REVIEW OF LITERATURE
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The procreation of human race shall for-ever remain an enigma and a mystery of the law of nature. A child in the mother's womb emerges in the environment by a hitherto yet unexplained process of labour, whereby a fetus of viable age is expelled from the uterus. This process of labour varies greatly in duration and severity and may be designated as normal or abnormal, according to the presentation and position.

Normal labour or Eutocia is the process of expulsion per vagina of the mature fetus presenting by vertex, followed by after births where the parturition is uncomplicated, spontaneous and not delayed.

The cause or causes of the normal onset of labour remain a subject of speculation and debate. There are many factors mechanical, maternal, extrinsic as well as fetal which contribute to the onset of this extraordinary event.

Csapo (1968), who put forward many theories, detailed that progestrone is the major endocrine controlling factor for the onset of normal labour. It is suggested, that the uterine muscle is inhibited during pregnancy by progestrone, which has an inhibitory effect on the uterine muscle, but at term this progestrone block is removed partly because of decreased synthesis by the placenta, as well as by a rising level of
oestrogens which potentiates uterine contraction as well as sensitizes the uterine muscle to the action of oxytocin. Turnbull (1957) and Klopper and Dennis (1969) have stated that oestrogens are supposed to exert their effect of increasing contractility of the uterus by acting on the lysosomal labilizers as well as improving the myometrial contractile protein (actomyosin); ATP and ATPase.

Prostaglandins and oxytocin are the other hormones incriminated in the onset of normal labour. Karim et al (1970) has found, that at term besides the more widely used PGF_2 prostaglandins, PGE_1 and PGE_2 also seem to have a useful potential in initiating or maintaining labour.

Besides the maternal hormonal effects in the initiation of labour, work done by Liggins (1973) has demonstrated that the fetal pituitary - adrenal activity may also regulate the onset of labour. It has been seen that high fetal cortisol level is implicated in the onset of labour, by acting on the placental enzymes to cause a fall in progesterone production and increase in oestrogens.

Garfield et al (1974), is of the opinion that oestrogen increases the number of gap junctions during late pregnancy, which helps in the onset of labour.

Oxytocin, a posterior pituitary extract which has been extensively used in therapy to induce labour is
also a potent uterine stimulator. O'Brien and Cefalo (1982) have reported that though the maternal endogenous plasma oxytocin is not elevated prior to the onset of labour, the sensitivity of the uterus to stimulation by oxytocin is enhanced, resulting in the onset of labour.

**INDUCTION OF LABOUR:**

Induction of labour means adoption of measures designed to initiate labour any time after 28 weeks of gestation which aims to secure delivery per vias naturals.

Oxytocin was pioneered by Blair Bell in the year 1909 and by Hofbauer in 1911, but it was first used for induction of labour by Watson (1922). Syntocinon, a synthetic preparation of the posterior pituitary extract is used in clinical practice.

Oxytocin is destroyed rapidly by enzymes oxytocinase; hence oral route is useless and for practical purposes, only intravenous method is employed for induction of labour.

Dale (1906) reported the in-vitro oxytocic effect of posterior pituitary extract, while Frankl Hochwart and Frohlich (1909) first reported its effect on the pregnant animal uterus.

Caldeyra Barcia and Posiero (1959) reported that the responsiveness of uterus to oxytocin increases eight fold between 20th and 39th week of pregnancy.
and most of this increased responsiveness occurred during the last 9 weeks. Because of this a slow intravenous infusion of a few units of oxytocin are usually effective in initiating labour at term.

Brazeau (1975), opines that oxytocin stimulates both electrical and contractile activity in uterine smooth muscle and it is unique in eliciting contractions of the fundus that are indistinguishable in amplitude, duration and frequency from those seen in late pregnancy and during spontaneous labour. A very low level of motor activity prevails in the human uterus during the first and second trimesters of pregnancy.

The various routes of administration of syntocinon are intranasal, intramuscular, intravenous and buccal.

METHODS OF INDUCTION:

Theobald et al (1948), innovated the method called 'Physiological Drip' or dilution technique of oxytocin for induction of labour, which is followed even today by us.

