DISCUSSION

The total number of suspected malarial patients admitted to the medical wards during this study period were 330. Out of these 186 (56.36%) were QBC positive cases, 36 could not be followed up as they were discharged very soon or left hospital against medical advice. The final case material thus consisted of 150 cases.

Of the 150 cases 92 (61.33%) were males and 58 (38.66%) females. Maximum 27.13% male and 46.55% female patients were in the age group of 20-30 years.

On the basis of QBC test 88 (58.66%) were positive for P. Falciparum and 62 (41.34%) for P. vivax. Improvement rate in P. falciparum were 82.95% while in P. vivax 90.32%.

All the 150 patients (100%) presented with fever. Fever is the hallmark of malarial infection. The basis of malarial fever remain obscure. The direct temporal association between schizont rupture and the febrile paroxysm has suggested that parasite might liberate pyrogen but none has been identified. It has been postulated that endogenous pyrogen is secreated by the tissue macrophages which ingest erythrocyte and parasite debris, released at the time of schizont rupture. Tumour necrosis factor/cachectin as endogenous pyrogen has been detected in sera of patients with malaria but its role in the pathogenesis of fever in malaria remains uncertain. Headache during the febrile paroxysm was complained by many patients and it was considered it to be

(46.)
an accomplishment of fever.

As a whole the incidence of cerebral malaria was 22%. Maximum number of male and female were in the age group of 20-30 years. The mortality rate in cerebral malaria was 24.24%. CSF examination showed no abnormality except raised pressure in 36.36% cases. The rigidity was also seen in 12% cases.

Waiz et al (1940) analysed 55 cases of cerebral malaria. In that study mortality was 11% and in CSF examination no abnormality was detected except raised pressure in 7 (12.65%) cases.

Looareesuwan (1992), studied cerebral malaria patients. He also observed mortality rate 10.40%.

Pasloske et al (1994) stated that acute cerebral malaria appears to result from excessive adherence of infected cells in cerebral vessels coupled with occlusion of cerebral flow in microvessels by infected cell response. According to them, several endothelial cell proteins have been identified as potential receptors for infected erythrocyte adherence to vascular endothelium including thrombospondin CD 36, IC Am-1, VCAM-1. The receptor on infected erythrocyte that mediate adhesion to endothelial cells is PFEMP-1.

Wright et al (1993) stated that in cerebral malaria, CSF leucocyte count remain unaltered. They added cerebral malaria in differential diagnosis of meningitis.
Chaudhary et al (1992) noted extrapyramidal syndrome in cerebral malaria. According to them localized signs in cerebral malaria are due to diffuse involvement of brain parenchyma by malaria parasite leading to cerebral oedema. None of the patient had any evidence of meningeal irritation.

Unconsciousness and convulsion, altered behaviour were noticed in 20% and 40% cases respectively. 2 case (1.5%) have presented as hemiplegia. Deb et al (1992) described unconsciousness and confusion, mental conclusion and abnormal behaviour and hemiplegia in 28.57%, 17.14% and 2.86% of the malarial cases. Hammann et al (1993) described the various neurological complication of malaria due to disseminated vasculomyelinopathic disorder.

Other important signs and symptoms noted were Splenomegaly (26%), Hepatosplenomegaly (6.6%), Diarrhoea 10.66%, Vomiting 24.66%, Shock 3.33% and Oliguria/anuria in 8%.

Anaemia was observed in 60% of the total cases. 89.65% females and 42.39% males had anaemia. We attributed this higher incidence of anaemia in females due to the presence of associated factors which are menstrual blood loss, pregnancy and low food intake. Sharma et al (1992) observed it in 100% cases of cerebral malaria. Gorski et al (1994) reported anaemia in most of the malarial cases and Niazi et al (1995), reported this in 49.27% cases of malaria. The findings of present study correlates well with most of the previous studies.

