If results in smokers' clinics seem fairly straightforward those in settings with less support are more interesting. Less intensive attempts to help people give up smoking achieve lower abstinence rates whatever method is used. Not surprisingly this is reflected in abstinence rates found when nicotine chewing gum is used in conjunction with different levels of support. At least three distinct levels of support can be identified in published reports: intensive support offered by specialised smokers' clinics,^{3-5 7 8 13 15 16} minimal support (some follow up but in a non-specialist setting),¹⁷⁻¹⁹ and little or no support, where the gum is offered as an adjunct to advice, usually from doctors.^{14 19-21} If the results of the published studies in each category are pooled giving totals of 3120, 239, and 1226 patients respectively then a trend emerges. The long term abstinence rate is 27% when nicotine chewing gum is given with intensive, specialised support, 21% with minimal support, and 10% as an adjunct to advice.

A success rate of 10% is lower than that achieved in smokers' clinics but it would be of enormous clinical importance if it was generally applicable to patients in general practice. Does the evidence suggest that nicotine chewing gum has a useful effect when used as an adjunct to advice?

Russell and colleagues showed that, added to simple advice given by a general practitioner, the offer of nicotine chewing gum increased the long term abstinence rate from 4% to 9%.²¹ Furthermore, this result was achieved when half of those offered gum did not even use it. Among those who used more than one box the success rate was an impressive 24%. Another study showed an advantage for nicotine chewing gum compared with placebo at three months when offered with advice by dentists.²² Yet in the British Thoracic Society's hospital clinic based trial in patients with diseases related to smoking neither placebo nor active gum improved results over those with advice.²⁰ The only placebo controlled trial of the gum in general practice was inconclusive: it showed a non-significant advantage for nicotine gum over placebo at six months (10% v 8%), but the results are difficult to interpret because of the small sample size. These figures rule out a large effect but are consistent with the possibility of a small one—which would still be useful.^{23 24}

The overall results from very different kinds of studies are encouraging even if they leave some questions unanswered. "Does the gum work?" is perhaps the wrong question. We should be asking how it works, and to what extent, in different conditions. It certainly has a specific effect, but success rates are lower when it is handed out to unselected smokers without support or follow up. Yet even in general practice it may be helpful to those who actually use it. We know it is most effective when given to selected smokers strongly motivated to stop with detailed instruction, monitoring of progress, and support by experienced therapists. Perhaps some of these conditions may be achieved in general practice. Careful thought is needed on how best to deliver nicotine chewing gum to those who genuinely need it.

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Postpartum mental disturbances and hormone changes

Eighteen times as many women are admitted to mental hospitals in the first month post partum as in each month of pregnancy.^{1 2} For centuries the puerperium has been known to be a most vulnerable period in a woman's emotional life, but the first detailed description of psychotic illness after childbearing appears to have been presented by Louis Marcé in 1858.³ He observed 44 cases, noted the association between psychiatric symptoms and the onset of lactation, and concluded that postpartum psychosis was determined by organic factors and constituted a disease entity. This concept gradually fell into disrepute, and most modern writers regard postpartum psychosis as an affective or schizophrenic disorder.⁴ Fortunately, the condition is relatively rare, but even so one or two of every 1000 women who give birth develop psychosis within six months after delivery.^{1 2}

Far more commonly, relatively mild and transient changes of mood including depression with episodes of weeping develop a few days after delivery. These mood changes affect 50-70% of parturient women⁵⁻⁷; they are usually brief, have a benign course, and have been named the "maternity blues,"⁸ "third day blues,"⁹ or the "transitory syndrome."¹⁰ Since the changes in mood coincide with profound changes in a wide range of hormones and other humoral agents related to pregnancy a causal link has been supposed probable.¹¹

The rate of disappearance of the different substances varies considerably depending on their half life in the blood, and this in turn has two components: a rapid first half life, representing the phase of removal from the blood compartment, and a longer second half life, representing removal from the extravascular compartments. Pregnancy associated

proteins and human placental lactogen disappear within a few days of delivery. Human chorionic gonadotrophin has a prolonged second half life of nine to 37 hours and is detectable in the blood up to two weeks post partum. Several enzymes are cleared slowly, while pregnancy zone proteins such as α fetoprotein and oxytocinase persist in the plasma for several weeks.

