favoured delayed feeding, largely on the grounds that postponement of feeds till 48 to 72 hours after birth reduced the risk of regurgitation and forestalled the development of oedema. It would be difficult to explain why a difference of 36 to 60 hours or so in the onset of feeding would materially affect the incidence of regurgitation; not much maturation of the nervous system would occur in that time. Beryl Corner[4] suggested that early feeding caused distension of the stomach and embarrassment of the respirations, but not many would agree that early feeding with small quantities of milk would carry much danger of that.

Numerous workers[5-11] showed that early feeding (in the first 24 hours) resulted in a higher level of blood glucose and a lower indirect serum bilirubin than late feeding (at 48 to 72 hours). R. D. G. Creery[12 13] rightly pointed out that early feeding will not prevent all hypoglycaemia, but that it does reduce its incidence and severity. L. Haas[14] advocated the use of milk, rather than dextrose, to prevent hypoglycaemia. B. A. Wharton and B. D. Bower[15] made similar findings but noted a higher mortality in those fed early; but they used large feeds two to three hours after birth, and others have not repeated this observation.

The prevention of hyperbilirubinaemia and of hypoglycaemia is of great importance, because they damage the brain. Hyperbilirubinaemia is mainly due to haemolytic disease or prematurity, but it is aggravated by excessive doses of vitamin K or the administration of sulphonamides or certain other drugs. Hypoglycaemia is specially common in the smaller of twins, in the dysmature (small-for-dates baby), in babies of diabetic mothers, in babies born after maternal toxaemia, in hypothermia (cold injury), and in the respiratory distress syndrome. It seems clear that the severity of hyperbilirubinaemia and of hypoglycaemia can be reduced by early feeding of premature babies; there is no satisfactory evidence against early feeding ; and it seems irrational to starve babies for 48 to 72 hours when they have been receiving continuous nutriment in utero.

Cigarettes and Atheroma

There is some evidence that aortic atheroma is greater in cigarette smokers than in non-smokers[1-3] but that its severity is not related to the patient's consumption of alcohol.[4 5] D. L. Sackett and his colleagues have recently reported a study of these relationships in an investigation of 1,019 patients who died between 1956 and 1964 in a large cancer hospital at Buffalo, New York.[6] All these patients had been thoroughly questioned about their lifetime's consumption of tobacco and alcohol. When they died their aortas were preserved in formalin and subsequently graded in respect of the extent and severity of the atheroma. Using carefully conceived analytical techniques, Sackett and his colleagues compared the grade of the aortic atheroma with the patient's age, sex, and use of tobacco and alcohol. A painstaking statistical approach was particularly important in controlling the factor of age, which confounds any assessment of the grade of atheroma, because the condition is much more severe in the elderly than it is in younger people.

There was a positive association between cigarette smoking and aortic atheroma. The severity of the atheroma increased both with the rate (packets per day) and the duration (number of years) of smoking. Those who had stopped using cigarettes before interview had a level of atheroma intermediate between those of non-smokers and current smokers. But the severity of aortic atheroma was no greater in pipe-smokers and cigar-users than it was in non-smokers. There was no statistically significant increase in the severity of aortic atheroma among non-smokers who took alcohol than in a control group who neither drank nor smoked. Furthermore, the severity of atheroma in those who used both alcohol and cigarettes was not appreciably greater than in those who used cigarettes only. It was therefore deduced that alcohol had little effect on the severity of the lesion. It is noteworthy that women tended to have somewhat more severe lesions than did men.

This investigation is particularly valuable because nearly all the patients died of cancer, a condition in which there is no association with aortic atheroma.[7] The historical details were impeccably recorded, and on all patients who died there was a post-mortem examination. It remains to elucidate the relationship between cigarette smoking and aortic atheroma.

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Depression after Childbirth

Every general psychiatrist regularly sees women referred for emotional and behavioural abnormality in the days or weeks after giving birth. The more severe disturbances are labelled puerperal psychoses, but show the same symptom patterns as other psychoses—endogenous depression, mania, schizophrenia, subacute delirium—and the connexion, if any, with childbirth is very obscure. In some cases at least the onset of psychosis (or anyway its recognition) probably simply coincides with the obstetric event rather than results from it.

Every general practitioner sees a much larger number of puerperal women with some degree of psychiatric disturbance, though he has less training to recognize and analyse this variety of trouble. The practical questions to be answered are four: (1) How often in a series of parturient women shall we see one with abnormal emotional and behavioural reactions ? (2) How will we tell which of these reactions will remit, which will require active treatment, and which will be serious, with risk of suicide or infanticide ? (3) How can we predict during antenatal care which women will have puerperal breakdowns ? (4) What is the treatment, especially the prophylaxis ?

Firm answers to these questions are likely to come only from co-operative studies by general practitioner, obstetrician,