CHAPTER IV

Concept of Etiological Background of Buerger's Disease
Review of Literature of Biological Factors

The exact etiology of Buerger's disease is yet to be established and this despite of the innumerable researches carried out for the last hundred years.

1. Infective Factor

Buerger (1908) was the first to suggest bacterial origin of this disease. But he failed to establish the cause of it. Yet he was positive that he could not find out any syphilitic background of this malady. Buerger (1914) further stated that thromboangiitis obliterans was an infectious disease in which a specific type of organism was at work. However, he could not detect any causative factor in this regard. He attempted to search for the presence of spirochaetae, tubercle bacilli, and the ordinary pyogenic organisms without any result. He presumed the infective background by acute thrombophlebitis, giant-cell foci and round-cell infiltration in the veins.

Rabinowitz (1923) prepared several blood cultures of a few thromboangiitis obliterans cases. He claimed to have found a sort of gram-negative bacillus from these cultures and to have produced some vascular
Molonoy and Hiller (1925) conducted bacteriological studies, but could not produce any convincing evidence of the micro-organisms that had been found out by Sabinowitz.

Brown and Allen (1928) strongly suggested infective origin of this disease; but they could not, in proof of their suggestion, find out any organisms.

Buerger (1929) carried out some experimental works on animals. Nevertheless, no organism or toxic factor was discovered by him in the affected vessels.

Horton and Doraey (1932) performed bacteriological investigations into the cause of thromboangitis obliterans. They had succeeded in producing the pathological lesions in experimental animals. They suggested that this disease was of infective origin, possibly with the streptococcus as the etiologic agent. But in the next year (1933) Parlow strongly criticized the work of Horton and Doraey. He remarked that the experimental work of these authors was definitely inconclusive and not substantive.

Telford and Stopford (1935) were most positive in their assertion, "syphilis has nothing to do with this
disease*. Of course they could not substantiate the presence of any organism for the genesis of this vascular disease.

Silbert (1935) opined that Buergor's disease was an inflammatory condition, but did not substantiate it. Som (1952) found the blood culture negative in Buergor's patients.

2. Fungal factor

Naide (1941) suggested the possibility of dermatophytosis producing Buergor's disease. Boyet et al (1949) and Boyd (1950) emphasized that patchy superficial phlebitis was commonly associated with dermatophytosis of the interdigital folds. Of course the role of fungi could not be assessed by any one of the above workers. Jimenez et al (1960) reported that the majority of their patients had a history of fungal infection between the toes. They suggested that micological studies might reveal an angiopathic variety of fungus. So far the evidence in favour of a primary fungal infective etiological factor in Buergor's disease was far from impressive. Orban (1961) discarded the possibility of infection in Buergor's disease. He emphasized that no convincing proof was available regarding infection, smoking of tobacco,
ergot, race and degenerative changes in sympathetic ganglia as the causes of this malady. Martin (1963) stated that although epidermophytosis often accompanies thromboangiitis but there was no evidence to suggest fungus infection as a cause of the disease. Marples (1965) stated that the disease might be more common in the Far East where dermatophytosis was more virulent. Schatz et al (1966) supported the infective background by demonstrating the inflammatory changes in the vessels in 9 out of their 12 cases. Craven and Cotton (1967) opined that the theory of dermatophytosis as a cause of Buerger's disease failed to explain the low incidence of this disease amongst patients with dermatophytosis. They also suggested an angiitic reaction to fungal infection, due to a local Shwartzman reaction to an endotoxin produced locally by bacteria or fungi. Eastcott (1969) stated that fungal infections were frequently present between the toes of patients with the Buerger syndrome. Hill and McElions (1972) stated that the patients who suffered from Buerger's disease might have associated fungal infection but it did predispose to a worse prognosis. Mannick and Coffman (1973) postulated that Buerger's disease was related to fungus disease of the foot. Hill et al (1973) suggested the combined action of micotic infection, tobacco smoking
and cold injury as the etiology of Buerger's disease. Of course the problem of fungal infection had never been clarified. Corelli (1973) stated that Buerger's disease was not related to any infection and it was an "abacterial" condition.