This method consists of starting the drip with a physiological or low dose of syntocinon viz. 0.5 I.U. in 540 ml of 5% Dextrose and giving it at a rate of 10 or 20 drops per minute. The drops are then increased every 15 minutes till 40 drops are achieved. In the next bottle 1 I.U. of syntocinon is added and the drip rate is increased gradually till effective uterine contractions occur. Theobald advocates, that not more
than 1500 ml is used in a day and the drip is discontinued in the evening and restarted next morning so that the patient is not disturbed in the night. This method is usually accompanied by low amniotomy and because of this, the patient is given a prophylactic antibiotic if the delivery takes more than 25 hrs after artificial rupture of membranes is done.

Donald (1979), has mentioned another method of induction known as 'Escalation technique'. This method consists of two bottle technique, the dummy solution is started first intravenously and then the syntocinon drip is administered by a Y-connection. The bottle consists of 2 I.U. of syntocinon and it is started at 20 drops per minute, which gives a total dose of 5 milli units/minute. The dose is increased every 15 minutes till 80 drops/minute. In the next bottle 8 I.U. are added and started again at 20 drops/minute and increased gradually till effective uterine contractions with good relaxations are obtained.

**BILIRUBIN METABOLISM**

The major part of bilirubin is derived from the catabolism of haemoglobin present in the senescent red blood cells and this accounts for 80% to 85% of the daily bilirubin production. Crosby (1955) noted that 1 gm Hb yields about 34 gm of bilirubin.

Pullman and Perault (1959) are of the opinion that it is from choleglobin that bilirubin is formed by the
enzyme microsomal hemeoxygenase, which requires O₂ and a cofactor reduced NADPH. Bilirubin is then formed from biliverdin by an enzyme biliverdin reductase.

Bilirubin is bound in the plasma to albumin. Although Vanden Berg and Muller (1916) were the first workers to describe that plasma bilirubin has got 2 types of reactions (direct and indirect), it was not until (1956) that a number of workers simultaneously demonstrated the difference between the chemical nature of the two pigments.

Billing and Lathe (1958) reported that indirect reacting pigment is free bilirubin, whereas the direct reacting bilirubin is chiefly bilirubin diglucuronide and that though the free bilirubin is highly soluble in aqueous solution in the physiologic range, glucuronide conjugate is water soluble and can be excreted by the liver and kidney.

The liver occupies a central role in the metabolism of the bile pigment and three distinct phases are recognised.

Hepatic uptake:

Unconjugated bilirubin, bound to albumin is presented to the liver cell and upon entry and pigment and albumin become dissociated, and the albumin is accepted by certain anionic bindings proteins referred to as Y and Z proteins (Isselbacher, 1974).
**Conjugation:**

Schmid et al (1957), have said that conjugation of the free bilirubin takes place principally in the liver and is dependent on a series of enzymatic steps culminating in the transfer of an active glucuronide moiety from the donor substance uridine diphosphoglucuronic acid (UDPGA), to bilirubin and the reaction is catalysed by an enzyme "Glucuronyl transferase" located primarily in the hepatic parenchymal cells.

**Excretion into bile:**

Zuelzer and Brown (1961), have reported that for excretion of bilirubin into bile, it should be in a conjugated (water soluble) form and further the process of excretion is energy dependent and a rate limiting step in hepatic metabolism of bilirubin.

After its appearance in the intestinal lumen, bilirubin glucuronide may be excreted in the stool or metabolised to urobilinogen and related products. Because of its polarity, conjugated bilirubin is not reabsorbed by the intestinal mucosa.

**NEONATAL JAUNDICE:**

Behrman (1975), is of the opinion that under usual nursery conditions jaundice is observed in the first week of life in approximately 60% of term infants and 80% of premature babies. He has listed various factors for the development of jaundice in the neonate.
1. Factors increasing the load of bilirubin to be metabolized by the liver.

2. Factors which may damage or reduce the activity of the enzymes, (Anoxia, infection, hypothermia etc.).

3. Factors which complete for, or block the enzymes (drugs).

4. Factors leading to absence or decreased amounts of enzyme (genetic defect and prematurity).

**PHYSIOLOGIC JAUNDICE** :

Zuelzer and Brown (1961), Berhman (1975) and Meherban Singh (1979), reported that the aetiology of physiological jaundice is mainly either hematogenous or hepatogenous. There is increased destruction of neonatal RBC's because of increased osmotic fragility and shortened span (90 days) of cord blood erythrocytes. It has been seen that the production of bilirubin is $2^{1/2}$ times more (6 mg/kg / day) as compared to adults and since the mechanism of conjugation and excretion is inefficient because of deficiency of glucuronyl transferase and Y accepting proteins, there is increased incidence of physiological jaundice.

