Polio vaccines and polioviruses

SIR,—In your leading article "Polio vaccines and polioviruses" (25 June, p 1617) you rightly note some of the disadvantageous aspects of the oral live poliovirus vaccine (dissemination in the community, reversion to virulence, and occasional paralysis or death in vaccinees and contacts).^{1,2} I feel impelled to write to you because you imply, nevertheless, that a live virus vaccine is to be preferred to one made of inactivated or killed viruses because "immunity lasts longer." This statement is not correct so far as killed poliovirus vaccine is concerned, since it has been proved to establish durable immunity.³ Moreover, it produces a herd effect and, properly used, can result not only in eradication of disease but in elimination of poliovirus from a population.³

If a goal of poliomyelitis vaccination is "to eradicate disease and to control the spread of—and eventually to eliminate—the causative agents from the population"⁴ this goal can only be approximated but not obtained with the use of the live virus vaccine. Where the killed virus vaccine has been used such a result has been achieved (Finland, Sweden, and other countries).

In a paper³ that may not have come to your attention you will find a discussion of details, data, and mechanisms related to the control of poliomyelitis via the proper use of the killed virus vaccine.

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¹ Assaud, F A, Cockburn, W C, and Perkins, F T, *Bulletin of the World Health Organisation*, 1976, **53**, 319.

² Schoenberger, L B, McGowan, J E, jun, and Gregg, M B, *American Journal of Epidemiology*, 1976, **104**, 202.

³ Salk, J, and Salk, D, *Science*, 1977, **195**, 834.

⁴ Nightingale, E O, *New England Journal of Medicine*, 1977, **297**, 249.

Lithium and thyrotoxicosis

SIR,—In your leading article (6 August, p 346) "Adverse effects of lithium treatment" it is stated that "there is at least one report of lithium-induced thyrotoxicosis."¹ I wish to report another case of thyrotoxicosis associated with lithium treatment.

In January 1975 a 54-year-old man was admitted to the alcoholism unit of this hospital for the third time since 1973. On re-examination it was considered that he was suffering from depression as well and he was included in a current trial involving the use of lithium in the treatment of chronic alcoholics with depression.² In fact he had lithium carbonate (1200 mg daily) throughout the trial, which in his case began in January 1975. He was discharged from the alcoholism unit in March 1975 and was maintained on lithium. In May 1976 he began to feel unwell, and among his symptoms at times were shakiness and slight dizziness. His general practitioner considered that these symptoms might be a sign of lithium toxicity and so the lithium was discontinued and he was referred to me.

When I saw him he said that for the past two months he had had aching of the muscles of the thighs, buttocks, and calves and very slightly in the arms and neck. For the same period he had noticed shaking for 5-10 min at a time in the form of a fine tremor. He had also noticed that his heart was pounding and at times appeared irregular, and this occurred two or three times a day for periods of about 5 min. He also mentioned that his gait was unsteady and that he was clumsy with his hands. Over this period of two months he had lost about a stone (6½ kg) in weight, despite the fact that he had an enormous appetite.

On examination his weight was 67 kg (10 st 8 lb). His voice was husky (this was later found to be due to hyperkeratosis of the cords). He was sweating, his pulse was 100 min and regular, his blood pressure 130/50 mm Hg, and there were no signs of cardiac failure. His ankle jerks were brisk. The thyroid gland was not palpable. Investigations on 9 June 1976 revealed the serum thyroxine concentration to be more than 257 nmol/l (20 µg/100 ml) (reference range 51-154 nmol/l (4-12 µg/100 ml)), T3 uptake 1.47 (reference range 0.8-1.5), and free thyroxine index >20 (reference range 4-12).

Treatment was started with carbimazole and on 24 November his serum thyroxine level was 9 nmol/l (0.7 µg/100 ml), T3 uptake 0.74, and free thyroxine index 0.5. On 23 February 1977 the thyroxine level was 36 nmol/l (20 µg/100 ml), T3 uptake 0.81, free thyroxine index 2.3, thyroid microsomes positive 1 in 40, blood calcium 2.35 mmol/l (9.4 mg/100 ml) (reference range 2.25-2.6 mmol/l (9.0-10.5 mg/100 ml)), and his weight was 160 kg (11 st 5 lb). He felt well subjectively and there were no symptoms or signs of thyrotoxicosis. There was no relapse into acute alcoholism.