3. Thrombotic factor

Telford and Stopford (1935) were positive that the coagulability time of blood could not yield any clue to its etiology. Theis and Freeland (1939) opined that hypercoagulability of blood might be responsible for some of the pathological changes of Buerger's disease. However, there was no proof of this conjecture.

Takata (1943) advised the performance of the delicate heparin tolerance test in Buerger's disease with a view to finding out the coagulability of blood in this disease, but no cause of the pathology could be detected in this line. Hagedorn and Barker (1948) reported an increased heparin tolerance in a few patients of Buerger's disease which might be interpreted as an increased tendency to coagulation.

Eisen et al (1951) carried out research and reported a rise in platelet adhesiveness.
Fisher (1957) opined that stagnation thrombosis is the cause of secondary vascular obstruction in the distal part.

Gore and Burrows (1958) believed that in Buerger's disease there was an augmented tendency to deposit fibrin upon the lining of the vessel, and this might give rise to intimal damage in the absence of disturbance of lipid metabolism in this disease.

Ming et al (1959) examined pathological specimens from 26 patients and concluded that the lesions of Buerger's disease might be due to multiple embolization, thrombosis and atheroma. Spittell et al (1960) performed a modified thromboplastin generation test to assess the coagulability of blood in Buerger's disease. They observed in a few patients an acceleration in the early phase of their coagulation mechanism. Barker (1962) suggested primary changes in the vascular endothelium which might be responsible for intensive cellular proliferation. This view was not in favour of primary angiitic process. He opined that a systemic hypercoagulable state probably existed as an associated phenomenon even if there was morphological or biochemical change in the endothelium. Goodman et al (1965) found a positive latex fixation
test in 17% of Buerger's disease as compared to 3.5 percent in a control group.

Several tests were performed by some workers to find out the hypercoagulability of blood but no conclusive evidence could be derived at in this regard.

Craven and Cotton (1967) studied the heparin precipitable fraction of fibrinogen (HPF) in plasma and obtained a higher level in Buerger's disease than those in normal persons or in patients with atherosclerosis obliterans. They had the impression that the intravascular thrombosis might not be related to inflammation of the arteries and primary coagulability defect might be held responsible for spontaneous arterial thrombosis. De Camp et al (1968) reported abnormal thromboplastin generation test in support of the systemic hypercoagulable state as an associated phenomenon in Buerger's disease.

Williams (1969) stated that despite the extensive literature which had accumulated around this disease, its etiology remained unknown. He suggested that the initial changes were thrombosis and inflammation of the thrombus and ultimately the pathology spread to involve the vessel coats.
4. Value of Serum Cholesterol

Hypercholesterolemia was observed by many in the atherogenesis but not in Buerger's disease.

Anistachkov (1913) produced vascular lesions in the rabbit after prolonged dietary hypercholesterolemia. Heath (1949), Graham et al (1951), Berkowitz (1960), Mathur et al (1961), and Bronte-Stewart (1961) suspected a high cholesterol level in atherogenesis. Of course the specific lipid changes and the degree of atherosclerosis could not be accounted for.

The influence of smoking on serum lipids was not precisely known. However, high serum cholesterol levels were found in young smokers by Karvonen et al (1959), Dawber et al (1959), Bronte-Stewart (1961), and Cramer et al (1966). Stephenson et al (1962) stated that the duration and intensity of hypercholesterolemia might have some influence upon the variation in gross extent of atheromatous lesions. They did not point out any change in Buerger's disease.

