RESULTS

RESULTS

Table I shows the number of normonatraemic and hyponatreamic newborns and those with neonatal jaundice 24 hours after birth. Hyponotraemia (serum sodium $\leq$ 130 m Eq/L) was shown in 1 case in group I (3.3%), 2 cases in group II (6.7%), 8 cases in group III (26.7%) and 13 cases in group IV (43.3%). Neonatal jaundice was detected in 4 cases in group I (13.3%), 5 cases in group II (16.7%), 8 cases in group III (26.7%) and 11 cases in group IV (36.7%). Also the table shows the total number of jaundiced newborns among normonatraemic and hyponatraemic newborns 24 hours after birth, out of 96 normonatraemic newborns 13 cases developed neonatal jaundice while 15 cases developed neonatal jaundice out of 24 hyponatraemic newborns.

The table shows the number of newborns with neonatal jaundice 24 hours after delivery among normonatraemic newborns in the 4 groups (3/29, 3/28, 3/22 and 4/17 in the 4 groups respectively) and those with hyponatraemia (1/1, 2/2, 5/8 and 7/13 in the 4 groups respectively).

Table II shows the clinical data in the 4 groups, there was no significant differences between the 4 groups with respect to maternal age, gestational age, gravidity, birth weight and one minute Apgar score. Table III shows the laboratory findings in the 4 groups.

Table IV shows the correlation between maternal serum and cord serum sodium levels at birth in the 4 groups. There is a highly significant correlation between the maternal serum and cord serum sodium levels of the newborns ($P < 0.01$). The cord serum sodium at birth is found to be directly proportional to maternal serum sodium.

Table V shows the correlation between cord serum bilirubin at birth and 24 hours after birth in each of the 4 groups. Serum bilirubin level is significantly higher 24 hours after birth than immediately after birth ($P < 0.001$) in all groups.

Table VI shows the correlation between serum bilirubin levels 24 hours after birth in the 4 groups. There was no significant difference between groups I and II ($P > 0.05$), I and III ($P > 0.05$), II and III ($P > 0.05$), a siginficant difference between group III and IV ($P < 0.05$) and a highly siginficant difference between groups I and IV, II and IV ($P < 0.01$).

Table VII shows the cord serum sodium level at birth and 24 hours after birth in the 4 groups. There was no significant difference in cord serum sodium level at birth and 24 hours after birth in the 4 groups ($P > 0.05$).

Table VIII shows the correlation between serum sodium levels 24 hours after birth in the 4 groups. The table shows no significant difference neither between group I and groups II and III ($P > 0.05$) nor between group II and III ($P > 0.05$). The levels were significantly elevated in group III than group IV ($P < 0.05$) and highly significantly elevated in group I than group IV ($P < 0.01$) and in group II than group IV ($P < 0.01$).

Table IX shows the correlation between serum bilirubin and serum sodium 24 hours after birth in normonatraemic and hyponatraemic newborns where there is a highly significant correlation ($P < 0.001$) between serum sodium and serum bilirubin 24 hours after birth in the hyponatraemic group. The serum bilirubin is inversely proportional to serum sodium in the hyponatraemic newborns.

Table X shows the comparison between normonatraemic and hyponatraemic newborns (cord levels) as regard serum bilirubin after 24 hours where it shows a highly significant difference between hyponatraemic and normonatraemic newborns as regard serum bilirubin after 24 hours ($P < 0.01$).

Table XI shows the correlation between cord serum sodium at birth and neonatal serum bilirubin 24 hours

after birth in the 4 groups where it shows a significant correlation between cord serum sodium and neonatal serum bilirubin 24 hours after birth in groups II, III and IV ($P < 0.01$).

Table (I): Number of normonatraemic and hyponatraemic newborns and those with neonatal jaundice 24 hours after birth in 4 groups.