**Clinical Features** :

1. **Time of Appearance** :

   Zuelzer and Brown (1961) and Behrman (1975), report that in the majority of cases physiological jaundice appears on the 2nd to 3rd day, reaches a maximum on 3rd
to 5th day, (5 to 6 mg) and gradually disappears by first week (2 to 3 mg%).

2. **Peak Values:**

As regards the actual plasma levels of bilirubin it is more difficult to set an arbitrary limit to what may be regarded as normal values. Hasia et al (1953) report that the peak values range from 2 to 12 mg% with a mean value of about 7 mg%. Values in excess of 12 mg% and direct reacting bilirubin more than 1 mg% are regarded as abnormal.

**TOXIC CAUSES OF JAUNDICE:**

Diamond (1959) and Lucey (1960) have mentioned, that certain drugs administered to the mother or infant may enhance neonatal jaundice, possibly in some instances by decreasing the conjugation of bilirubin by competing for glucuronide donor, or by directly influencing the conjugating process and in others by increasing haemoglobin degradation. Zuelzer and Brown (1961), have mentioned the effect of water soluble preparations of Vit K and certain enzyme agents (oxidants) which are capable of producing hemolysis and thereby neonatal jaundice. In recent years an association between neonatal jaundice and premature induction of labour, especially drug assisted induction has been increasingly recognised (Mast, 1971; Ghosh and Hudson, 1972; Chalmer, 1975 etc.) so much so that there is some doubt as to how much
hyperbilirubinemia may be regarded as strictly physiological as previously accepted. In view of this following is the review by various workers regarding the relationship of oxytocin used for induction of labour to the development of neonatal jaundice.

Mast et al (1971), were the first workers to derive a correlation between the induction of labour by oxytocin and the subsequent development of neonatal jaundice. They observed that oxytocin enhanced the incidence of neonatal jaundice and the severity of jaundice was directly correlated to the increasing concentration of oxytocin.

Ghosh and Hudson (1972), carried out an extensive study to ascertain the relationship between the use of oxytocin and the development of neonatal hyperbilirubinemia. Their study comprised of 197 newborns, 100 births induced by oxytocin drip, while the remaining 97 newborns were delivered spontaneously without the use of oxytocin. Babies weighing less than 2200 gm, those delivered by lower segment caesarean section, those in which drugs had been used and those babies having infection and dehydration were excluded from their study. The authors reported an incidence of hyperbilirubinemia > 12 mg% in 17.4% cases in which oxytocin had been used as compared to only 6% in those in which delivery occurred spontaneously. Further, the
workers observed that the incidence of jaundice was significantly higher (24%) in those babies induced by oxytocin, than those in which spontaneous labour was accelerated by oxytocin; the incidence being only 9%. Since the peak levels of unconjugated bilirubin were achieved later and were also much higher than those observed with physiological jaundice and since all the other possible factors incriminated for neonatal jaundice were excluded, the authors concluded that perhaps, oxytocin used for promoting labour may have a role in the production of neonatal hyperbilirubinemia. The authors have hypothesised that oxytocin stimulates rapid uterine contractions, which reduces placental blood flow and which in turn hampers the hepatic enzyme system, leading to hyperbilirubinemia.

Davies et al (1973), also tried to correlate the effect of oxytocin on the neonatal serum bilirubin levels. Neonates were divided into three groups, Group A, consisting of 28 babies, where labour was spontaneous and no oxytocin was used, Group B, of 14 babies in which oxytocin was used to expedite the spontaneous onset of labour and Group C, comprising of 36 infants where oxytocin was administered after labour was induced by amniotomy. Only full term normal weight babies, not suffering from Rh incompatibility or infection were included in their study. Serum bilirubin was estimated
on the 2nd and 5th day after birth. The workers observed that on both the days in Group C, the value of serum bilirubin of 6.8 mg% and 7 mg% respectively were significantly higher than both groups A and B and the values were statistically significant ($P \leq 0.05$). Further, although the values of serum bilirubin were higher in group B than group A, the values were not statistically significant. They also observed, that the rise of serum bilirubin levels is directly proportional to the rate of infusion, total dose and duration of administration of oxytocin. Since in Group C (where oxytocin was used to induce labour), the total dose and duration of administration of oxytocin was more than in the other two groups, highest serum bilirubin level were recorded.

