken if the following conditions obtain: (1) careful selection of cases; (2) adequate provision for the care of the patients at home, including home help if necessary; (3) a chest physician constantly on call to deal with emergencies; (4) adequate facilities for adhesion-section and for phrenic crushes; and (5) a reliable transport system to convey patients from their homes to the clinic for refills and X-ray control, and, if necessary, to institutions for minor surgery.

I think, however, that domiciliary collapse treatment has come to stay, until the time when it will again be possible to admit tuberculous patients to hospital without much delay.

Since 1945 I have induced in the Hounslow chest-clinic area nearly 300 pneumoperitoneums and artificial pneumothoraces in the patients' homes. A preliminary report on the first 200 cases has lately been published,[1] and I think that the results certainly bear comparison with the results of institutional treatment.

I should like to plead for the more frequent use of the pneumoperitoneum as a preliminary collapse measure, especially as this will give satisfactory results even in less experienced hands and where there are less facilities for minor surgery and frequent X-ray control.

Hounslow, Middlesex. R. HELLER Chest Physician.

1. Heller, R. *Tubercle*, 1949, **30**, 204.

NIPPLE-SHIELDS

SIR,—Your correspondents who advocate the use of lead nipple-shields are, unhappily, following in the footsteps of those who have from time to time made thorny the path of preventive medicine.

The first principle in the control of poisoning by specific agents is replacement of the toxic material by one that is harmless, and the arguments adduced in favour of lead shields have a familiar ring to those who recall the struggle of the last half-century to reduce the toll of industrial disease. One of the reasons for the great reduction in the incidence of lead-poisoning among industrial workers since 1900 has been the successful adoption of this principle. At one time lead was an essential ingredient of glazes, and the pottery industry was notorious for its lead-poisoning. Now, leadless glazes have been found to be so successful that their use has been prescribed by law. This is but one example of many that could be mentioned.

The use of lead in occupations is so fraught with risks to health that it is hedged around by innumerable restrictions, yet it is seriously suggested that this most dangerous of metals is a safe substance to interpose between an infant and the breast. Exhortations to cleanliness and restricted use are futile, for there is no-one to see that these instructions are carried out.

I would draw the attention of your correspondents to the late Sir Thomas Legge's second axiom for control of industrial disease. He said: "If you can bring an influence to bear external to the workman (i.e., one over which he can exercise no control) you will be successful; if you cannot, or do not, you will never be wholly successful." For workman read nursing mother, and the rule holds good.

Usher Institute, University of Edinburgh. CATHERINE SWANSTON Lecturer in Industrial Health.

SIR,—Whether or not lead shields can induce healing of a damaged nipple, it seems clear that their use must be given up. I have always thought that any good they did was more likely to be by keeping the injured surface from contact with clothing than from a deposit of lead salts. If so, a shield made of glass or some safe type of plastic material should do equally well and avoid risk to the child.

The very great majority of these injuries will heal quickly—in 24–48 hours rather than the week implied by Dr. Gordon last week—if, from the first experience of pain, suckling is withheld, the milk removed by hand, and the nipple surface protected. This has been the practice at this hospital for many years and has proved most satisfactory. The risk of mastitis is almost wholly saved and the woman spared suffering with its inhibitory action on the milk's outflow. When resumption of feeding reproduces the damage it is almost always because the nipple cannot be drawn by the baby far enough into its mouth. The likelihood of this should have been foreseen in pregnancy and a shield worn to improve its range of protraction.

May I support Mr. Nockolds and Dr. Moragas (Oct. 29) on the advantages of treating breast abscesses by aspiration—with one reservation? I doubt if a child should be allowed to feed at a breast which is acutely infected with *Staphylococcus aureus*. In pre-penicillin days I have known one infant die of enteritis from