Group	Serum sodium		Neonatal jaundice	
	Level	No	Classified	Total
Group I (n = 30)	Normal	29	3	4 (13.3%)
	Hyponatraemic	1 (3.3%)	1	
Group II (n = 30)	Normal	28	3	5 (16.7%)
	Hyponatraemic	2 (6.7%)	2	
Group III (n = 30)	Normal	22	3	8 (26.7%)
	Hyponatraemic	8 (26.7%)	5	
Group IV (n = 30)	Normal	17	4	11 (36.7%)
	Hyponatraemic	13 (43.3%)	7	
Total	Normal	96	13 (13.5%)	28
	Hyponatraemic	24	15 (62.5%)	

Group I : < 500 cc normal saline + 10 units oxytocin

Group II : > 1000 cc normal saline + 10 units oxytocin

Group III : < 500 cc glucose 5% + 10 units oxytocin

Group IV : > 1000 cc glucose 5% + 10 units oxytocin

Hyponatraemia : cord serum sodium level ≤ 130 m Eq/L.

Neonatal Jaundice : serum bilirubin > 5 mg/dl.

Table (II): Clinical data of all groups.

Group	Parameter	Maternal age	Gravidity	Gestational age in weeks	Birth weight in kg	Apgar score at 1 minute
I	Mean S.D. S.E.M	25 5.64 1.03	2.37 1.25 0.23	39.6 0.77 0.14	3.5 0.3 0.05	8.4 0.76 0.14
II	Mean S.D. S.E.M	25.5 4.5 0.82	2.77 1.05 0.19	39.8 0.57 0.1	3.4 0.29 0.05	8.4 0.76 0.14
III	Mean S.D. S.E.M	26.06 5 0.91	2.54 1.04 0.26	39.6 0.76 0.14	3.4 0.3 0.05	8.5 0.68 0.12
IV	Mean S.D. S.E.M	25.6 4.17 0.76	2.9 0.96 0.18	39.6 0.86 0.16	3.35 0.3 0.05	8.5 0.73 0.13

Group I : < 500 cc normal saline + 10 units oxytocin

Group II : > 1000 cc normal saline + 10 units oxytocin

Group III : < 500 cc glucose 5% + 10 units oxytocin

Group IV : > 1000 cc glucose 5% + 10 units oxytocin

Table (III): Laboratory data of the 4 groups.

roup	Parameter	At birth				24 hours after birth				Maternal serum sodium m Eq/L
		Hb % in gm	Reticutocytic count %	Total serum bilirubin gm %	Cord sodium m Eq/L	Hb % in gm	Reticulocytic count %	Total serum bilirubin mg %	Serum sodium m Eq/L	
I	Mean	15.8	0.69	0.85	137	15.3	1.78	3.5	137.4	137.6
	S.D	0.72	0.34	0.18	2.81	0.65	0.79	1.12	2.36	2.6
	S.E.M	0.13	0.06	0.033	0.513	0.12	0.14	0.204	0.431	0.474
II	Mean	16.0	0.89	0.76	137	15.4	2.27	3.9	137.4	138
	S.D	0.71	0.45	0.22	3.15	0.55	1.05	1.16	2.92	3.05
	S.E.M	0.13	0.08	0.04	0.575	0.1	0.19	0.212	0.533	0.557
III	Mean	16.0	0.81	1.13	134.8	15.4	1.96	4.3	135.6	135.8
	S.D	0.71	0.54	0.36	5.23	0.62	1.33	1.67	4.72	4.83
	S.E.M	0.13	0.1	0.066	0.955	0.11	0.24	0.305	0.862	0.882
IV	Mean	16.1	1.43	1.15	131.3	15.4	2.89	5.6	132.2	132.3
	S.D	0.79	0.85	0.34	3.79	0.71	1.32	2.17	3.8	3.76
	S.E.M	0.14	0.16	0.062	0.962	0.13	0.24	0.396	0.694	0.67

Group I : < 500 cc normal saline + 10 units oxytocin

Group II : > 1000 cc normal saline + 10 units oxytocin

Group III : < 500 cc glucose 5% + 10 units oxytocin

Group IV : > 1000 cc glucose 5% + 10 units oxytocin

Table (IV): Correlation between maternal serum and cord serum sodium levels at birth in 4 groups (m Eq/L).