Robertson (1962) discussed the role of cholesterol in the vascular changes. He opined that active metabolic process of lipid deposition into the intima through the
endothelial lining or the result of local synthesis might be the mechanisms of atherogenesis, and high concentrations of cholesterol, phospholipids and triglycerides were necessary. Szilágyi et al (1964) reported that none of their Buerger's patients displayed hypercholesteremia, and its persistent absence in this disease had some diagnostic value. Goodman et al (1965) reported that the serum cholesterol levels were within normal limits in patients with Buerger's disease and a high level in those with arteriosclerosis obliterans. Herrington and Grossman (1968) stated that in Buerger's disease changes in blood lipids, cholesterol levels, and deposits of calcium in vessel walls were characteristically absent. Morris-Jones and Jones (1973) reported a female Buerger's patient who had normal laboratory investigation reports. Her serum cholesterol level was 250 mg. % and glucose tolerance test was normal. Zelikovsky et al (1973) found no abnormality in a female Buerger's patient in their all laboratory investigations. Shead et al (1978) stated that hyperlipidaemia was a feature of atheromatous condition and not of thromboangiitis obliterans.

5. Value of Blood Glucose Level

Semple (1953) stated that the atherosclerosis which complicated diabetes was known to have a predilection for small vessels. In this type it simulated Buerger's disease.
and therefore it was imperative to exclude diabetes.

McPherson et al (1963) found that none of the patients with Buerger's disease except one had diabetes mellitus.


6. Role of Vitamin deficiency

Hill et al (1973) stated that there was no clear vitamin deficiency syndrome seen in any of their patients.

7. Immunological factors

Gore and Burrows (1958) postulated that an allergic basis was responsible for the episodic thrombotic disorder of Buerger's disease, and the thrombus or its breakdown
product might be the cause of the focal or segmental vasculitis.

However, the conception of "Autoimmunity" as the cause of vascular disorders received increasing attention. There might be an imbalanced immune response. The immune proteins might have some toxic complexes.

Immunological factors might be responsible for digital ischaemia in rheumatoid arthritis, systemic scleroderma, dermatomyositis, systemic lupus erythematosus, primary biliary cirrhosis, macroglobulinaemia, cryoglobulinaemia and acute polyarteritis nodosa. But no report was available in favour of immunological disorders in Buerger's disease.


Blumenthal et al (1966) performed histopathologic and immunopathologic studies and suggested that there could well be an immunologic relationship between diabetes
mollitus and Buerger's disease. But no cases of diabetes were found in this review. Peracchia and Vassallo (1966) performed electron microscopy studies in Buerger's disease. They showed abnormality in the collagen at molecular level, suggesting that the condition might be a collagen disorder.

Morris-Jones and Jones (1973) reported a female Buerger's disease. No L.E. cells were found in the blood. The immunoglobulin, Ig G, Ig A, and Ig M were within normal limits, and the smooth muscle and mitochondrial antibodies were absent on immunofluorescence testing. They stated, "Investigations in our personal case, and in male cases under our care, such as E.S.R., plasma proteins, immunoglobulins and antinuclear factor testing, have not provided any evidence to suggest that this is an autoimmune phenomenon."

Zelikovsky et al (1973) investigated Buerger's disease in a woman and found no abnormality in their laboratory investigations. The electrophoresis showed some increase in gamma fraction. Immune electrophoresis was normal. Neither pathological albumins nor L.E. cells were found.

Corelli (1973) suggested smoke allergy was the cause of Buerger's disease. The antigens produced allergic
hyperergic angitis and inflammatory changes of the blood vessels.

Von Knorring et al (1974) suggested immune pathology in Buerger's disease. But they stated that no general acceptance had been found for any etiology of Buerger's disease. They opined that the involvement of nutrient vessels in Buerger's disease might lead to proliferation of the fibroblasts in the bone cavity, resulting in myelofibrosis, for instance as in disseminated myocardial fibrosis arising from diffuse coronary arteritis or sclerosis. Knospe and Crosby (1971) speculated whether destruction of the bone marrow sinusoidal microcirculation by an immune mechanism might induce human aplastic anaemia.

