Suppressing lactation

Many doctors, but few patients, view breast-feeding with enthusiasm. Only too often the doctor is not asked for advice but simply to supply a means to suppress lactation. Nevertheless, he should still be concerned to choose the right agent: one which will inhibit milk production with the greatest efficiency and the minimum of discomfort and danger. One alternative which is not considered often enough is to choose no specific agent at all. A tight binder, sympathy, and occasional sedatives will carry many women through the initial discomfort of engorged breasts. Without the stimulus of suckling, the high prolactin concentrations at delivery fall to normal within a week and lactation soon peters out. Fluid restriction is unnecessary: it inflicts further discomfort and seldom has much effect on milk secretion.

For a long time the regular standby for suppressing lactation has been oestrogen. Innumerable therapeutic trials have advanced the claims of a great many oestrogenic agents: stilboestrol, ethynyloestradiol, quinestrol, oestradiol valerate, and chlorotrianisene have all been used with success. Curiously enough, antioestrogens such as clomiphene citrate and tamoxifen are also effective in suppressing lactation, while so, too, is testosterone enanthate, perhaps because of its antioestrogenic biological activity. Androgens have justly fallen into disrepute for treatment of women, but one point may be urged in favour of testosterone. Emotional factors loom large in the storms of the puerperium. On the few occasions when the psychological aspect has been taken into account in trials of agents for suppressing lactation testosterone has turned out to have something to recommend it over other agents.[1]

While undoubtedly oestrogens do suppress lactation they have many drawbacks. They can prevent milk production from starting, but they are much less effective at stopping established lactation. Oestrogens affect not only the breasts but many other organs, notably the uterus. They may increase lochial loss or precipitate withdrawal bleeding. Rebound lactation may occur when the treatment is completed: many patients who leave hospital with lactation well under control turn up later at the general practitioner's surgery for a second course of oestrogen. All these are minor problems, however, compared with the risk of venous thrombosis. The connection between oestrogens and thromboembolism is clearly established for both the oral contraceptives and oestrogens given to suppress lactation.[2] This has effectively brought an end to the story of oestrogen treatment for suppressing lactation.

On the heels of need has come a new drug, 2-bromo-α-ergocryptine mesylate, bromocriptine. This is a dopamine agonist which, like the physiological neurotransmitter, stimulates the production of the prolactic inhibiting factor and thus causes a fall in plasma prolactin concentration. There is no doubt about its effectiveness: numerous clinical trials have shown that bromocriptine is at least as effective as oestrogen in suppressing lactation.[3,4] Its side effects are minimal. Postural hypotension, headache, and dizziness may occur, but the most common untoward symptom is nausea. This can be avoided by giving the pills with meals and by building up slowly to the required dose. Bromocriptine has successfully faced the scrutiny of the Medicines Commission and may be regarded as being as safe as a new drug can be.

But that is not all. There is still some cause to restrain the enthusiasm for this new drug, admittedly more in the nature of potential hazards than proved dangers. Bromocriptine affects neurotransmitter activity, and its action may not be limited to the effects of prolactin on the breast. Such a theoretical possibility does not militate against the use of bromocriptine for treating endocrine disorders associated with hyperprolactinaemia. But lactation is a physiological state, not an endocrine disorder. A full course of bromocriptine for the suppression of lactation costs over £5. If every woman who does not wish to breast-feed were given a course of bromocriptine the cost would be formidable. A great many women will get by without severe discomfort on the simple measures already suggested. They ought to be tried first, and powerful drugs such as bromocriptine should be reserved for the occasional case where stronger measures are called for.

[1] Schwartz, D J, *et al*, *Obstetrics and Gynecology*, 1973, **42**, 599.
[2] Daniel, D G, Campbell, H, and Turnbull, A C, *Lancet*, 1967, **2**, 287.
[3] Rolland, R, and Schellekens, L, *Journal of Obstetrics and Gynaecology of the British Commonwealth*, 1973, **80**, 945.
[4] Weinstein, D, Ben-David, M, and Polishuk, W Z, *British Journal of Obstetrics and Gynaecology*, 1976, **83**, 679.

Tourist hepatitis

In these days of extensive travel some of the cases of acute viral hepatitis seen in Britain have almost certainly been acquired elsewhere. Hepatitis is a recognised hazard for "overlanders" who return from India either by bus or hitch-hiking, but it may also be acquired less far afield. Hepatitis type A, which is perhaps the greater risk for the traveller, may be transmitted either directly (through contact with an infected individual) or indirectly, through contaminated drinking water, particularly in areas with a high prevalence of the infection. Poor sanitation increases the risk. The short incubation period (two to six weeks) of hepatitis type A makes it relatively easy to determine when and where the infection occurred. Hepatitis type B, on the other hand, has a longer and more variable incubation period (six weeks to six months), making the source of the disease, unless related to blood transfusion or parenteral drug administration, much more difficult to identify. Travel to areas where acute hepatitis type B is common or where the carriage rate of HBsAg is high may possibly be associated with an increased risk of acquiring this disease through droplet spread (since HBsAg is found in saliva). Other factors, such as sexual behaviour,[1] about which exact information is difficult to obtain, may also be important.

Since 1968, when hepatitis became notifiable in Britain (as infective jaundice), the number of cases notified each year has fallen.[2] Notification may not be a very reliable guide, but the incidence of both hepatitis type A and type B does seem to be relatively low. In Eastern Europe and Russia, where the system of notification is much better and all patients and suspects are said to be subjected to compulsory admission to hospital, a much higher incidence is still found. Hepatitis is probably also more common in Mediterranean countries. Rates of 200 cases per 100 000, five times the probable rate in Britain, have been reported from Israel.[3] There is no notification in Italy, France, and Spain. Notifications may be paradoxically low in areas where hepatitis type A is hyperendemic, such as Taiwan, where infection is acquired early in life and is usually asymptomatic; but visitors to such countries are highly susceptible.

Iwarson and Stenqvist[4] have recently studied the problem of tourist hepatitis in Sweden. They defined it as clinical hepatitis occurring within two months of a visit to a non-Scandinavian country, since in Sweden the prevalence of