Group	Parameter cases	Mean serum sodium	r	df (n-2)	P
I	Maternal	137.6	0.94	28	<0.01 *
	Neonatol	137			
II	Maternal	138	0.96	28	<0.01 *
	Neonatol	137			
III	Maternal	135.8	0.99	28	<0.01 *
	Neonatol	134.8			
IV	Maternal	132.3	0.98	28	<0.01 *
	Neonatol	131.3			

* Highly significant.

Table (V): Cord serum bilirubin at birth and 24 hours after birth in the 4 groups (mg%).

Group	I		II		III		IV	
Sample	At birth	After 24 hours	At birth	After 24 hours	At birth	After 24 hours	At birth	After 24 hours
Mean	0.85	3.5	0.76	3.9	1.13	4.3	1.15	5.6
S.D.	0.18	1.12	0.22	1.16	0.36	1.67	0.34	2.17
S.E.M	0.033	0.204	0.04	0.212	0.066	0.305	0.062	0.396
df	28		28		28		28	
t	16.91*		14.54*		10.23*		11.13*	
p	<0.001		<0.001		<0.001		<0.001	

* Highly significant.

Group I : < 500 cc normal saline + 10 units oxytocin

Group II : > 1000 cc normal saline + 10 units oxytocin

Group III : < 500 cc glucose 5% + 10 units oxytocin

Group IV : > 1000 cc glucose 5% + 10 units oxytocin

Table (VI): Comparison between serum bilirubin level 24 hours after birth in the 4 groups.

Comparison between group	I	II	I	III	I	IV	II	III	II	IV	III	IV
Mean	3.5	3.9	3.5	4.3	3.5	5.6	3.9	4.3	3.9	5.6	4.3	5.6
S.D.	1.12	1.16	1.12	1.67	1.12	2.17	1.16	1.67	1.16	2.17	1.67	2.17
S.E.M	0.20	0.21	0.20	0.30	0.20	0.40	0.21	0.30	0.21	0.40	0.30	0.40
df	58		58		58		58		58		58	
t	1.37		2.22		4.7		1.08		3.78		2.6	
p	>0.05 *		>0.05 *		<0.01 ***		>0.05 *		<0.01 ***		<0.05 **	

* Insignificant

** Significant

*** Highly significant

Table (VII): Cord serum sodium at birth and 24 hours after birth in the 4 groups (m Eq/L).

Group	I		II		III		IV	
Sample	At birth	After 24 hours	At birth	After 24 hours	At birth	After 24 hours	At birth	After 24 hours
Mean	137	137.4	137	137.4	134.8	135.6	131.3	132.2
S.D.	2.81	2.36	3.15	2.92	5.23	4.72	3.79	3.8
S.E.M	0.513	0.431	0.575	0.533	0.955	0.862	0.692	0.699
df	28		28		28		28	
t	0.6		0.51		0.62		0.92	
p	>0.05 *		>0.05 *		>0.05 *		>0.05 *	

* Insignificant.

Table (VIII): Serum sodium levels 24 hours after birth in the 4 groups.

Comparison between group	I and II		I and III		I and IV		II and III		II and IV		III and IV	
Mean	137.4	137.4	137.4	135.6	137.4	132.2	137.4	135.6	137.4	132.2	135.6	132.2
S.D.	2.36	2.92	2.36	4.72	2.36	3.8	2.92	4.72	2.92	3.8	4.72	3.8
S.E.M	0.431	0.533	0.431	0.861	0.431	0.694	0.533	0.861	0.533	0.694	0.861	0.694
df	58		58		58		58		58		58	
t	0		1.663		5.45		1.59		5.16		3.07	
p	* >0.05		* >0.05		*** <0.01		* >0.05		*** <0.01		** <0.05	

* Insignificant

** Significant

*** Highly significant

Table (IX): Correlation between serum bilirubin and serum sodium 24 hours after birth in normonatraemic and hyponatraemic newborns.