Dutz et al (1975) discussed immunity and vascular disease. They, however, did not mention Buerger's disease as an immunological abnormality.

Kim et al (1976) stated that the question of whether pathologic involvement of the sympathetic chain was part of the pathologic process of generalised arterial disease had also been suggested but never thoroughly explored as a possible cause of the unpredictability and variability of results.
8. Hormonal factor

Some endocrine basis peculiar to the male sex which can play a dominant part in the causation of Buerger's disease may be thought over. But proof has not yet been forthcoming.

Kuntze (1962) remarked that the Buerger's disease was probably a primary endothelial destruction of unknown etiology, perhaps only in susceptible vessels. The male incidence suggested that oestrogens gave some protection to the female subjects.

9. Role of Blood Viscosity

Silbert et al (1930) reported that in the year 1912 Mayosima observed high haemoglobin percentage and increased viscosity of blood in thromboangiitis obliterans, and he supposed that these might be the causes of this disease. In 1930 Silbert, Kornswieg and Friedlander came forward to work along the above line. They noted diminution of blood volume in Buerger's patients. But its etiological responsibility for the production of this disease was not established. Friedlander and Silbert (1931) performed chemical analysis of blood. They could not draw any conclusion on blood biochemistry with the causation of this vascular disease.
10. Value of Blood Group

Kingsbury (1971) found a convincing preponderance of blood group A over 0 in a large number of patients with occlusive arteriosclerosis. They did not mention any blood group in Buerger's disease.

Morris and Bouhoutaos (1973) stated that occlusive disease and myocardial infarction occurred more commonly in Group A individuals and that blood group 0 seemed to confer some immunity to occlusive vascular disease. They did not mention any blood group in Buerger's disease.

11. Role of Tobacco Smoking in Buerger's Disease

Almost all patients of Buerger's disease were smokers. Many investigators thought that smoking of tobacco was an etiological background of this vascular disorder. Nevertheless, no definite conclusive evidence could be demonstrated by ardent workers in favour of smoking as an etiology of Buerger's disease. The valuable observations of these workers have been discussed here with a view to obtaining some clues in regard of the incidence of smokers in Buerger's disease, mechanism of the vascular changes in smoking, the effect of smoking on Buerger's disease, the respiratory activity of smoking, the immunological role of smoking, the effect of smoking on adrenal
glanda, the miscellaneous effect of smoking, and the progress and prognosis of vascular disorder in smoking.

The incidence of smokers in Buerger's disease

It was formerly believed that the low incidence of Buerger's disease in female subjects could be due to their low consumption of tobacco. Herroll and Allen (1936) postulated that the ratio of female to male smokers was 1:6. This was much smaller than the ratio of female to male with Buerger's disease which was 1:70 to 1:500. Therefore, smoking alone did not explain the differential sex incidence of Buerger's disease. Stewart et al (1945) demonstrated the effects of smoking in old age and did not mention the effect of smoking in the young patients of thromboangiitis obliterans. Begg (1965) opined that generally heavy smokers suffer from occlusive vascular disease. Abramson (1965) pointed out that the incidence of Buerger's disease in women was very rare even though smoking was as common among women as among men. Badie et al (1968) reported high incidence of heavy cigarette smokers of Buerger's disease. Brown et al (1969) reported cent per cent tobacco smoking in 28 Japanese and 12 American patients of Buerger's disease. Richards (1972) expressed his opinion that in all occlusive
arterial diseases and especially in Buerger's disease the incidence of heavy cigarette smoking was high.