Since, however, postpartum mental disorders affect only some parturient women while all of them experience these profound biochemical changes, it seems more likely that an abnormal reaction to one or other of these changes rather than the changes themselves is responsible for the mental disturbances. No convincing correlation of antepartum hormone concentrations with postpartum mental disturbance has been recognised.¹²

The finding that plasma concentrations of β endorphin are raised during pregnancy and labour and fall rapidly after delivery raised the possibility that withdrawal of these endogenous opioids might be a possible cause of "maternity blues."¹³ Newnham and his colleagues have pursued this possibility further and have found a negative correlation between the woman's estimate of her pain in labour and the β endorphin concentrations post partum, suggesting an analgesic role for β endorphins in labour.¹⁴ They also found a positive correlation between β endorphin concentrations at delivery and the woman's attitude to her pregnancy at 36 weeks and a negative correlation between the postpartum "blues score" and the β endorphin concentration at 36 weeks. On the other hand, the blues score did not correlate either with the β endorphin concentration at delivery or 24 hours post partum, nor with its rate of fall in the first 24 hours. These findings do not confirm the withdrawal hypothesis but neither do they refute it, especially as alterations in central opiate activity might well be more relevant than changes in peripheral blood. They found, moreover, no consistent social, psychological, or obstetric correlation with the occurrence of maternity blues, in contrast with what has been claimed by some authors.¹⁵⁻¹⁸

Another study offered support to altered target organ function rather than to change in humoral factors. Metz *et al* found that platelet α_2 adrenoceptor capacity at seven to 10 days post partum was higher in women with maternity blues than in those without.¹⁹ Platelet α_2 adrenoceptor capacity is affected by circulating concentrations of oestrogen and progesterone and by the falls in these concentrations after childbirth; brain α_2 adrenoceptor capacity is enhanced in depressive illness and probably parallels that of platelets.^{20, 21} Thus a failure of α_2 adrenoceptor capacity to fall after delivery from the high concentrations of pregnancy to the lower concentrations of the normal non-pregnant state might be a factor in the causation of maternity blues, and its persistence might lead to the more severe later form of postnatal depression.

Lindström *et al* found that the cerebrospinal fluid of psychotic postpartum women had higher concentrations of opioid receptor active components than did normal lactating women; in four of these psychotic patients the "fraction II" activity was very high and on electrophoresis and high performance liquid chromatography the material migrated as bovine β casomorphin, postulated to have been derived from casein formed during lactation.²² Receptor active material with the same characteristics was also found in the plasma of four psychotic patients but not in a pooled sample from four healthy lactating women. The authors cite other evidence that neuroactive peptides of extraneuronal origin may cross the blood-brain barrier to cause psychiatric

disturbance and suggest that their findings may stimulate the search for peptides derived from outside the central nervous system in the plasma of schizophrenic patients.

Despite the attractiveness of the hypotheses linking hormonal changes with puerperal mental disorders, no useful conclusions—whether prognostic, prophylactic, or therapeutic—have emerged from the older studies. The newer developments, such as the studies of opioid peptide and α_2 adrenoceptor briefly discussed above, open up new perspectives for research into these disorders. We can only hope that these may lead to better understanding and so to a rational basis for preventive and curative treatment.

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Time for a new name for "frozen shoulder"

1984 was the 50th anniversary of the introduction by Codman of the term "frozen shoulder."¹ Perhaps, as Roy and colleagues have suggested, "the term frozen shoulder, which has for too long encouraged many doctors to do as little as possible about this common and distressing condition, should be abolished."² Their view is shared by Neviaser, who has argued that "the misnomer 'frozen shoulder' should be deleted from the medical literature."³

Why is "frozen shoulder" a misnomer? Firstly, "frozen shoulder" is not cold but hot. On histological examination the tissues are infiltrated with inflammatory cells and (as all medical students know) inflammation means calor, as well as dolor and rubor, but never fridgedoor.⁴ Moreover, radioisotope scans show gleaming hot spots at the shoulder.⁶