Serum sodium 24 hours after birth	> 130 m Eq/L (n = 96)		≤ 130 m Eq/L (n= 24)	
Parameter	Serum sodium 24 hours after birth	Serum bilirubin 24 hours after birth	Serum sodium 24 hours after birth	Serum bilirubin 24 hours after birth
Mean	136.7	3.99	127.3	6.38
S.D.	2.43	1.24	1.71	2.25
r	-0.27		-0.63	
df	94		22	
p	>0.05 *		<0.001 **	

* Insignificant
** Highly significant

Table (X): Comparison between normonatraemic and hyponatraemic newborns (cord levels) as regard serum bilirubin 24 hours after birth.

Cord serum sodium	> 130 m Eq/L (n = 96)	≤ 130 m Eq/L (n = 24)
Mean	3.99	6.38
S.D.	1.24	2.25
S.E.M	0.127	0.459
df (n=2)	118	
t	5.02*	
p	<0.01	

* Highly significant

DISCUSSION

Neonatal jaundice is a common problem and in about 5% of all babies the serum bilirubin rises above 200 μ mol/L (12 mg/dl) **(Gray, 1988).**

Neonatal jaundice may be physiological or pathological. Pathological jaundice may be due to excessive haemolysis, metabolic disorders, breast milk jaundice, transient familial hyperbilirubinemia, biliary obstruction and infections **(Gray, 1988).**

Excessive haemolysis may be due to Rhesus, ABO system incompatibility or associated with red cell enzyme deficiencies most commonly glucose-6-phosphate dehydrogenase deficiency **(Gray, 1988).**

Metabolic disorders include hypoxia causing decreased hepatic uptake of bilirubin, substances which compete with bilirubin for binding on the Y and Z proteins (steroids, free fatty acids, thyroxine and drugs as chloramphenicol), hypothyroidism, cystic fibrosis and certain inborn errors of metabolism as galactosemia **(Gray, 1988).**

Breast milk jaundice develops in about 1% of infants who are breast fed toward the end of the first week of

life. Biliary obstruction occurs with hepatitis, inspissated bile syndrome or biliary atresia. Infections may be acquired during intrauterine life causing hepatic damage, during delivery or in the early neonatal period **(Gray, 1988).**

The great concern over hyperbilirubinemia in the newborn is its association with kernicterus which occurs with greater frequency in prematures **(Cunningham et al., 1989).**

Sims and Neligan (1975), reported an increase from 2.2 to 5.5% in the proportion of infants with bilirubin concentration of 15 mg% or greater between 1972 and 1973.

Ghosh and Hudson (1972), Oski (1975), Chew and Swann (1977), D'souza et al. (1979) and Hamad et al. (1985), reported an increased incidence of neonatal hyperbilirubinemia after oxytocin induced or augmented labors. However **Friedman and Sachtleben (1976) and Boylan (1976),** failed to domenstrate this relationship.

Ghosh and Hudson (1972), found a bilirubin level of 12 mg % or more in 6% of neonates delivered without oxytocin infusion, 9% of neonates delivered after oxytocin augmentation of labor and in 24% of neonates delivered after oxytocin induction of labor. They suggested that

oxytocin infusion may cause reduction of uteroplacental circulation that may predispose to a minimal fetal hypoxia not sufficient to cause fetal distress but could disturb the hepatic enzyme system necessary for conjugation of bilirubin in the newborn.

Oski (1975), reported that 12.4% of neonates born after oxytocin administration become highly jaundiced, 8% after spontaneous delivery and 6% after amniotomy. Risk of jaundice developing in infants born after oxytocin administration was 1.6 times greater than that of the infants in the other 2 groups ($P < 0.000001$). Oski suggested that oxytocin infusion causes stronger uterine contractions with increased red blood cell mass of the infant with more bilirubin production due to destruction of normal red blood cells. Also he suggested that oxytocin may interfere with normal hepatic maturation process.

