DISCUSSION
The present endeavour has been based on a study comprising of 80 cases of labour conducted in the department of Obstetrics and Gynaecology, M.L.B. Medical College, Jhansi. An attempt was made to conduct the study on a balanced distribution of cases comprising of almost an identical half (30 cases in control group and 7 effected by lower segment caesarean section), where the delivery was unaugmented by oxytocin and the remaining 43 cases where the delivery was augmented by oxytocin (20 cases each of induced and accelerated labour and 3 cases of failed induction). The primary aim was to substantiate or contradict the widespread speculation, that oxytocin per se augments the incidence of jaundice. Mast et al (1970), was the first worker to have drawn attention to the fact, that induction of labour by oxytocin enhances the incidence of neonatal jaundice. Later on Ghosh and Hudson (1971), subscribed to his findings in his study of 197 newborns, where 100 births were induced by oxytocin drip and the rest served as control.

Davies et al (1972), Calder et al (1974), have like us, conducted their study on 78 and 120 infants respectively, while Beazley and Alderman (1975), carried out a more extensive study on 1353 babies. More recently Leigon et al (1980), studied 84 infants and contradicted the finding of the earlier workers that oxytocin increases the incidence of neonatal jaundice.
Physiological jaundice is of widespread occurrence and according to Behrman (1975), under usual nursery conditions is observed in 60% of term and 80% of premature infants. Neonatal jaundice may also be due to a conglomeration of various other factors viz Rh and ABO incompatibility, neonatal infections extensive ecchymoses, cephalhaematoma, prematurity and low birth weight, asphyxia and various drugs (Behrman, 1975).

Estimation of bilirubin was done in cord blood just after delivery and on the 5th day in each case. Since it was not possible to take blood sample daily from the newborns, bilirubin was estimated on the 5th day, to exclude the possibility of physiological jaundice, in which peak levels of bilirubin are achieved from the 2nd to 4th day (5 to 6 mg%) and decrease below 2 mg% between 5th to 7th day (Behrman, 1975). Ghosh and Hudson (1970) and Davies et al (1973), have estimated serum bilirubin on the 2nd and 5th day after birth. Gould et al (1974) took blood sample on the 1st and 6th day while Beazley and Alderman (1975), estimated it on the 3rd and 6th day. Chew et al (1977) determined serum bilirubin on three days viz. 1st, 3rd and 6th day, while Leigon et al (1980), estimated serum bilirubin in cord blood and subsequently daily till decreasing levels were obtained.

The age and parity of the various study groups (Table-II and Table-III) conforms to the standard procedure for induction and acceleration of labour. Nearly 50% of
our cases belonged to the age group of 21-25 years. Augmented labour was preferred in primigravidas, since they stayed in the hospital for a longer time enabling us to take the blood sample on the 5th day. Augmentation was however avoided in grand multiparas, for fear of rupture of uterus, hence a majority of our cases had parity less than P_4. In the accelerated group 60% of cases belonged to 1st and 2nd para, whereas in the induced group 60% of cases were primigravidas. Fields et al (1959), have reported 0.5% incidence of rupture of uterus with oxytocin, which increases with advancing parity. In view of this Calder et al (1974), have only included primigravidas in their study. Behrman (1975), has reported, that low birth weight babies (premature and small for date babies) are at a greater risk to have physiological jaundice, where the jaundice is severe and more prolonged than that observed in full term, normal weight babies. In view of this, premature deliveries were excluded from our study and the gestational age of all the study groups (Table-IV) was above 37 weeks. This particular precaution has also been observed by various other workers in this field, (Ghosh and Hudson, 1972; Roberts and Weaver, 1974; Boylan, 1976 and Freidman, 1976).

The details of labour in the various study groups (Table-V), demonstrates that premature rupture of membranes was of common occurrence in the augmented group, as compared to unaugmented delivery. Induction was done in
those cases, in which there were no signs of onset of labour, like painful uterine contractions, tightly closed uneffaced cervix, and absence of show. Acceleration was accomplished prior to the administration of oxytocin by low amniotomy in only 10 cases, whereas in the remaining 10 cases, no amniotomy was done. None of our cases were given epidural anaesthesia to avoid the controversy set by Chalmers et al (1975), that epidural anaesthesia along with oxytocin has a detrimental effect on the serum bilirubin levels. Induction of labour was done in 7 cases of postmaturity, 6 cases of leaking, 5 cases of preeclamptic toxaemia and 2 cases of anterior placenta praevia. Care was taken to select only those cases for induction which had a favourable cervix as evidenced by a Bishop's score of more than 7 in all the cases, to rule out cervical factors. The total duration of labour in the augmented group was considerably shorter than control group, since oxytocin stimulates rapid uterine contractions resulting in early labour.

