DISCUSSION
AGE

In the present study incidence of tetanus neonatorum was 31.66%. Batt et al (1979) have reported an overall incidence of 14.3% of tetanus neonatorum. Suri (1967) reported 26.8% over all incidence of tetanus neonatorum. Childhood tetanus constituted 32.22% of total tetanus cases. A. Athavale et al (1974) reported in their series an incidence of 39% of total tetanus cases due to childhood tetanus. Adult tetanus contributed to 36.12% of total tetanus cases. Cerden et al (1961) reported a very high incidence of tetanus in Punjab. It was 90.4 per 100000 cases per year. Adults are more active physically which make them more prone to injuries and infections. So there was a higher incidence of tetanus in this age group.

Over all male and female ratio in present study was 2.4:1. Mathur (1980) reported a male:female ratio of 5.4:1. In tetanus. Kachanovi (1952) suggested that male were more sensitive to tetanus toxin as compared to females and hence a high male preponderance.

Our study of the effect of propanolol on the tetanus patients is slightly different from other similar studies that we concluded all patients of tetanus for the propanolol regime as compared to others who used the propanolol only in those patients of tetanus showing
excessive sympathetic over activity. This regime was contemplated so that in some patients the prophylactic action of propanolol would prevent the sympathetic over activity. Day one in all observations denotes the pretreatment levels because this was the day when propanolol therapy was instituted.

**PULSE RATE**

Our observations indicate that in the treatment group there was a significant decrease in the degree of tachycardia starting 24 hours after the treatment and becoming more marked at the end of 1st week. As opposed to the control group where the pulse always remained at the higher level. This finding agrees with the finding of Kerr et al (1968). The same response was also reported by Robert (1969) and Sainani et al (1972).

This tachycardia persisted for a longer period of time in the untreated group but eventually came down to normal levels by about day 14th. Corresponding to the recovery phase from tetanus. This finding agrees with that of Mathur et al (1987).

The tachycardia has been variously attributed to sympathetic over activity (Kerr, 1967 & 1968), secondary infection (Macrae, 1967), Hyperpyrexia (Clark & Taylor, 1966), myocarditis (Alhandy et al, 1960) or the association with hypertension.
Although any of these causes may be at work within a patient of tetanus, the only cause which appears to be common in all patients was sympathetic over activity. This is all the more substantiated by the subsidence of tachycardia after propanolol therapy.

Kerr et al (1967) reported that tachycardia occurred in both the presence and absence of secondary infection and pyrexia. Clark and Taylor (1966) reported that tachycardia occurred associated with hypothermia in tetanus. In myocarditis high heart rates are not associated with hypertension although Clark and Taylor (1966) have often observed the combination in patients of tetanus.

HYPERTENSION

The hypertension observed was in 31% and 4% on day 8th in control and study group respectively in accordance with the findings seen by a number of the investigators.

The temporary hypertension has been recorded by Lassen et al (1954), Kloetzel (1963) and Albett (1967) and was often precipitated by pulmonary collapse. Kerr et al (1965) also showed that raised blood pressure was due to presence of normal or low carbon dioxide.

Hypertension due to renal cause associated with high or low values of blood urea, urinary flow and specific gravity has been never postulated (Kerr et al, 1968). In
the polyneuritis and porphyria and bulbar poliomyelitis origin of hypertension is neuronal and (Tylen and Dawson, 1981) has some features common with the changes seen in tetanus. But difference is spasm superimposed on a background of considerable muscular rigidity are characteristic of untreated tetanus. Neither is this hypertension solely because of excessive muscular activity because the hypertension persisted even after curarization and appeared in a couple of days after the onset of muscular spasm.

Following the period of fluctuating hypertension some times severely affected patients developed hypotension accompanied by hyper pyrexia and profound peripheral vasoconstriction with a glove and stocking distribution (Alhady et al, 1960; Eliftan, 1964; and Macrae, 1967). In our study no such type of hypotension was seen in any patient. The hypotension may be due to body fluid and electrolyte disturbance or to episode of hypoxia and over indulgent drug therapy (Adams et al, 1966; Albert, 1967), or septicemia (Cliftan, 1964; Adams et al, 1966) or in toxic myocarditis reported by Lassen et al, 1954). Alhady et al (1960), Cliftan (1964), Adams et al (1966) and Macrae (1967) reported that hypotension can occur due to tachyphylaxic during the treatment with sympathetic mimetic agents. The sympathetic stimulation will also produce systemic vasoconstriction and it causes local hypoxia with increase cell permeability resulting in hypotension (Treeman, 1933; Mourit et al, 1958).
The increase sympathetic activity as a cause for the hypertension in tetanus has been confirmed both by increased catecholamines secretion (Kerr et al, 1968) and by the increased blood levels which was reported by Keitty et al (1966).

The drug propranolol is prototype of beta adrenergic blockers and can be used to suppress the somatic manifestation of tetanus in heart by blocking adrenergic receptors (Ablquist, 1978) and to control the cardiovascular disturbance.

Our findings favour the results reported by Sainani et al (1972) and Mathur et al (1987). But disagree with the results reported by Alhady et al (1960), Clifftan, (1964) and Taylor and Dawson (1961).

**HYPERTHERMIA**

In our study there was less significant reversal of hyperthermia because from day 1st to day 4th fall in percentage of patients suffering from hypothermia fell down 22% to 16.5% but from day 4th to day 8th hyperthermia increased in patients from 16.5% to 21.39%. These findings are accordance to the findings of Mathur et al (1987) and Sainani et al (1981).

Glossop and Low (1957), Kloetzel (1973) and Stirnemann (1966) reported that raised body temperature often occurs in paralysed patients with severe tetanus
without secondary infection, which develops due to peripheral vasoconstriction (Miller et al, 1958 and Macrae, 1967). Disturbance of temperature regulating centre is another cause of hyperpyrexia (Baten, 1942; Glossop and Low, 1957; Matgema, 1961 and Pearie, 1967).

Apart from the above obvious causes the urinary catheter, Ryle's tubes, venflon for I/V drug may also be important causes for hyperthermia. In addition, the environmental temperature is also an important cause for hyperthermia.

Summing up the entire observations, it can be said that although each of parameters namely, blood pressure, pulse rate, hyperpyrexia, may have a number of possible individual causes, but the three taken together can best be explained on the basis of a common cause; namely sympathetic over stimulation. The recognition of this in association with tetanus because the more important cause hyperpyrexia and persisting tachycardia are important causes of mortality in tetanus. The control of this associated sympathetic over activity thus forms an essential and important part in treatment of every patient of tetanus.

The addition of an alpha adrenergic blocker has also been suggested by Prys-Roberts and Corbett because they believe that alpha receptor stimulation may be the cause of cardiac arrhythmias and labile hypertension. Thus a cause for the use of alpha adrenergic blocker
along with beta blockers can be made in this kind of therapy.

We have suggested the use of propanolol in all patients in apart from those who develop sympathetic over activity because we feel that prophylaxis may improve the results by preventing the sympathetic over activity.

HOSPITAL STAY

In our study the hospital stay in the treatment group became shortened as compared to control group. 82% of treatment group are discharged within 2 weeks and 67% from the control group within the first two weeks.

MORTALITY

Our study showed the mortality in both the groups control and treatment group respectively 32% and 24%. Reduction in rate of mortality after treating with propanolol in tetanus was reported by Sainani et al (1972) and Mathur et al (1987).