Inhibition of Lactation by Oestrogens

SIR,—There is clear evidence today that more than 70% of parturients do not propose to breast-feed their infants, and will require inhibition of lactation. In 1963, in a double-blind study,[1] I found that stilboestrol showed a significant superiority to a placebo in inhibiting lactation. No patient was restricted in her fluid intake, nor were the breasts bound. All had had normal pregnancies and spontaneous deliveries.

The stilboestrol was given in an eight-day course, with dosage reduction every second day, a total of 195 mg. being administered. Lactation was inhibited successfully (when assessed on day seven) in 89% of stilboestrol-treated cases, compared with 32% of placebo-treated cases. However, when followed-up for 42 days, permanent inhibition of lactation only occurred in 53% of the stilboestrol-treated group, and 30% required a further course of stilboestrol because of the recurrence of painful lactating breasts. Hodge[2] has shown the superiority of stilboestrol over a placebo, using a dose of 105 mg. stilboestrol over three days, and assessing the patient on the fourth day, and found substantially similar results (stilboestrol-treated cases, 88% successful; placebo-treated cases 32% successful). Stirrat *et al.*,[3] using 105 mg. of stilboestrol over five days, confirmed the "immediate" success rate of stilboestrol when compared with a placebo, but noted that over a 21-day period of follow-up failure (as judged by "painful lactation") occurred in 38% of the stilboestrol-treated patients, and suggested "it may be that a higher level of oestrogen circulating for a longer period would be more effective."

These three studies confirm that inhibition of lactation is best effected by oestrogens, but suggest that shorter courses, in lower dosage, as suggested by Professor T. N. A. Jeffcoate and others (5 October, p. 19), may be inefficient. If the suggested relationship between oestrogen used in the puerperium to inhibit lactation and puerperal thromboembolic disease is substantiated the use of other hormone combinations (such as a mixture of oestradiol valerate and testosterone enanthate) may be advisable in "high-risk" women. Meanwhile it must be stressed that the incidence of thromboembolism is very low, and in the abnormal woman inhibition of lactation using oestrogens is still the treatment of choice.—I am, etc.,

DEREK LLEWELLYN-JONES.
Department of Obstetrics and Gynaecology,
University of Sydney,
Sydney, N.S.W., Australia.

REFERENCES
1 Llewellyn-Jones, D., *Med. J. Malaya*, 1963, **18**, 13.
2 Hodge, C., *Lancet*, 1967, **2**, 286.
3 Stirrat, G. M., Anderson, G. E., and Grant, O., *J. Obstet. Gynaec. Brit. Cwlth*, 1968, **75**, 313.

Rickettsial Endocarditis

SIR,—Your report of four cases of rickettsial endocarditis (5 October, p. 40) prompts us to report two further cases from the north of Scotland.

A man, aged 45 years, had consulted his practitioner for an acute attack of pain in his left leg and calf which was worse on exercise. He had a lump in his groin and it was thought that he had inguinal adenitis. This condition was very slow to resolve. Fourteen weeks later, just prior to admission, he had a similar pain in his left leg and a recrudescence of pain in his right limb. Coincidentally he noticed haematuria for the first time. There was no other relevant medical history.

On admission he looked ill, his right leg was cold and white, and his left ankle swollen. There was a tender swelling in his right femoral triangle. He had hard discrete enlarged glands in his left axilla and his spleen was enlarged to three fingerbreadths below the left costal margin. There was no jaundice. His appetite was poor and he had lost about one stone (6.5 kg.) in weight. Leukaemia was suspected, and blood examinations, *x*-rays, arteriograms, and lymph gland biopsy were undertaken at this stage. Confirmation of the block to his right femoral artery was obtained, but since the diagnosis was still in doubt he was seen by one of us (J. K.). The low-grade fever which had developed, the finger clubbing, together with the detectable cardiac murmurs suggestive of aortic and mitral valve disease, suggested subacute bacterial endocarditis. Four negative blood cultures were followed by the demonstration of *Rickettsia burneti* complement fixation titres 1:1,024 (Phase I) and greater than 1:10,000 (Phase II).

Intensive treatment with tetracycline produced an excellent response, and the patient's condition very markedly improved.