Blackburn et al (1973) tried to evaluate the effects on the neonate of prostaglandins, as compared to oxytocin, when used for induction of labour. The parameters assessed in respect to $\text{PGF}_2^\alpha$ were $\text{pH}$, $\text{pCO}_2$, $\text{pO}_2$, base excess and lactate content in the arterial blood and Ht%, haematocrit, blood glucose, serum Na and K and serum bilirubin levels in the neonate. Out of a total of 23 cases, 11 deliveries were conducted after prostaglandin administration and 12 after oxytocin. The authors observed that both prostaglandin and oxytocin had no adverse effect on the newborns with regard to
all the above parameters as well, no rise of serum bilirubin was detected in both the groups.

Calder et al (1974) studied 120 primigravidas which were divided into 4 groups of 30 cases each. Group A, consisted of those induced by amniotomy with oxytocin infusion. Group B, where delivery was induced by amniotomy and administration of PGE₂. Group C, where labour was induced by extramniotic PGE₂. Group D, labour was spontaneous without use of oxytocin or PGE₂. All the deliveries in Group A, B and C were conducted by epidural analgesia and Marcaine administration, while in group D, 14 had epidural analgesia, 14 had a combination of pethidine and promethazine and the rest 2 cases received epidural analgesia along with pethidine. The indications for induction of labour were similar in all the groups and the inducibility rating did not differ much. Maternal history of intake of oral contraceptives and cigarette smoking, as well as feeding history of the infant was taken in each case. Patients with Rh incompatibility were excluded from the study. Serum bilirubin was estimated on the 5th day in each infant.

The authors observed that the serum bilirubin levels in group A (9.6 ± 3.9 mg%) and group B (9.1 ± 4.9 mg%) were both significantly higher than those in group D (6.4 ± 3.9 mg%) with p value of < 0.005 and
\( 0.025 \) respectively. The mean value for group C 
(17.4 ± 4.4 mg%) did not differ significantly from 
group A and B, and neither from Group D, which served 
as controls. Another important observation of their 
study was, that in group A, 14 of the 30 infants had 
serum bilirubin above 10 mg%, while in group B, C and 
D there were 10.9 and 6 babies having levels of more than 
10 mg%. It is thus evident, that the incidence of 
hyperbilirubinemia was much more in group A, where 
oxytocin was used, than group D where delivery occurred 
spontaneously, the difference being statistically 
significant. Further since various factors like birth 
weight, gestational period, use of oral contraceptives, 
cigarette smoking and feeding history of the infant 
were comparable in all the groups, they could not per 
se be considered important for the rise of bilirubin 
in group A and B. Unlike Davies et al (1973), the 
author however could not derive a correlation between 
the total dose of oxytocin and neonatal hyperbilirubin-
emia.

Concluding their study, the authors have sugges-
ted that hyperbilirubinemia associated with oxytocin 
infusion is probably as a result of artificial 
interruption of pregnancy, rather than to a direct effect 
of oxytocin. The workers subscribe to the views of 
Davies et al (1973), that a deficiency of corticoste-
roids with artificial interruption of pregnancy is the
main factor leading to hyperbilirubinemia, since the hepatic enzymes are corticosteroid inducible.

Gould et al (1974), made another prospective study to evaluate the effect of oxytocin infusion, use of oral contraceptives by the mother, epidural anaesthesia and breast feeding on the neonatal serum bilirubin levels. They estimated the serum bilirubin levels in 181 newborns on the first and sixth day of life. The authors observed no significant difference in the neonatal serum bilirubin levels on both the days with the use of oxytocin, as compared to the control. Similarly they failed to derive a correlation between use of oral contraceptives, epidural anaesthesia and breast feeding on the serum bilirubin levels.

