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.,

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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.,

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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.,

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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.,

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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

as well recognized as is its hereditary nature.[2] However, it is not so well known that this condition can be mimicked by chronic lead intoxication.

Recently, Simpson *et al.*[3] described a patient, an "acetylene burner" for more than 20 years, whose symptoms and signs they attributed, after electromyographic and nerve conduction studies, to "myelopathy" due to chronic lead intoxication. Lead poisoning following oxyacetylene torch-cutting of lead-painted steel on ships has been reported.[4] Our patient was involved in such similar work, and presented us with a clinical dilemma made the more interesting because of its medico-legal implications.

A man, aged 35 years, presented in February 1968 with a history that he had been well until March 1967, when increased wear of the toe-tip of his left shoe had been followed by an increase in foot-drop which hindered his walking. During the previous 10 years he had been employed intermittently using an oxyacetylene burner to cut up ship metal painted with red lead. He had not been protected adequately from the fumes produced by the cutting process, and while working in this atmosphere he had experienced hitherto unexplained bouts of colicky abdominal pain together with leg cramps which he had attributed to "the strain of doing the job." Periodically he had changed his occupation to work on building sites until the abdominal pain disappeared, at which time he returned to "cutting-up" until, again, the pains became unbearable.

On examination he had a spastic gait further hindered by a left foot-drop. In addition there was increase in tone of all limbs, hyperreflexia, knee and ankle clonus, and extensor plantar responses. Weakness of left knee flexion was accompanied by weakness of plantar- and dorsiflexion of the left foot. Although fasciculation was widespread, there was no muscle-wasting. Urinary lead excretion on two consecutive 24-hour samples was 136 μg. and 170 μg. respectively (normal range is less than 84 μg. 24-hour urine sample). On 15 February 1968 electromyographic studies were performed. The results were interpreted as "showing changes which were quite consistent with a clinical diagnosis of motor neurone disease, but which were not absolutely diagnostic of this condition."

The patient was given oral penicillamine 300 mg. t.d.s. to aid lead excretion. After eight days a skin rash developed and this therapy was withdrawn. At this time the patient stated he had improved and was once again able to "carry my baby and walk upstairs." He failed to attend the outpatient clinic until August 1968, when physical examination showed that the generalized spasticity remained with both knee and ankle clonus. However, the right plantar response was, and has since remained, flexor, and the only gross weakness was that of eversion of the left foot. This improvement which had taken place in the patient's clinical condition, together with the facts given above, has suggested the diagnosis of chronic lead intoxication mimicking the syndrome of motor neurone disease.

This patient presented with a series of neurological events originally described in cases of motor neurone disease due to chronic lead intoxication more than 60 years ago.[5] It will be interesting to observe the total extent of the recovery in our patient following a further course of lead chelation therapy using Versene (calcium disodium edetate). Unless he has further contacts with lead his condition should, if our diagnosis is correct, at the worst remain the same or, at the best, improve considerably. In our view, this case report not only supports a recent plea that it is still important today to make a detailed assessment of every patient's occupational history to facilitate accurate diagnosis,[6] but it also stresses the importance of seeking an alternative explanation, especially in young people, as to the cause of syndromes which are otherwise uniformly fatal.

We are grateful to Dr. E. T. Baker-Bates for his critical assessment of this case report and for his permission to publish the details.

—We are, etc.,

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G.P. Obstetric Units

SIR,—As correspondence has continued following my letter (14 September, p. 678), I would like to reply briefly to some of the points in the letters you have published. Much of this correspondence could well have been avoided if your readers had confined their criticism to what I actually wrote.

Your leading article (7 September, p. 567) advocated extension of existing general-practitioner maternity units by the provision of small isolated units in "every town, suburb, and large village in the country," and further suggested that these might well be fashioned out of any four- or five-bedroomed house. My letter was critical of this suggestion, and did no more than draw attention to the proved hazards of many small (not all) isolated units, and to the fact that institutional is replacing domiciliary confinement very rapidly. To accuse me of denouncing all general-practitioner units and wishing to exclude general practitioners from obstetrics is both irresponsible and unfair to the College that I represent. Since 1954 the Royal College of Obstetricians and Gynaecologists has issued five major reports on various aspects of the maternity services and those who work in them. In every single one particular attention has been drawn to the important role of the general-practitioner obstetrician, which I need not elaborate here.

If the type of small isolated unit envisaged in your leading article were to be provided widely throughout the country, the adequate consultant cover, which Mr. David Brown so rightly advocates (26 October, p. 252), would be as impossible to achieve as would 100% institutional confinements in consultant units without general-practitioner participation. Surely the answer must lie somewhere between the two extremes of admitting all women to specialist units, and extending the right to practise obstetrics to every general practitioner regardless. There are a number of joint committees deliberating these problems at the present time, but the primary concern must always be not the sectional interests of consultants, general practitioners, or midwives, but the safety of the mother and her baby.—I am, etc.,

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Treatment of Venereal Disease

SIR,—The revival of the controversy in your columns concerning the epidemiological treatment of venereal disease by Dr. F. J. G. Jefferiss (28 September, p. 802), Dr. A. S. Wigfield (12 October, p. 122), and Mr. Ambrose King (p. 122) recalls a somewhat stormy debate on the subject at the Medical Society for the Study of Venereal Diseases as far back as 1953, when Mr. A. J. King and myself were the principal contenders.[1 2] Although then, as now, firmly opposed to indiscriminate treatment without the best available examination and testing at the patient's first visit, in the prevailing climate of opinion with the venereal diseases at a low ebb I was a heavy loser in the show of hands which followed.

Since that time the prevalence of these disorders has more or less doubled, many clinics are now under strain from rising case loads, and there is considerable public and medical concern. Many, if not most, British venereologists and virtually all involved in the subject in the U.S.A. have now come to regard the treatment of contacts of patients with syphilis and gonorrhoea whose initial examination has proved negative as a basic procedure if these diseases are to be controlled. Particularly is this so in the smaller clinics which are not operating every day. Mr. King himself has shifted his ground a little, for he is on record[3] as stating that in trichomoniasis the sexual partner should "always" be treated "even if it proves impossible to find the organism," but illogically regards it as "an undesirable expedient" if the far more serious syphilis and gonorrhoea are concerned.

It is believed that the Ministry of Health is likely shortly to make recommendations aimed at more effective control of these diseases by means of improved methods of case-finding in which the speed of bringing suspected contacts to the clinics is regarded as of first importance. It is hoped that these recommendations will give sufficient weight to the use of epidemiological treatment by venereologists (who can be expected to understand the virtues—and limitations—of the safeguards outlined in Mr. King's letter in relation to local circumstances) as a desirable expedient for the individual patients involved, especially when the follow-up is likely to be poor. Otherwise the expanding band of social workers may well become dispirited if the hardly secured promiscuous sources of gonococcal infections, whose initial smears happen to be negative, are not offered immediate treatment but are allowed to return to the pool of infection until the organism has been finally isolated. And also if syphilitic disease in the secondary contacts is not promptly nipped in the bud in the incubation period.—I am, etc.,

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