The total dose of oxytocin and the induction delivery interval was calculated in all the cases of augmented labour (Table-VI). A direct positive correlation was observed between the induction delivery interval and the dose of oxytocin, cases of accelerated labour having a shortened induction delivery interval (4.5 ± 2.72 hrs), had lower mean value of oxytocin (382 ± 290.346 m.I.U.), whereas, with a greater induction delivery interval
(10.125 ± 2.98) as seen in the induced group, the corresponding oxytocin doses were 849 ± 335.636 m.I.U. Since spontaneous labour had already set in before acceleration was done by oxytocin, the induction delivery interval (length of labour) was shorter in these cases as compared to those of induced labour.

The induction delivery interval has not been calculated by any worker, though Davies et al (1972), Beazley and Alderman (1975) and Chalmers et al (1975) have however calculated the total dose of oxytocin used for induction and have correlated if with the incidence and severity of neonatal jaundice.

A total of 80 newborns were examined and investigated for evidence of jaundice (Table-VII). All the newborns had an uneventful delivery with an Apgar score of more than 7 (indicating nonstressed status of the fetus at birth), had birth weight more than 2.5 kg and gestational age above 37 weeks. It was our endeavour to exclude mothers having history of jaundice and history of intake of oral pills. Rh and ABO incompatibility, low birth weight babies (premature and small for dates), babies manifesting with infection asphyxia, cephalhaematoma were excluded from the present study and accordingly only full term, normal weight (>2.5 kg) newborns, without blood group incompatibility, asphyxia at birth and not suffering from any infection were included in the study.
Chalmer's et al (1975), was the first worker to have reported, that newborns having Apgar score below 7 at birth had a higher incidence of jaundice of 13.6% with a relative risk for oxytocin patients of 1.3, whereas in those with Apgar score of 8 or over, the incidence of jaundice was 8%, the relative risk of oxytocin patients being 1.8.

In view of this, Leigon et al (1980), excluded all asphyxiated babies and selected only those newborns having apgar score of more than 7 at birth.

CORD BLOOD BILIRUBIN LEVELS:

The mean cord blood bilirubin levels (mg%) in our study were, (Table-VIII), Control group (1.483 ± .43), Accelerated group (1.49 ± .2125), Induced (1.515 ± .2497) and Caesarean group (1.43 ± .258), values being statistically insignificant from each other (P > 0.05). Thus we observed, that cord bilirubin levels remained in the normal range of 1 to 5 mg% (Behrman, 1975), and no risk was evident with augmented, as compared to unaugmented labours.

In the recent past, only two workers have estimated cord bilirubin levels just after birth. Leigon et al (1980), reported that there was no significant difference (P > 0.05) between cord bilirubin levels in stimulated as compared to unstimulated deliveries. Singh et al (1981) also estimated cord blood bilirubin levels in normal deliveries and those augmented by oxytocin and reported mean values (mg%) of 0.9129 ± 0.1732 in normal deliveries.
and 0.883 ± 0.2724 in augmented cases, values in both being statistically insignificant.

**Bilirubin Levels on 5th Day:**

In the control group, serum bilirubin (mg%) had mean value of 2.08 ± 1.269, whereas in the accelerated, induced and caesarean section group the means levels were, 2.310 ± 1.50, 2.895 ± 1.996, and 1.85 ± 0.498 respectively. (Table-VIII). Our findings, therefore reveal that, those newborns who were delivered after induction with oxytocin (20 cases each in accelerated and induced group), had higher mean levels of bilirubin on the 5th day as compared to lower levels of bilirubin observed in the unaugmented groups, though the values were statistically insignificant (P > 0.05). The possible explanation behind the slight increase of serum bilirubin levels in our study in augmented labours, perhaps may be due to some toxic effect of oxytocin on the bilirubin metabolism.