Roberts and Weaver (1974), like the studies of Ghosh and Hudson (1972) and Davies et al (1973), have also reported an increased incidence of neonatal jaundice in babies born after oxytocin induced labours. The authors studied 215 births in which labour was spontaneous in 139 deliveries (64.7%), induced in 39 (18.1%) and accelerated by oxytocin in 37 (17.2%). Care was taken to exclude those infants with Rh incompatibility and those weighing less than 2.5 kg. It was observed that out of 215 infants, 73 infants developed jaundice of over 5 mg% (34%). On further analysis, the authors reported that 44 of the jaundiced newborns
(31.7%) belonged to spontaneous labour group, 14 cases
(35.9%) belonged to induced group and 15 were from
accelerated group. Only 20 babies in their study had
jaundice above 12 mg% out of which 10 belonged to the
spontaneous onset group and the rest 10 to induced and
accelerated group. The authors, therefore, concluded
that though the incidence of jaundice was higher in
the induced group, jaundice can not be virtually elim-
inated by reducing the use of oxytocic agent, since
hyperbilirubinemia was also reported in the spontaneous
group.

Thiery et al (1975), have also tried to observe
a correlation between the use of oxytocin and the
development of neonatal jaundice. They studied 74 normal
mothers having a ripe cervix and carrying a healthy
fetus, who were electively induced at term by combining
low amniotomy with oxytocin or prostaglandin F2 or oral
PGE2. Care was taken not to give any drug which could
have an adverse effect on serum bilirubin levels. The
delivery was vaginal in all cases and the Appar score
was normal in each case. Serum bilirubin was determined
at birth from the umbilical vein and on the 3rd day
by peripheral vein. The authors observed, that the
total serum bilirubin levels on the first and third
day in the control group had mean values of 1.58 ±0.6
mg% and 4.95 ±0.36 mg% respectively. Similarly serum
bilirubin values on these days with oxytocin were 1.73 ± 0.11 mg% and 6.17 ± 0.49 mg% and with prostanglandins the values were 1.63 ± 0.11 mg% and 5.34 ± 0.5 mg% respectively. From these findings the authors, unlike the previous studies concluded, that oxytocin or prostaglandin does not have any adverse effect on the serum bilirubin levels of newborns.

Beazley and Alderman (1975), have carried out yet another extensive study to evaluate the relationship between neonatal jaundice and the use of oxytocin for induction of labour. They divided their case material comparing of 1353 babies into 2 groups, first group consisting of 538 babies delivered spontaneously and 149 babies where spontaneous onset was accelerated by oxytocin, and the second group, where labour was induced by oxytocin in 666 babies. In this study, the workers besides observing the mean bilirubin concentrations on the 3rd and 6th day in all the groups, tried to derive a correlation between neonatal hyperbilirubinemia (≥ 12 mg%) to birth weight, total dose of oxytocin and duration of labour. They observed that the mean serum bilirubin concentrations of 6.50 ± 3.20 mg% in babies born after spontaneous labour did not differ significantly from those babies born after accelerated labour or those in which labour was induced, values being statistically insignificant. Further serum
bilirubin levels of > 12 mg% was observed in 8.44% in the spontaneous group with or without the use of oxytocin, and in 8.25% in the group where labour was induced by oxytocin, values in both the groups not being statistically insignificant (P > 0.05). The authors, however, observed a significant correlation between the incidence of neonatal hyperbilirubinemia and the total dose of oxytocin received by the mother to accelerate or induce labour. It was seen that in those babies where serum bilirubin was > 12 mg%, the total dose of oxytocin used was 18.77 ± 19.10 units, while it was only 9.91 ± 17.41 units in those having serum bilirubin less than 12 mg%, values of the total dose of oxytocin being statistically different in the two groups (P ≤ 0.001).

Another important observation of this study was, that the incidence of hyperbilirubinemia increased in direct proportion to the total dose of oxytocin, while only 6.8% babies developed jaundice of > 12 mg% when the dose was 10 units, 18% babies had jaundice > 12 mg% when the dose was 30 units. Unlike the positive correlation of hyperbilirubinemia with the dose of oxytocin, no significant correlation was observed between the birth weight and duration of labour, to the incidence of hyperbilirubinemia in spontaneous or accelerated labour. The authors drew the following conclusion from their study, unlike previous workers, their study was
unable to show a significant difference, between the mean bilirubin concentration of babies, born after spontaneous, or induced labour. However, the mother of babies who developed jaundice of ≥12 mg%, received a significantly high mean total dose of oxytocin. The authors have hypothesised that hyperbilirubinemia in induced labours might be a result of immaturity of the hepatic enzyme system.