**Mechanism of vascular changes in smoking**

Vasoconstriction in the human hand due to smoking was demonstrated in 1909 by Bruce et al when he studied the effect of smoking upon the blood pressures and upon the volume of the hand. This work of Bruce encouraged other workers to study the effects of cigarette smoking on the peripheral circulation. Lilienthal (1914) studied the relation of smoking in clinical subjects. He was the first person to suggest the existence of an intimate relation between smoking and Buerger's disease. The most serious effect of tobacco smoking in peripheral vascular disease was vasconstriction. Lampson (1935), Roth et al (1944), and Eckstein et al (1957) demonstrated a decrease in skin blood flow during tobacco smoking. The vasoconstrictor effect of nicotine was experimentally demonstrated by Kotlegoda (1953). He presumed that the vasoconstricting effect of nicotine was due to liberation of epinephrine or norepinephrine from the vessel wall or in its vicinity. The concept of liberation of noradrenaline from a peripheral store by nicotine was also supported by Gordon and Adams-Ray (1957), Burn and Rand (1958) and Burn et al (1959). Friedell (1953) did not find any significant
vasoconstrictive effects after smoking in the persons over 35 years of age which might be due to decreased tissue reactivity in older patients. Hilton (1954) studied the effects of nicotine on the blood vessels of skeletal muscles in the cat and not in patients of Buerger's disease. Vogt (1954) demonstrated a large amount of norepinephrine in the hypothalamus in experimental animals following the administration of nicotine. But no such information was obtained in Buerger's disease. It was also presumed by Nordonam and Adams-Ray (1957) that local liberation of norepinephrine, a vasoconstrictor substance, from the adrenal medulla and chromafin cells of the skin took place due to the influence of nicotine. Burn and Pond (1958) also opined that the constrictor action of nicotine and acetylcholine in the rabbit's ear was due to the release of norepinephrine. Burn et al (1959) postulated that the peripheral effects of nicotine were due to liberation of noradrenaline from a peripheral store. Simon and Iglauer (1960) studied the circulatory effects of tobacco and did not find any circulatory change after smoking cigarette. Eckstein and Horaljy (1960) noted reductions in the resting volume of the extremity after smoking. They believed that venous constriction might be the cause of this change. Wood (1960) stated that inhalation of tobacco could reduce the cutaneous blood flow and
especially of the toes. The circulation of skin was greatly compromised after smoking. Burn (1960) remarked that nicotine produces vasoconstriction by stimulating sympathetic ganglia with consequent discharge of impulses along postganglionic fibres and also by stimulating the adrenal medulla, with consequent discharge of epinephrine. On the contrary Bottenstein et al (1960) expressed the view that important vasoactive ingredient in tobacco is nicotine, and he observed increase in blood flow to muscle and decrease in blood flow to the fingers by intravenous injection of nicotine. Freund and Ward (1960) observed constriction of arteries after cigarette smoking. A decrease in venous oxygen saturation was thought to be the cause of vasoconstriction. Therefore, it could not be explained with a precision whether smoking was the only factor of vasoconstriction or breathing episode was the contributory factor. Greenberg et al (1952) studied the respiratory role during smoking. They postulated that the respiratory activity alone during smoking might give rise to vasoconstriction. Wright (1963) supported the idea of vasoconstricting effect of tobacco.

Sensitivity and allergy to smoking

Harkavy et al (1932) and Sulzberger (1933) believed that vascular tree might be sensitive to tobacco. A
transient fall of cutaneous temperature due most likely to cutaneous vasoconstrictive effect of nicotine was demonstrated by Johnson and Short (1934). Chobot (1935), Trasoff et al (1936) and Westcott and Wright (1938) failed to confirm the contention that the skin reaction produced by tobacco extract was due to a sensitization process of the blood vessels. Abramson et al (1939) studied the effects of smoking upon the vessels of the limbs; but their observations could not substantiate the effect of smoking in the development of thromboangiitis obliterans. Mulinos and Shulman (1940) also studied the blood flow in a limb during smoking. He noted diminished blood flow after smoking in normal subjects. Goetz (1942) postulated that the peripheral vasoconstriction caused by smoking results from immediate reflex action due to inhalation and also due to the pharmacological effect of nicotine. Cooke (1947) observed the allergic sensitivity of smoking on cutaneous vessels. Fontana (1960) recommended skin test with tobacco extracts in determining the possible importance of tobacco as an etiological factor in vascular disease. Kjeldaen and Mozes (1969) believed that toxic substances in tobacco smoke was the cause of Buerger's disease.