The incidence of thyroid deficiency in patients taking lithium is now well established.^{3,4} The case described by Franklin,¹ to which you referred in your leading article, was "a case of thyrotoxicosis developed in the fourth year of lithium treatment in a 45-year-old woman who had had a pre-existing non-toxic goitre." The author also indicated that "there is no proof that lithium caused thyrotoxicosis" in that case. The case described here is of interest in that symptoms of thyrotoxicosis occurred during lithium therapy in a patient who had no pre-existing goitre.

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¹ Franklin, L M, *New Zealand Medical Journal*, 1974, **79**, 782.

² Merry, J, et al, *Lancet*, 1976, **2**, 481.

³ Schou, M, et al, *British Medical Journal*, 1968, **3**, 710.

⁴ Lazarus, J H, and Bennie, E H, *Acta Endocrinologica*, 1972, **70**, 266.

The grateful patient

SIR,—I would like to try to restore the balance upset by Mrs J Anderson's Personal View (27 August, p 573).

I have been a general practitioner for 28 years which I, unlike her, enjoy remembering. For the past seven years I have practised from a health centre. Certainly things have changed in this time, but not in the way described by Mrs Anderson. My partners, receptionists, and I continue to receive tokens of gratitude from our patients which take the form of verbal thanks, letters, presents, and even small legacies. Yesterday I returned home with three separate presents of home-grown vegetables, and this is by no means unusual. I do not keep a count but I am certain that not a week goes by without a present. Last Christmas my presents, over 70 in number, contributed to our cheer at Christmas and throughout the year, not least because of the sentiment which I knew had inspired their gift.

My practice, though fortunately situated, is by no means unusual. My practice administrator is not a dogsbody but a respected colleague and our whole team works to achieve a mutually confident relationship between the patients and their doctors. Of course there are many difficulties, but the important fact remains that I enjoy seeing the vast majority of my patients, including those who have had

the misfortune to acquire a fat record envelope, and they seem to enjoy seeing me.

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Effect of abortion on obstetric patterns

SIR,—Mr A F Pentecost (27 August, p 578) raises an important point that is still unresolved when commenting on the published account of an interview with one of us (RWB) on changes in obstetrics in the 'seventies (23 July, p 251). In that interview it was implied that the reported decline in the numbers of babies of low birth weight born in Sheffield might be in some measure due to the removal of high-risk pregnancies by legal abortion. Mr Pentecost suggests that such a beneficial effect is likely to be outweighed by the increase in premature labours as a direct consequence of abortion.

His statement that "it is an established fact that one or more terminations of pregnancy are liable to result in more women coming in at 26 weeks with ruptured membranes" must be challenged. In the study he quotes¹ to support his view seven out of eight women who had premature rupture of the membranes had previously been aborted of pregnancies of more than 10 weeks' gestation, and it is likely (as the authors comment) that they had been terminated by the traumatic techniques in use at that time. Stallworthy *et al*,² reporting the results of legal abortion in Oxford in the late '60s, commented that Hegar dilators of larger than size 12 were used in the majority of cases and in a fair number they were larger than size 14. Dilatation to such a degree is likely to result in frequent damage to the internal os leading to subsequent cervical incompetence and premature rupture of the membranes. Modern aspiration techniques rarely require the cervix to be dilated to more than Hegar 10, so that subsequent premature labour due to cervical incompetence is unlikely to occur. Johnstone *et al*³ studied the diameter of the internal os six weeks after induced abortion and found values similar to those of the nulliparous cervix if the dilatation at the time of abortion did not exceed 10 mm.

Evidence is often quoted⁴ from a number of papers⁵⁻⁷ in support of Mr Pentecost's view. In one of these, by Jurukovski and Sukarov,⁵ the only evidence presented is a statement, ascribed to Dr Potts, that there is a positive correlation between cervical incompetence and previous abortion.⁵ The second paper⁶ (from Denmark) does not mention damage to the cervix specifically and gives no long-term follow-up results. In the third paper (from Hungary) Klinger⁷ gives no data to support his statement that part of the increase in the prematurity rate in Hungary was due to legal abortion. The increase from 7.0% in 1954, when abortion was prohibited, to 10.5% in 1968 was during a time, as the author admits, when there were major changes in age, parity, and social class among pregnant women in Hungary. Low birth weight is known to correlate strongly with poor living conditions, irrespective of the prevalence of induced abortion.

It seems unlikely that it will ever be possible to carry out the clinical trial that will finally settle the matter because of the difficulty of obtaining matched controls, problems of follow-up, and variable techniques of abortion. However, two large trials^{8,9} in which careful