Smoking during Pregnancy

During the last trimester of pregnancy the rate of growth of the foetus depends on a number of factors of which the more important are its sex, the number of previous pregnancies, the size of the mother, and the number of foetuses in the uterus. Some of these variables may have a considerable effect on birth weight. Females at term are on average 5 oz. (140 g.) lighter than males, first-born are 4 oz. (113 g.) lighter than second-born, and babies of short mothers are often a pound (454 g.) or more lighter than those of tall mothers.¹ The average weight of a twin of 38 weeks' gestation is about 1½ lb. (680 g.) less than that of a singleton of the same gestation, and a triplet weighs ¾ lb. (340 g.) less than a twin.² Other influences which retard foetal growth are maternal disorders such as hypertension and pre-eclampsia. Serious defects of the foetus such as anencephalus and mongolism may also be associated with impaired growth.

While most of these variables are not susceptible to control, there is one important cause of retardation of foetal growth—tobacco smoking—which we ought to be able to influence. The first evidence of the effect of tobacco smoke on birth weight came from animal experiments,³ but there have since been a number of human studies all of which have shown that women who smoke during pregnancy have, on average, smaller babies than women who do not smoke. The smallness of these infants cannot be explained by differences of parity or duration of gestation,⁴ or of maternal size, or social class.⁵ These results have recently been confirmed by C. S. Russell, R. Taylor, and C. E. Law⁶ who also showed that differences of age, education, and work done during pregnancy between smokers and non-smokers could not account for the differences in birth weight. It seems to be established beyond all doubt that smoking during pregnancy has a restricting effect on foetal growth.

The extent to which growth is impaired depends on the amount smoked.⁷ On average, smoking causes a depression of birth weight of about 6 oz. (170 g.), but growth is unaffected by smoking before the start of pregnancy,⁸ and if the mother stops smoking early in pregnancy normal foetal growth can be expected.⁹

The mechanism by which smoking retards foetal growth is not fully understood. It has been suggested that non-smokers eat more than smokers and indulge particularly in foods of high sugar content.¹⁰ But it seems that there is no difference between smokers and non-smokers in their weight halfway through pregnancy, and both groups gain equally in weight over the next 10 weeks.¹¹ Furthermore there is some evidence that maternal dietary intake has to be seriously curtailed before foetal growth is impaired.¹²

A more likely explanation, suggested by C. R. Lowe,¹³ is that smoking restricts the blood flow to the placenta and so reduces the quantity of nutrients to the foetus. This view is consistent with what is known about the action of nicotine on the cardiovascular system and with the haemo-
dynamic hypothesis which has been advanced to explain variations in foetal growth rates in experimental animals. It may be asked whether the infants of smoking mothers, because of their smaller size, are less likely to survive and develop normally than the infants of non-smokers. It is well known that small babies in general are at greater risk of disability and death than babies of normal size, but there is some evidence that infants whose low birth weight has been caused by smoking are more likely to survive than infants of the same weight whose mothers did not smoke. Since there is some risk that this statement may be misinterpreted, it should be pointed out that it means only that if a patient has a small baby one would expect the smallness to be due to smoking rather than to some other condition.

It is possible that when contracted pelvis was a greater obstetrical problem than it is today some benefits could have been claimed for cigarette smoking during pregnancy, because, by causing the foetus to be smaller than normal, it reduced the possibility of disproportion. The earlier studies in fact showed that smoking women required surgical induction and forces delivery less frequently than non-smokers, and there was little difference in the perinatal mortality of the two groups. But the 1958 British Perinatal Mortality Survey showed that the rate was 40% higher when mothers smoked during pregnancy, and this was not due to parity or social-class differences between smokers and non-smokers.

T. M. Frazier and colleagues also recorded higher stillbirth and neonatal mortality rates for the offspring of smokers and Russell, Taylor, and Law found that smoking women had a much higher foetal and infant wastage than non-smokers; the combined death rates from abortion, stillbirth, and neonatal death were respectively 79 and 41 per 1,000 pregnancies.

There are many more women smoking now than there were some years ago, and this is reflected in their rising mortality from lung cancer. It seems that the time has come when women should be told frankly that if they smoke they not only put their own lives in jeopardy but, if they continue to do so during pregnancy, also expose their unborn infants to an unnecessary risk.

Neonatal Peritonitis and Exchange Transfusion

Peritonitis is a fairly uncommon condition in the newborn baby. A. G. Birch and colleagues saw 99 cases during 25 years at the Children's Medical Center in Boston and P. P. Rickham saw 17 in three and a half years in Liverpool. About four cases per year were seen, therefore, in two busy neonatal surgical units. Most of these babies had meconium peritonitis, and the usual cause of it is antenatal perforation of the gut above a congenital intestinal obstruction. The clinical picture is that of the obstruction, and the operative treatment and prognosis are not much affected by the presence of meconium in the peritoneum.

Bacterial peritonitis, due generally to a postnatal perforation, is much less common. About half of these cases are due to perforation of a peptic ulcer, as was shown in the excellent review of H. E. Thelander in 1939. The majority of other cases are due to intestinal perforations. Very occasionally perforated appendicitis or Meckel's diverticulum will be seen in the newborn, but most babies with intestinal perforation have an apparently spontaneous lesion of the large, or less often the small, intestine. Perinatal sepsis, trauma, vascular insufficiency, and pressure from faecal masses have all been suggested as possible causes.

It is therefore remarkable that in this issue of the B.M.J. two articles would record 10 examples of this rare and obscure condition following soon after exchange transfusion.


This has only previously been recorded in sporadic cases. Mr. J. J. Corkery and his colleagues, of Sheffield, report four cases at page 345, and Drs. R. L'E. Orme and Sheila M. Eades six cases at page 349. The clinical picture was remarkably consistent. A newborn baby was given one or more exchange transfusions, apparently without technical difficulty. Some hours or days later the baby refused to feed or vomited. Five of the 10 babies passed blood per rectum, and all were found to have abdominal distension. Of the eight children who had an abdominal x-ray three were found to have a pneumoperitoneum and five showed signs of intestinal obstruction.

To the paediatric surgeon these are the essential signs of neonatal peritonitis, of which abdominal distension is the sign which is unflaggingly present. The importance of investigating this sign whenever it is seen cannot be overemphasized. The delay which sometimes occurred before transfer to a surgical unit may have been due to a belief that the signs were those of septic umbilical phlebitis, a rare complication of exchange transfusion, but this is unlikely to produce progressive distention and certainly not pneumoperitoneum.

Nine of the 10 children had a laparotomy, which showed a perforation of the colon, and in the tenth child multiple perforations of the bowel were found post mortem. Some complex surgery was needed, and one of the eight survivors required separate resections of jejunum, ileum, and colon. To have achieved these results in a condition with a high mortality is most creditable. There is certainly nothing to be gained by delaying surgery when this condition is suspected.

What remains unsolved is the reason for this association of colonic perforation and exchange transfusion. It seems reasonably certain that the agent must be the catheter in the umbilical vein, and this might produce septic, haemo-