Leukopenia was observed in 9.67%, 7.95% and leucocytosis 6.45%, 11.36% cases in P. vivax and P. Falciparum respectively. As a whole 8.66% cases showed leucopenia and 9.33% showed leucocytosis. Mortelo et al (1969) observed leucopenia in 12% and leucocytosis in 5% of cases with vivax malaria. Sharma et al (1992) observed leucopenia in 6.7% and leucocytosis in 13.3% of cases with P. falciparum malaria. The results of present study is consistent with previous studies. At day5 leucopenia and leucocytosis were 5.47%, 10.94% respectively. Reily states that the leucocyte response does not appear to be suppressed by chemotherapy even in those individual who were leucopenic prior to institution of therapy, since most showed an increase to normal levels while on specific therapy.

Thrombocytopenia was observed in 31.33% at day1 and 16.66% at day5. In vivax it was 16.12% and in falciparum 42.04% respectively at the time of admission. Vreeken et al (1978), observed
thrombocytopenia in all his patients. Deb et al (1992) observed thrombocytopenia in 15.7% cases. Thus the incidence of thrombocytopenia has a varied range. In this study at day 5 thrombocytopenia was present in 16.66% patients. Thus reveals that the increase in platelet count is a slow process.

Clinically jaundice was found in 14.66% cases while raised bilirubin levels were present in 27.33% cases. The incidence of jaundice in P. falciparum was 32.94%. 39.39% cases of cerebral malaria also had jaundice.

Philips et al (1986) observed jaundice in 41.42% of patients with cerebral malaria and 4.35% of patients with uncomplicated falciparum malaria. Waiz et al (1990) reported jaundice in 34.54% of patients with cerebral malaria. Jaundice was also observed in 20% by Mishra et al (1992) and 57% by Deb et al (1992).

Serum bilirubin was raised in 27.33% and SGPT in 10% cases at the time of presentation. Bilirubin was raised in 19.34% in P. vivax and 32.94% in P. falciparum malaria. While SGPT 4.84% and 13.63% in P. vivax and P. falciparum respectively. Mishra et al (1992) reported raised serum bilirubin level in 20% cases with elevation of liver enzymes in 3.03%. Sharma et al (1992) observed hyperbilirubinemia in 23% with normal SGPT. The impairment of liver function is because of haemolysis of erythrocytes.

In the present study 29 cases (19.33%) showed raised serum bilirubin and 9 (6%) raised SGPT levels at D5. This improve-
ment in liver function test is attributed to the dramatic reduction in the number of total parasites, thus affecting the less number of red blood corpuscle and hence less hemolysis. Considering the rise in serum bilirubin values the rise in SGPT values in malaria was modest and occurred only in a minority of the patients.

In this study, bleeding and clotting time were increased in 6 patients (4%). They presented with massive gastric bleeding, non survived till the 5th day. Sharma et al (1992) reported prolonged BT in 6.7% and CT in 3.33% cases of falciparum malaria. This may be because of disseminated intravascular coagulation.

Impairment of renal function is another complication of malaria. Raised blood urea was observed in 22.57%, 32.81% and raised serum creatinine levels were seen in 32.25%, 38.63% in vivax and falciparum respectively. As a whole 28% had raised blood urea levels and 36% creatinine levels. Ahmad et al (1989) reported impaired renal function in 48% of the total malaria cases of which falciparum were 66% and vivax 30%.

Halte et al (1990) reported acute renal failure in 33% patients with severe falciparum malaria. Deb et al (1992) reported elevated level of blood urea in 57.2% and raised serum creatinine levels in 48.57% falciparum infection.

In the present study 20.66% of the total patients had elevated blood urea levels and 26% had raised serum creatinine levels on the 5th day. This improvement in renal function is because of early recognition of the complications and aggressive management
and thus improvement in renal microcirculation.

Hypoglycemia was present in 6.45% P. vivax and 13.63% P. falciparum cases. Over all 10.66% patients had hypoglycemia at the time of clinical presentation. At day 5 none had hypoglycemia. Sherman et al (1979) reported that in animal malaria parasitized red blood cells consume upto 75 times more glucose than uninfected cells and concluded that this factor may contribute to hypoglycemia in the severe cases with high degree of parasitaemia.