Mannick and Coffman (1973) said that no conclusive evidence was known which could substantiate tobacco smoking.
as an etiology of Buerger's disease. However, Hill and Smith (1974) stated that in Indonesia Buerger's disease was caused by cigarette smoking. Heraud and Clovot (1977) stated that arteries in young subjects due to Buerger's disease generally appeared in heavy smokers. Abramson (1977) suggested that smoking might be held responsible for atheromatous conditions, and might not be a specific cause of Buerger's disease. Naik et al (1978) reported the incidence of bidi smokers in Buerger's disease. This observation convinced these authors that the smoke from unprocessed tobacco stuffed in bidi leaves and processed tobacco of cigarettes had unequal injurious effect to peripheral vascular system. Individual susceptibility to prolonged tobacco is presumed to be the cause of Buerger's disease. Holt and Keast (1977) remarked that impaired immunological control mechanisms might develop after inhalation of tobacco smoke.

**Effect on Adrenal Glands**

From experimental study Silvetti et al (1961) showed that nicotine could act by stimulating the sympathetic nervous system and by releasing catecholamines from the adrenal medulla.
Miscellaneous effects

Wald et al (1973) observed damage of vascular walls by tobacco smoking due to carboxyhaemoglobin level which might cause occlusive arterial disease. Astrup (1964) stated that haemoglobin could not readily part with its oxygen in smoking. Astrup et al (1967) investigated the possible reasons for damage to blood vessels by smoking, and observed cholesterol accumulation in vessels in rabbits. Birnstingl et al (1967) opined that vascular damage in heavy cigarette smokers was due to absorption of carbonmonoxide.

Effects and progress of smoking in Buerger's disease

Silbert (1930) discussed the effect of tobacco on clinical course of Buerger's disease. He believed that smoking was the most important contributing factor in producing the disease and if smoking was stopped the progress would be less, and he opined that smoking had the vasoconstricting action of nicotine which was the most common immediate cause of the development of gangrene. Silbert (1935) supported smoking as an etiology of Buerger's disease. He believed that cessation of the smoking might arrest the progress. Silbert (1945) admitted that the relation between smoking and progress of arterial insufficiency was not widely observed. Wessler et al (1960)
analysed the relation between smoking and progression of arterial insufficiency. They did not agree with the concept that a continued smoking could cause progressive vascular damage. Corelli (1961) also believed that tobacco smoke was a cause of Buerger's disease, and cure might be permanent if the patient refrained from it. Oldham (1961) opined that in thromboangiitis obliterans the ill-effects of smoking were infinitely worse than that in atherosclerosis. Goodman et al (1965) observed a mixed response of the progress after abstaining from smoking. According to Schatz et al (1966), remarkable arrest of the vascular deterioration occurred if the patients stopped smoking. However, it was contradicted by Winaor and Hyman (1965). Bloor (1966) in a careful study of smoking habits in Buerger's disease failed to find any effect of smoking on the course of the disease. Whereas in the same year Schatz et al (1966) remarked that this disease almost always progressed if the patient continued to smoke, and became arrested if smoking was stopped.

Brown et al (1969) noticed remissions of symptoms in two patients who stopped smoking. Morris-Jones and Jones (1973) opined that the disease continued in some well established cases, despite the cessation of smoking and also went into remission despite continuation of smoking. Corelli (1973) held the view that tobacco smoke
was the cause of Buerger's disease and its progress could be arrested if smoking was stopped.

