

time. Similarly, patients with a history of bleeding during early pregnancy are induced at the 38th week. Patients who go more than five days beyond the expected date of delivery are treated by induction, provided their menstrual cycle has been normal and the date of their last menstrual period is not in doubt.

This policy has resulted in a high induction rate. The rate in this department for the years 1954, 1955, and 1956 was 37.9% of the total number of deliveries. In these three years 3,818 patients were delivered and the number of inductions performed was 1,448. The perinatal mortality rate in these cases was 2.8%; this figure is uncorrected, and includes 18 abnormal babies (44.4%). Caesarean section was required in 37 cases, giving an incidence of 2.5% in induced cases. This figure is in marked contrast to that of 14.5% of Mr. Cope and Mr. Pearson. In spite of this difference, the overall rate for caesarean section here is 5.2%, compared with Hammersmith's 5.4%.

Why is there such a large difference? How is it that only 2.5% of our induced cases require caesarean section? The answer to this question is of great importance, because Mr. Cope and Mr. Pearson have concluded that "the risk to the mother of surgical induction of labour is that of caesarean section." Our method of surgical induction differs very little from theirs, except that we always use the technique of low rupture, and we never use castor oil as a preliminary measure, as we believe (*Journal*, December 14, 1957, p. 1438), that it has now taken its rightful place in the obstetrical museum. The main difference is evidently in the use of the oxytocic drip.<sup>1</sup> It is the practice here to use an intravenous oxytocic drip in all cases where labour has not commenced within 48 hours of rupture of the membranes, and recently the time limit has been reduced to 24 hours in cases of toxæmia. The result of this practice is illustrated by reference to the caesarean sections performed on induced cases in 1956; 18 sections were carried out (an incidence of 3.9% for this particular year), but only 3 (16.7%) were because the patient had failed to go into labour. In the same year an oxytocic drip was required in 43 (3.26%) of the induced cases to prevent prolongation of the induction delivery interval. We believe that without the oxytocic drip many of these cases would have come to caesarean section, and that our caesarean section rate in induced cases would then have been nearer to Hammersmith's.

The success of an intravenous oxytocic drip under the circumstances described depends very much on the way in which it is managed and not only upon the dosage used. An example which illustrates this statement is the case of the attendant who desires to discontinue the drip because of maternal distress; provided the contractions are neither excessive nor unduly prolonged, the correct treatment is sedation and not withdrawal of the oxytocic. In conclusion, if surgical induction is indicated, it can be carried out without an increased risk of caesarean section due to failure to initiate labour, provided a proper place is given to the use of intravenous oxytocic infusion.—We are, etc.,

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#### REFERENCE

Lennon, G. G., *Proc. roy. Soc. Med.*, 1957, 50, 793.

### Oesophageal Atresia

SIR,—We congratulate Mr. Christopher Parish and Mr. C. F. A. Cummins on their paper on oesophageal atresia (*Journal*, May 17, p. 1140). This condition has now been a "surgical proposition" long enough for results from different centres to be valuable. We were particularly interested in the material on which the paper is based, as it shows certain differences from our own.

Our records show that 66 cases of oesophageal atresia (using this term to cover atresia with and without fistula, but not including fistula without atresia) have been admitted to the Neonatal Surgical Unit at Alder Hey Children's Hospital during the last five years. 32 cases (48%) were full-term infants, weighing 5½ lb. (2.28 kg.) or more; 34 cases (52%) weighed less than 5½ lb.

Of the 32 Full-term Infants, 20 had no other major congenital abnormality, of whom 16 recovered and 4 died, a recovery

rate of 80%; 12 had one or more additional severe congenital abnormalities, and of these 4 recovered and 8 died, a recovery rate of 33%. The combined recovery rate for the 32 full-term infants was therefore 62.5%.

Of the 34 Infants Under 5½ lb. (2.28 kg.), just under 50% of which had one or more additional abnormalities, 14 (41%) recovered and 20 died. There were 12 infants weighing between 4½ and 5½ lb. (1.83–2.28 kg.), 4 of whom had other abnormality or abnormalities, and of these 8 (66%) recovered and 4 died. Of the 22 infants under 4½ lb. (1.83 kg.), 6 (27%) recovered and 16 died.

The recovery rate for the 66 infants, all of whom were operated upon, was 52%, about the same as for the 17 Cambridge cases and not very different from other published series. Our experience confirms the now well-known fact that multiple abnormalities and prematurity are the two most important factors influencing mortality in oesophageal atresia. Our figures are not very large, but seem to suggest that a baby between 4½ lb. and 5½ lb. (1.83 and 2.28 kg.), has as good a chance of survival as a larger infant. A baby under 4½ lb. (1.83 kg.) has a poor prognosis, especially if any other abnormality is present. The smallest infant to recover weighed 2½ lb. (0.92 kg.).

Cardiac abnormalities were the most commonly associated lesion in our cases, but absent kidneys and pulmonary aphasia also accounted for some deaths. Cases with other alimentary atresias, rectal or duodenal, usually, but not always, recovered when the patient was full term. Like Mr. Parish and Mr. Cummins, we have been impressed by the survival of babies in whom the diagnosis has been made late; two of our babies were over a week old.

Why is the Cambridge material different from ours? Is the birth rate of premature infants with congenital abnormalities really lower than in the Liverpool region, or is our high incidence of small infants due to a longer period of propaganda, resulting in doctors and midwives sending in infants however small and however grossly deformed?

We do not believe that the details of operative technique are really very important. It is the post-operative surgical regime and nursing care which largely determine the survival rate. As the percentage of premature infants with congenital abnormalities including oesophageal atresia seems to be rising year by year, we are particularly interested in the development of special methods of dealing with these tiny infants.—We are, etc.,

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SIR,—I read with great interest the article by Mr. Christopher Parish and Mr. C. F. A. Cummins on oesophageal atresia (*Journal*, May 17, p. 1140) and am in accord with most of their comments. However, I must disagree with their statement that "we have often been struck by the very poor tissue of which the upper pouch is composed." Personal operation experience of 46 babies suffering from oesophageal atresia has shown that the upper pouch is bulbous, with a thick muscular wall and mucous membrane. In contradistinction, the lower segment is usually poorly developed, presumably because it has largely been non-functioning during intrauterine life. Histological examination of the fistulous portion excised at operation has shown that there is, however, a normal, though thin, muscle layer and mucous lining. Most American authors have also commented on the poor tissue of the lower segment—e.g., Haight.<sup>1</sup>

Although my colleagues and I<sup>2</sup> for our earlier cases advised routine "feeding" gastrostomies, this practice has been discontinued and we now introduce a fine polythene tube (1.25 mm. total diameter, 1.0 mm. bore) into the oesophagus at operation. It is important not to use too large a tube, which would certainly occlude the oesophageal lumen at the site of anastomosis (so that the baby could not swallow his saliva) and might invite a pressure necrosis at the suture line.

I feel some caution is indicated with regard to early feeding by mouth. My own practice is to have a "lipiodol"