Chalmers et al (1975) also studied the effect of oxytocin on the serum bilirubin levels of the newborns. Their study comprised of 17496 deliveries, which were grouped into three categories:

1. Spontaneous onset of labour which served as control.
2. Deliveries induced or accelerated by oxytocin.
3. Deliveries conducted by low amniotomy.

Besides evaluating the relationship of oxytocin to neonatal jaundice, he also studied the correlation of other factors related to the development of neonatal jaundice viz. birth wt. gestational period, apgar score, length of labour and method of infant feeding. Hyperbilirubinemia was labelled when the serum bilirubin levels were more than 10 mg%. The authors observed that in the induced group 12.4% cases developed hyperbilirubinemia, as compared to only 8.1% and 6% cases in the spontaneous onset group and where amniotomy was done, respectively,
values being statistically significant. (P ≤ 0.00001).
Further they observed an inverse relation of birth weight and gestational age to the severity of neonatal jaundice. Babies weighing less than 2500 gms, had the highest incidence of jaundice (24.8%), while the incidence of jaundice in babies weighing 2500 to 3490 gms and those weighing over 3500 gms was 8.9% and 5.7% respectively. The proportion of cases selected for oxytocin administration in their study was fairly constant after 36 weeks of gestation and it was observed that in each gestational period the incidence of jaundice was higher than the corresponding gestational age of the spontaneous onset group.

Further the authors analysed the degree of hypoxia, as estimated by Apgar score, to the incidence of jaundice. They observed that infants with an Apgar score of 7 or less had an incidence of jaundice of 13.6% with a relative risk for oxytocin patients of 1.3, whereas for those with an Apgar score of 8 or over the incidence was 8% and the relative risk being 1.8 \((r^2=45.8, \ P \ 0.001)\). The author further noticed that there was no significant difference in the incidence of jaundice to the length of labour, but the relative risk for the oxytocin group, was from 1.6, for those in labour under 12 hours, to 2 for those in labour from 12-24 hours and 2.6 for > 24 hours, values being statistically significant (P ≤ 0.001).
Their study also revealed, that there was a greater incidence of jaundice in patients who received both oxytocin and epidural anaesthesia than those who did not receive oxytocin. With breast feeding, the authors observed that the relative risk of jaundice with oxytocin administration was high irrespective of whether breast feeding was continued or discontinued by artificial means. In mothers normally lactating they observed that the relative risk of jaundiced infant after oxytocin was 1.4; in mothers where lactation was suppressed by oestogens the relative risk was 1.8 and in those in whom lactation was suppressed by classical measures the relative risk was 2.4 (n=57.2; P ≤ 0.001). The authors thereby concluded that the neonates delivered after induction by oxytocin are at a greater risk for the development of hyperbilirubinemia than those delivered spontaneously, though they admit that the exact etiology and the significance of these changes remain a mystery.

Boylan (1976), designed his study to assess the role of oxytocin to accelerate labour, on the serum bilirubin levels of the newborn. Out of the total 197 cases in their study, 108 were delivered after oxytocin administration and 88 delivered spontaneously. Infants born premature or weighing less than 2500 gm were excluded from their study. He observed that in all, 17 cases (8.6%) developed jaundice, out of which 8 had been
delivered after oxytocin administration, while 9 were delivered spontaneously. On the strength of their observations the authors concluded that infusion of oxytocin during labour does not seem to heighten the incidence of neonatal jaundice.

Chalmers et al (1976), in yet another study to establish the relationship of oxytocin with neonatal jaundice, observed that the incidence of jaundice was highest in the induced group (13%) as compared to the accelerated group (11.3%) and the spontaneous onset group (8.1%), values in the former two groups being statistically significant than the latter. However values in the induced and accelerated group were not statistically different from each other. The authors have ascribed the increased incidence of jaundice in induced labours to some direct toxic effect of synthetic oxytocin.