Beazley and Alderman (1975), reported that the incidence of unexplained neonatal hyperbilirubinemia after spontaneous labor was 6.3%. They suggested that the hyperbilirubinemia induced by amniotomy and simultaneous oxytocin infusion may reflect immaturity of hepatic enzyme system. They found a highly significant association ($P < 0.001$) between the mean total dose of oxytocin used for induction and the incidence of neonatal hyperbilirubinemia

and that the incidence of hyperbilirubinemia increased sharply when the total dose of oxytocin exceeded 20 units as it did in 12% of induced labors.

Chew and Swann (1977), found that 15 out of 99 infants delivered by oxytocin induction developed serum bilirubin concentration greater than 12 mg% compared with one infant out of 54 delivered without oxytocin infusion. They found no significant difference in incidence between the high dose (10 units at 60 drops / minute) and low dose (4 units at 40 drops/minute) regimens of oxytocin infusion.

D'souza et al. (1979), reported that elevated cord serum bilirubin levels, probably enhanced by breakdown of fetal red cells may be oxytocin dose dependent. **Hamad et al. (1985),** reported that hyponatraemia and hypo-osmolality associated with oxytocin infusion play a major role in the development of higher bilirubin levels.

Singhi et al. (1984), reported that induction or augmentation of labors was associated with about three folds increase in the incidence of neonatal jaundice (serum bilirubin > 5mg/dl) in the first three days of neonatal life. Moreover **Signhi and Singh (1979),** reported elevated bilirubin level at postnatal age of 72 $\pm$ 12 hours in the neonates whose mothers received oxytocin infusion during labor compared to control group. They implicated

the concomitant administration of large quantities of electrolyte free-dextrose solution as a contributory factor in the mechanism of neonatal jaundice.

Johnson et al. (1984), reported no association between oxytocin administration and neonatal jaundice due to smaller amounts of oxytocin infused and suggested a dose dependent effect of oxytocin. They showed that neonatal hyperbilirubinemia associated with oxytocin infusion is a multifactorial problem resulting from the interaction of the oxytocin, the type and amount of intravenous fluids and the length of gestation.

Boylan (1976), reported that the infusion of 10 units oxytocin during labor had no significant effect on mean bilirubin levels at the fourth day. Any increase in the incidence of neonatal jaundice associated with oxytocin infusion is likely to be due to lower mean gestational age.

Friedman and Sachtleben (1976), found no significant differences in the incidence of bilirubinemia exceeding 10 mg/dl when labor was unstimulated, oxytocin induced or oxytocin augmented.

In the present work, the oxytocin dose was constant in the 4 groups (10 units). There were no differences as

regard maternal age, parity, gestational age, birth weight or one minute Apgar score among the 4 groups. However the incidence of neonatal jaundice was 13.3%, 16.7%, 26.7% and 36.7% in group I, II, III and IV respectively (table I). As regard serum bilirubin 24 hours after birth table VI shows no significant difference between groups I and II, I and III or II and III ($P > 0.05$), a significant difference between group III and group IV ($P < 0.05$) and a highly significant difference between groups I and IV and II and IV ($P < 0.01$). This suggests that the increased incidence of neonatal jaundice is more likely to be due to the use of salt free solutions (glucose 5%) during labor when their volume exceed one litre and not simply to oxytocin itself.

Morgan et al. (1977) and Schwartz and Jones (1978), reported that oxytocin by virtue of its antidiuretic effect results in expansion of maternal extracellular fluid, hyponatraemia and hypo-osmolality. This causes fetal hyponatraemia and hypo-osmolality. As a result red blood cells swell and become osmotically more fragile.

Dahlenburg et al. (1980), Tarnow-Mordi et al. (1981) and Singhi et al. (1985), reported that intravenous infusion of salt free solutions during induced or augmented labors caused maternal and neonatal hyponatraemia.

Dahlenburg et al. (1980), found that the mean neonatal serum sodium levels of mothers received intravenous glucose 5% were significantly lower than in babies whose mothers received no fluid ($P < 0.001$). They found that 29 newborns out of 106 whose mothers had received intravenous glucose 5% during labor developed hyponatraemia. The mothers of 14 out of the 29 hyponatraemic newborns received glucose 5% only (14/58) and 15 received glucose 5% plus oxytocin (15/48).