Stricht et al (1973) stated that the evolution of Buerger's disease was favourable after complete suppression of cigarette smoking except in grossly ischaemic limbs. Rosenberger et al (1973) noted exacerbation of the symptoms in heavy smokers. Lawton (1973) stated that cigarette smoking was more dangerous for atherosclerosis of the coronary arteries than that of peripheral arteries. Mannick and Coffman (1973) observed that smoking did not aggravate the symptoms of intermittent claudication. Shionoya et al (1976) remarked that the treatment of Buerger's disease had its cornerstone the avoidance of tobacco.

**Buerger's disease in nonsmokers**

Perla (1925) and later on Allen and Brown (1928) found Buerger's disease in nonsmokers. Barker (1931) found this disease in only 1% nonsmokers. Goodman et al (1965) reported only one nonsmoker of this disease. Stojanovic et al (1973) reported Buerger's disease in 15 patients who never smoked.
Discussion of the observations of various blood and biochemical changes in Buerger's disease

Different investigators at different times carried out their works on various etiological factors of Buerger's disease. The author also during more than two decades explored the role of many etiological factors such as coagulative efficiency of blood, blood sugar, blood cholesterol, blood urea, E.S.R., and blood grouping. However, none of these could be definitely established as the etiological cause of Buerger's disease except the infective process which has been discussed subsequently.

In Buerger's disease intravascular thrombosis was encountered in most of the cases. The pertinent question that naturally came to the mind was, what caused the intravascular thrombus formation? No definite and conclusive cause of this could be known. Sen and Chakrabarti (1965) gave an account of the clotting efficiency of blood in Buerger's disease, but no abnormality could be seen. No definite abnormality of the coagulative factors of blood in this disease could also be noted by Tolford and Stopford (1935), Thos (1939), Takats (1943), Hagedron and Barker (1948), Spittell et al (1960),
Baricor (1962), Craven and Cotton (1947), and Do Camp et al (1968).

The exact cause of Buerger's disease having remained unidentified, ardent workers in their endeavour to explore exhaustively this dark etiological region shed revealing light on it from all possible angles and penetrated the obscurity. So it was that a group of them started researches to find out if there were any alteration in blood and biochemistry in this vascular disorder, but in spite of their best efforts nothing definite, past all question and doubt, could be established by these pioneers regarding either the reason for the slight changes actually observed by them or their responsibility for the production of the vascular lesion. Chakrabarti (1965) did not find any abnormality of blood sugar and blood cholesterol in this disease. In this present work blood sugar, blood cholesterol and blood urea were within normal values. Chakrabarti (1961) also reported high E.S.R. in presence of gangrene and ulcers due to tissue destruction and superadded infection.
Graphic Representation

Blood Groups
A - ■ - 55
B - - 44
O - - 54
AB - - 7

Fig. 1
Blood Groups
Blood groups in Buerger's disease

The blood groups of patients of Buerger's disease were examined in 160 cases with a view to finding out the relationship of any blood group with the etiopathogenesis of this disease. Kingsbury (1971) and Morris and Boutoutesos (1973) studied the blood group in vascular disorders other than Buerger's disease. Therefore, an attempt was made to obtain any clue in this regard.

Total 160 patients of Buerger's disease were examined for blood group. A mixed type of blood groups was noticed in this disease. Group A was present in a relatively largest number of cases. Fifty-five or 34.31% cases belonged to group A. The incidence of group O was the next common and 54 or 33.75% cases had group O. Group B cases were evident in 44 or 27.50% cases and the incidence of group AB was insignificant. Only 7 or 4.37% cases of Buerger's disease had group AB. The collective analysis of different blood groups in patients of Buerger's disease revealed the existence of blood groups A and O in large number of cases, and the difference of the number of cases of these two groups was not significant. Group B patients were also found in a good number of cases.
fact all three groups viz. A, B and O were usually come across in cases of Buerger's disease and no convincing preponderance of any of these three blood groups over the other could be found. It may be opined that no specific blood group is responsible in the etiological background of Buerger's disease.

From this data it can be presumed that people with AB blood group are more immune to Buerger's disease than the people with other blood groups.