Unlike the statistically insignificant rise of serum bilirubin obtained in our study, Ghosh and Hudson (1972), Davies et al (1973), Calder et al (1974), Roberts and Weaver (1974), Chalmers et al (1975) and (1976) and Chew et al (1977), have reported a significant rise of serum bilirubin in newborns with induced deliveries, as compared to those born by normal spontaneous labour.

Ghosh and Hudson (1972), observed, that both the incidence and severity of jaundice was enhanced with oxytocin stimulated labours. He observed hyperbilirubinemia
(7 12 mg%) in 17.4% of cases and a significantly higher incidence of jaundice (24%) in those babies induced by oxytocin.

The authors have hypothesised, that oxytocin stimulates rapid uterine contractions which reduced placental blood flow and which in turn hampers the fetal hepatic enzymes system leading to hyperbilirubinaemia, Davies et al (1973) and Calder et al (1974) relate the increased incidence and severity of jaundice observed in their study, to the artificial interruption of pregnancy before term which reduces the surge of corticosteroid at term and serve the hepatic enzymes are corticosteroid inducible. There is deficiency of hepatic enzymes leading to hyperbilirubinemia, Chalmers (1975) and (1976), are of the opinion that hyperbilirubinemia observed in their study may perhaps be due to some direct toxic effect of synthetic oxytocin on the bilirubin metabolism.

that it is unlikely that oxytocin per se increases the risk of neonatal jaundice. They are of the opinion that since the half life of oxytocin is just 4 minutes and oxytocin is readily degraded by kidney and liver, it is unlikely that sufficient amounts of oxytocin enters the fetal circulation to interfere with the metabolism of bilirubin. They further state that in the previous studies, where increased bilirubin levels have been observed, it may be due to prematurity, postnatal asphyxia or increased placental transfusion.

One of the most significant finding of our study, was the positive correlation observed between the total dose of oxytocin administered and the subsequent rise of mean serum bilirubin levels in the newborns (Table-IX). It was seen that with increasing doses of oxytocin (m.I.U.) from $\leq 500$, 500-1000 and $\geq 1000$, the corresponding serum bilirubin levels (mg%) were 1.8, 2.48 and 5.275 respectively, values in the last group ( $\geq 1000$ m.I.U.) of oxytocin, being statistically significant from those observed with controls (P $\leq 0.05$). Similarly the mean serum bilirubin values amongst these three groups were found to be statistically different from each other. Like us, a positive correlation between total dose of oxytocin and neonatal jaundice has also been observed by Davies et al (1972) and Beazley and Alderman (1975). Calder et al (1974) and Leigton et al (1980), have however failed to correlate the total dose of oxytocin to the subsequent development of neonatal jaundice.
Another important finding of our study is, that the incidence of neonatal jaundice above 5 mg% was observed in a higher percentage of cases in augmented deliveries, (15% in induced and 10% in accelerated labour), as compared to only 3.3% in the control group, difference a being statistically significant. This finding is easily explained since cases of augmented labour manifesting with jaundice of ≥ 5% mg%, received higher mean total dose of oxytocin.

Like us, Ghosh and Hudson (1971), Calder et al (1974), Roberts and Weaver (1974) and Chalmer et al (1975), have also reported a higher incidence of jaundice ≥ 12 mg% in their studies. The claim of Leigon et al (1980), that since the half life of oxytocin is just 4 minutes, it can not exert a toxic effect on bilirubin metabolism is far from true, because oxytocin when given for prolonged period in higher doses may maintain a steady level in the body to enable it to enter the fetal circulation and interfere with the metabolism of bilirubin.

Since all the factors incriminated in the causation of neonatal jaundice viz low birth weight, gestational age below 37 weeks, Rh and ABO incompatibility, asphixia and infection in the baby were excluded in our study, our findings of slight increase of bilirubin levels in induced deliveries, as well as a significant positive correlation of increasing serum bilirubin levels with increasing
doses of oxytocin, suggests that possibly oxytocin per se has a toxic effect on the bilirubin metabolism. The exact mechanism of action of oxytocin in the aggravation of neonatal jaundice, however, still remains to be elucidated and needs further research.