Tarnow-Mordi et al. (1981), reported that out of 95 mothers who received glucose 5%, 31 had infants with serum sodium levels of 130 m Eq/L or less. They found a highly significant inverse relationship between cord plasma sodium concentration and amount of fluid administration suggesting that hyponatraemia was due to intravenous treatment with predominantly sodium free solutions. They claimed that oxytocin in the usual dose has no effect on maternal and neonatal serum sodium levels.

Singhi et al. (1985), reported that the mean cord sodium levels were significantly lower in the oxytocin and glucose groups than in the control group (no oxytocin or glucose). Hyponatraemia developed in 47% and 30% of the infants in the oxytocin and glucose groups respectively, in contrast to only 5.8% of the infants in the control

group. They also found a significant negative linear correlation between serum sodium and the dose of oxytocin ($P < 0.001$) and log of the volume of glucose infused.

In the present work hyponatraemia developed in 3.3%, 6.7%, 26.7% and 43.3% in groups I, II, III and IV respectively (table I). As regards neonatal serum level of sodium table VIII shows no significant difference ($P > 0.05$) between groups I and II, I and III, II and III, a significant difference ($P < 0.05$) between group III and IV and a highly significant difference ($P < 0.01$) between groups I and IV and II and IV. This agrees with **Singhi et al. (1985)**, where the serum sodium is found to be inversely proportional to the volume of glucose 5% infused during labor. As neonatal hyponatraemia developed in groups I and II (one and 2 cases respectively), this may be attributed to antidiuretic effect of oxytocin. This agrees with **Schwartz and Jones (1978)**.

Spencer et al. (1981), reported that intravenous electrolyte free solutions significantly depress maternal serum sodium levels and that in combination with oxytocin caused more depression of serum sodium (mean volume of glucose 5% was 644-993). They found a significant correlation between maternal and cord serum sodium levels ($P < 0.01$).

In the present work a highly significant correlation ($P < 0.01$) is found between maternal serum and cord serum sodium levels in the 4 groups (table IV), this agrees with **Spencer et al. (1981).**

Schwartz and Jones (1978), singhi and Singh (1979), Johnson et al. (1984), Singhi et al. (1985) and D'souza et al. (1986), suggested that the neonatal hyponatraemia may be a factor in the pathogenesis of neonatal jaundice.

singhi and Singh (1979), reported a significant negative correlation between cord serum sodium levels and serum bilirubin levels 72 hours after delivery.

Singhi et al. (1985), reported that jaundice was developed in 54% of hyponatraemic versus 21% of normonatraemic infants ($P < 0.001$).

The present work shows that neonatal jaundice developed in 62.5% of the hyponatraemic compared with only 13.5% of the normonatraemic infants (table I).

D'souza et al. (1986), reported no difference between hyponatraemic and normonatraemic infants as regard cord serum bilirubin at birth. However 46% of hyponatraemic infants developed neonatal jaundice later on compared with 13% of normonatraemic infants after labor induction ($P < 0.01$).

The present work shows a highly significant difference between hyponatraemic and normonatraemic groups as regard serum bilirubin 24 hours after birth (P <0.01) (table X).

The present work shows a highly significant negative correlation between serum sodium and serum bilirubin 24 hours after birth in the hyponatraemic group (P <0.001) (table IX). The same finding was reported by **Singhi and Singh (1979)**, where they found a significant negative correlation between cord serum sodium level and serum bilirubin levels at 72 hours of age.

Johnson et al. (1984), reported that in 73 newborns whose mothers received oxytocin in 1-3 litres of intravenous isotonic solutions for sodium (5% glucose-Ringer's lactate), no significant differences were found between oxytocin and control group as regard serum sodium levels at birth and serum bilirubin 2 days later.

In the present work the neonatal serum sodium levels were significantly lowered in group IV than the other 3 groups (P <0.01) (table VIII), where as no significant difference was found between groups I and II, group I and III or group II and III. This shows that significant changes occur in neonatal serum sodium levels when the volume of intravenous salt free solutions (glucose 5%)