DISCUSSION
The present study was conducted to observe the effect of oral versus parenteral vitamin K intake on the prothrombin time of newborn babies. The study was conducted on 51 full term babies (gestational age more than 37 weeks) weighing more than 2 kg and who were born after uncomplicated vaginal delivery. All the babies had normal APGAR score at the time of birth (one minute APGAR score).

In the past, many studies have been conducted to evaluate the role of vitamin K to prevent the haemorrhagic disorder in the newborn infants but most of these were conducted to evaluate the efficacy of parenteral vitamin K.

Mean activity of vitamin K dependent coagulation factors in the newborn is about 50% of adult values. This falls to half to one third of its initial activity by the second or third day especially in breast fed babies (Oski and Haiman, 1972). Medical literature contains numerous challenges to the recommendation that all newborn babies receive prophylactic vitamin K at birth. These workers have suggested that defective coagulation in the newborns was not because of vitamin K deficiency, but owing to the liver immaturity (Van Doorn et al and Gobel et al, 1977 and Malis et al, 1980). Van Doorn et al all failed to find vitamin K deficiency in cord blood.
Malis et al (1980) studied 24 cord blood samples and found no evidence of vitamin K deficiency after he had done several different assays. Gobel et al (1977) studied 154 healthy infants at 72-94 hours of age who had not received vitamin K at birth and they did not find depressed prothrombin level in any infant. All the infants in Gobel's series had received feeding during the first 24 hours of age and none was exclusively breast fed. Weight of evidence today, however, does show that vitamin K deficiency is infant responsible for defective coagulation (Corrigan and Kryo, 1980). Yoshioka et al (1982) found depressed prothrombin levels in 6 out of 8 term infants at 3 days of age.

The present study was planned to evaluate the role of oral water soluble vitamin K in the haemorrhagic disease of newborn babies. Since a water soluble preparation of vitamin K$_1$ was not available, the synthetic water soluble vitamin K analogue (Menadione sodium disulphite) was used instead, which is the same form of drug that is being used by parenteral route. This form of vitamin K has a possible advantage that it is water soluble and is easily absorbed, even in the absence of bile salts (Shearer et al, 1974). In moderate doses water soluble vitamin K is quite safe but large doses may cause, hemolysis and jaundice (Aballi et al, 1962).

In the present study, all the 51 cases selected were absolutely breast fed. Breast feeding has been
implicated as a necessary factor in the pathogenesis of haemorrhagic disease of the newborn (Sutherland et al, 1967). Vitamin K is approximately four times more concentrated in cow's milk than in breast milk (Dam et al, 1942).

All the 51 cases were randomly allotted to four different groups viz., A, B, C and D. Parenteral 1 mg vitamin K was given to the cases of study group A; 0.5 mg parenteral vitamin K was administered to study group B; oral 1 mg vitamin K was given to the cases of study group C while no vitamin K was given to the cases of study group D. Vitamin K was administered within 2 hours after birth. O'Connor and Addiego (1986) studied 41 full term infants and arranged them in three groups A, B and C. No vitamin K was given to the cases of study group A, 2 mg water soluble vitamin K₁ (Phytonadione) was given to the cases of study group B and 1 mg vitamin K₁ was given to the cases of study group C. Vitamin K was given within 2 hours after birth. In another study by Sen et al (1989), 120 full term infants who were delivered by uncomplicated vaginal delivery and were arranged in four different groups A, B, C and D. Cases of study group A were given 1 mg vitamin K (Menadione sodium bisulphite) intramuscularly, cases of group B were given 0.5 mg vitamin K (Menadione sodium bisulphite) intramuscularly, cases of study group C were given 1 mg vitamin K (Menadione sodium bisulphite) orally while no vitamin K was given to the cases of
group D. Vitamin K was administered within 2 hours after birth.

In the present study mean birth weight of babies in the four study groups A, B, C and D was 2.72±0.18, 2.85±0.19, 2.62±0.19, and 2.74±0.22 kg respectively. Birth weight differences among the four study groups were not statistically significant.

In the present study the sex distribution (M : F) in four study groups A, B, C and D was 1 : 1, 3 : 1, 1 : 1, and 1 : 1.1 respectively.

In the present study the mean ages at which the blood samples for prothrombin time were collected in the four study groups A, B, C and D were 49.13±13.03, 48.03±15.13, 48±14.14 and 48 ±14.14 hours respectively. The difference between mean age was not statistically significant. In a study conducted by Sen et al (1989) the mean ages at which the sample for prothrombin time were collected in the four study groups A, B, C and D were 48±13.14, 49.33±13.23, 47.9±13.4 and 47.27±10.66 hours respectively. Study done by O'Connor and Addeigo (1986) does not gave any table to depict mean ages of collecting blood samples but the authors have mentioned that samples were collected on the 3 day of birth.

The routine mode of vitamin K administration to the newborn babies has been the injection of 1 mg
of vitamin K intramuscularly. In the present study, vitamin K was given intramuscularly as well as orally. None of the cases who received oral vitamin K had hemolysis and/or jaundice, none was observed to have vomiting. Sen et al (1989) used the synthetic analogue (Menadione sodium bisulphite) of vitamin K orally as well as parenterally while O'Connor and Addiego (1986) used the water soluble vitamin K₁ both orally and intramuscularly.

The present study showed that the prothrombin time (which is a good measure of blood coagulation factors II, VII, IX and X) of babies in group A, B, and C (19.24±3.32, 19.17±3.14 and 20.13±3.31 seconds respectively) was almost identical, showing that 1 mg of vitamin K orally was just as effective as 1 mg or 0.5 mg vitamin K given intramuscularly. It was also seen that group D babies who did not receive vitamin K had a marked prolongation of prothrombin time (34.10±4.02 seconds). The prothrombin time of group D babies was significantly prolonged (p <0.001) as compared to prothrombin time observed in group A, B and C babies. On an average prothrombin time of group D babies was prolonged 2.2 times as compared to other groups who had received vitamin K₁ either orally or parenterally. Thus group D infants who had not received any vitamin K were at risk to develop hemorrhagic disease.
Sen et al (1980) reported prothrombin time values in study group A (who received 1 mg vitamin K intramuscularly) 17.1±2.66, in group B (who received 0.5 mg vitamin K intramuscularly) 17.2±4.42, and in group C (who received 1 mg vitamin K orally) 17.0±2.36 seconds respectively. These values were almost identical and differed significantly (p < 0.001) from that of group D (who did not receive vitamin K at all) being was 33.1±12.20 seconds). These findings are almost similar to those observed in the present study.

O'Connor and Addiego (1986) reported the following prothrombin time values in their study groups A, B and C: Prothrombin time values in study group B (who received 2 mg water soluble vitamin K, orally) - 9.83±0.56 seconds; Prothrombin time in study group C (who received 1 mg vitamin K intramuscularly) - 10.33±1.20 seconds. Thus the prothrombin time values in study group B and C were almost identical and difference was not statistically significant. Prothrombin time in study group A (who did not receive vitamin K), the values were raised, being 12.33±3.42 seconds. The prolonged time in group A cases was statistically significant (p < 0.01) when compared to group B and group C babies.