Freedman et al (1976) besides evaluating the incidence of jaundice in relation to induction by oxytocin like other authors, also tried to derive a correlation between the jaundice and the mode of delivery. Babies with Rh or ABO incompatibility and low birth weight babies were excluded from their study. The authors in their study failed to show a significant difference in the incidence of hyperbilirubinemia ≥ 10 mg% between unstimulated, oxytocin induced and oxytocin augmented
labours. However, the authors observed a significant difference in the incidence of jaundice in traumatic deliveries (assisted breech and midforceps) as compared to normal vaginal deliveries ($P \leq 0.001$) and this difference was irrespective of stimulated or unstimulated deliveries. The authors, ascribed the increased incidence of jaundice with traumatic delivery to focal haemorrhages occurring in the body.

Chew et al (1977), made a comparative study of the association of oxytocin and prostaglandin used for induction of labour with the incidence of neonatal jaundice. Group A comprised of patients induced by low amniotomy and oral prostaglandin, Group B, where labour was induced by amniotomy and oxytocin and Group C, where labour was spontaneous. Serum bilirubin in the baby was determined in the first, third and sixth day of life and serum bilirubin beyond 12 mg% was considered as hyperbilirubinemia. The authors observed that on each of the days of bilirubin determination the serum bilirubin levels in group B were significantly higher than that of group A, ($P \leq 0.001$). The low incidence of jaundice with induction by prostaglandin is attributed to their capacity of promoting steroidogenesis, which as mentioned earlier are responsible for induction of hepatic enzymes.

Leijon et al (1980), in a recent study, besides
evaluating the relationship of oxytocin to neonatal jaundice have also studied the effect of various other factors involved in neonatal jaundice viz. gestational age, placental transfusion, feeding routine, pH, apgar score and erythrocyte volume fractions. In his later study, 84 infants, 43 born after induced labour and 41 after spontaneous labour were included for the study. Immediately after delivery, cord blood was collected for Hb%, pH, hematocrit and serum bilirubin levels. Subsequently the child was followed daily and bilirubin estimation was done daily till decreasing levels were obtained. The workers observed no difference in the mean values of birth weight, gestational age, apgar score, pH and hematocrit in the two groups of babies. Further the authors, unlike some previous workers observed, no significant rise in the serum bilirubin levels in the induced group as compared to the spontaneous onset group (P > 0.05). Also no relationship was observed between total dose of oxytocin and serum bilirubin levels. However, they observed decreased levels of bilirubin in babies weighing > 4 kg and also reported that a short labour (< 150 min) was related to a higher bilirubin levels. The authors have given various reasons to the increase of bilirubin seen with induced deliveries in other studies. Firstly they hypothesise that stronger uterine contractions following
induction increases the frequency of asphyxia in the neonate and hence a rise of bilirubin levels. Since in their study, delivery was monitored by intrauterine pressure measurements and fetal heart rate patterns, pH and Apgar score did not differ in the 2 groups, the authors ruled out the element of asphyxia in their study. Secondly, they lay stress that increased placental transfusion also influenced the serum bilirubin levels, hence due consideration has to be given to the time of clamping the cord, time of giving oxytocic agents and the position of the infant with regard to the uterus after delivery, factors which influence the placental transfusion. In their study the authors had kept all these factors constant in the two groups i.e. clamping of the cord after 60 sec. in each baby, and keeping the baby at the level of vaginal outlet preventing placental transfusion.

From these observations, the authors have concluded, that it is not likely that oxytocin per se increases the risk of neonatal jaundice. They lay emphasis on the fact that since the half life of oxytocin is 4 min. and oxytocin is readily degraded by the kidney and liver, it seems unlikely that sufficient amount of oxytocin enters the fetal circulation to interfere with the metabolism of bilirubin. They are of the opinion, that in studies where increased bilirubin levels has
been observed, it is most likely due to frequency of preterm deliveries, postnatal asphyxia or increased placental transfusion.

Singh and Sinha (1981), have reviewed the relationship of oxytocin to the development of neonatal jaundice. A total of 250 cases of normal, accelerated, induced and LSCS deliveries were included for the present study, out of which only 216 infants fulfilled the criteria for inclusion. Cord blood was collected for Hb%, blood grouping and serum bilirubin and subsequently serum bilirubin was collected on the third and fifth day of birth. The authors observed no significant difference in the serum bilirubin levels in infants born after accelerated or induced labour as compared to normal spontaneous labour on all the three days.