The second case was a man who first came under medical supervision in 1959 at the age of 28 years, after an insurance examination. He was referred to a cardiology clinic, where he was found to have aortic stenosis and incompetence, not at that time giving rise to any disability. He was under observation for the ensuing years and remained well until December 1964, when a check *x*-ray of his chest showed a small apical cavity in his right lung. He was found to be suffering from tuberculosis. Standard treatment was carried out at home and the outcome was satisfactory. He was admitted to hospital as an emergency in August 1968 with a complaint of breathlessness on slight exertion and even at rest—becoming more severe in the few days before admission. Eating had become difficult; his appetite was very poor and he frequently vomited what he had eaten. He had felt very nervous and tense for the previous one to two weeks and suffered from depression.

On admission he was in moderately severe congestive cardiac failure and the heart was markedly enlarged. Loud systolic murmurs were audible at the mitral and aortic area conducted to the neck; there was a loud diastolic murmur present down the left sternal border. It was noted after a few days in hospital that he was running an irregular pyrexia, and further examination showed that he had developed finger clubbing and enlargement of the spleen. He was pale and the skin had a yellowish tinge. Blood count showed a moderate degree of anaemia (Hb 65%); no leucocytosis was apparent. *X*-ray of the chest confirmed an enlarged heart with evidence of pulmonary congestion. There was no evidence of any increased activity in his old tuberculous lesions. In view of the pyrexia, finger clubbing, and splenomegaly the possibility of infective endocarditis arose. Four blood cultures were negative. The complement fixation test for Q fever carried out at this time showed a titre of greater than 1:16,000 (Phase II). Subsequent testing for Phase I antibodies showed these present to a titre of 1:4,096. In the light of this he was treated intensively with tetracycline; his temperature gradually returned to normal and his general condition markedly improved.

Both patients are awaiting transfer to cardiac units. It is hoped to publish fuller details, together with the results of the epidemiological investigations undertaken, at a later date.—We are, etc.,

H. WILLIAMS.
J. KNOX.
W. LANCASTER.
Raigmore Hospital,
Inverness.

SIR,—I was delighted to read the report of the clinicopathological conference on "Four Cases of Rickettsial Endocarditis" (5 October, p. 40) and to see you still honoured Ricketts. But I was sorely disappointed when you and those who assisted in preparing the report later allowed the organism to be called *C. burneti*.

Howard Taylor Ricketts (1870–1910), of Findlay, Ohio, discovered in 1907 that the Rocky Mountain spotted fever is transmitted by the wood-tick (*Dermacentor occidentalis*), and in 1910 (with R. M. Wilder) that Mexican typhus (tabardillo) is transmitted by the body-louse (*Pediculus vestimenti*). This had already been demonstrated for European typhus by Charles Nicolle.[1]

Ricketts died in 1910 from tabardillo, louse-borne typhus. It is idle to speculate what further advances he would have made. Derrick isolated the organism in 1937 and called it *Rickettsia burneti* because Burnet and Freeman had classified it as a rickettsia. This is not to decry Cox's valuable work in isolating Rickettsias in stock holders and slaughterhouse workers, his cultivation of the organism, and the preparation and standardization of rickettsia vaccines. H. R. Cox in papers published in 1938, 1941, and 1948 consistently uses the word "Rickettsial." Let us do likewise and give eponymous immortality to a medical martyr.—I am, etc.,

R. E. SMITH.
Board of Graduate Studies,
Faculty of Medicine,
University of Birmingham.

REFERENCE
1 Garrison, F. H., *History of Medicine*, 4th edn., 1929, Philadelphia.

Sniffing of a Shoe-cleaner

SIR,—A group of teenagers in two schools in this area have been sniffing from handkerchiefs a proprietary brand of liquid cleaner for leather shoes which is retailed in two-ounce (50-ml.) bottles by a well-known chain store, and which is therefore presumably widely available. The preparation consists of a mixture of trichlorethylene, perchlorethylene, and methylene chloride, with smaller quantities of dipropylene glycol and methyl ethyl ketone, and is described as producing on inhalation a feeling of pleasant elation for about 10 minutes, followed by severe headache.

This self-induced narcosis seems likely to be the explanation for cases of hitherto unexplained headache which have been occurring in the affected schools.—I am, etc.,

N. V. HEPPLE.
Public Health Department,
Harrogate, Yorks.

Chronic Lead Intoxication Mimicking Motor Neurone Disease

SIR,—"In many cases the diagnosis of lead poisoning is obvious, in others the possibility may never suggest itself."[1] These words remain as true today, as has been shown in your columns recently (13 January, p. 117, and 2 March, p. 574). The inevitably progressive course and poor prognosis of idiopathic motor neurone disease is