AORTOILIAC ENDARTERECTOMY

THESIS SUBMITTED FOR PARTIAL FULFILLMENT OF

M.S. DEGREE IN GENERAL SURGERY

BY

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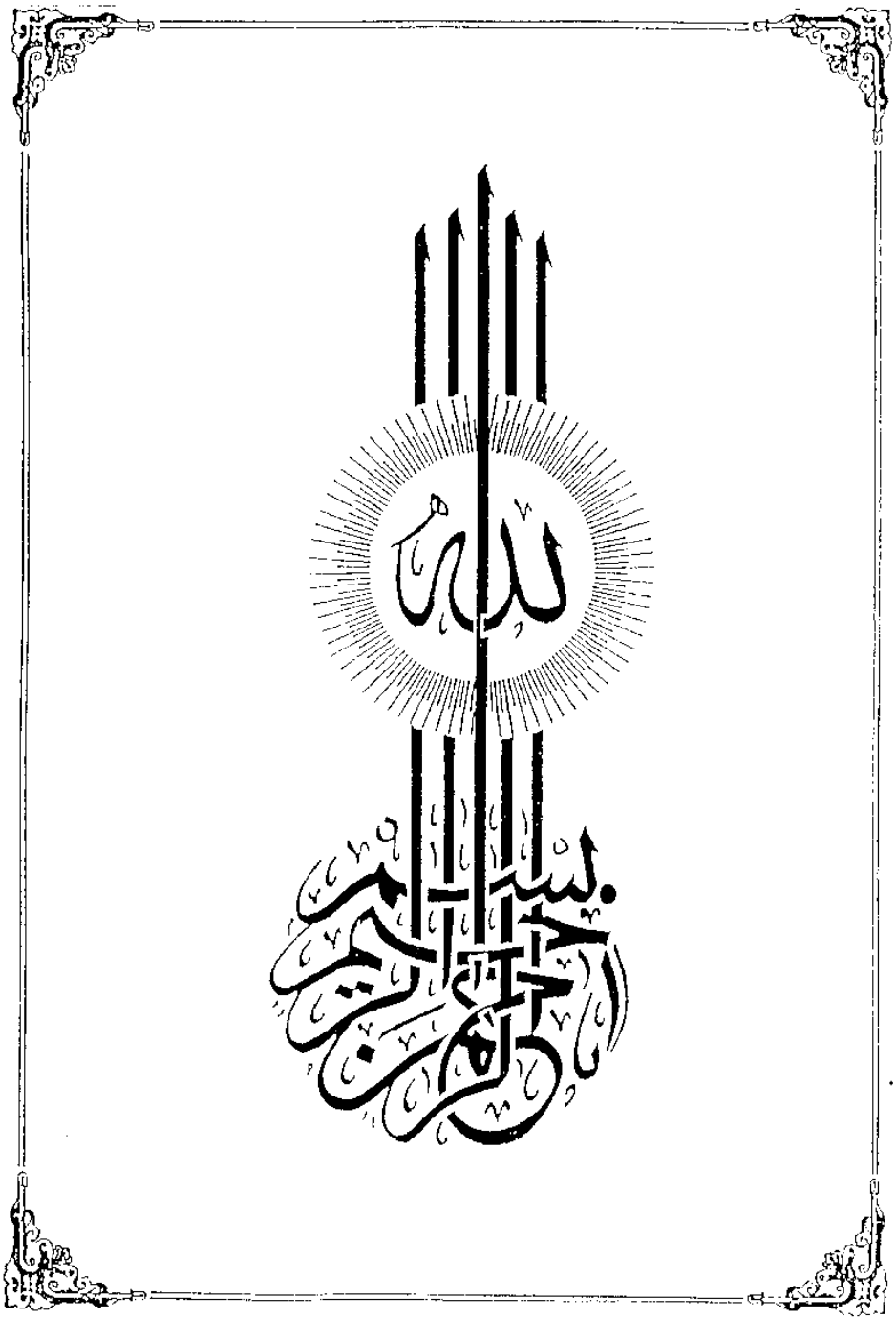

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INTRODUCTION

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HISTORICAL ASPECTS

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HISTORICAL ASPECTS OF AORTOILIAC ENDARTERECTOMY

The lower extrimity has been the site of many of the earliest approaches in the progress of vascular surgery.

Ligation of peripheral aneurysms was performed by the ancients. The records of their knowledge were in large part lost during the Middle ages.

the Russian physiologist, Eck, successfully performed the first experimental vascular anastomoses in the creation of canine portocaval shunts in 1877.

At the end of the nineteenth century, Alexis Carrel conducted classic experiments on arterial anastomosis and organ transplantation. He became the second surgeon to be rewarded with a Noble Prize. Murphy, 20 years after the animal experiments of Eck, first anastomosed a human artery in the distal femoral artery.

The initial successful vein graft operations were reported in 1906 by Goyanes, by Lexer in 1907 and by pringle in 1913.

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Goyanes and Pringle's vein grafts were performed for aneurysm of popliteal artery. Lexer's vein replacement was performed for an axillary aneurysm. Weglowski subsequently reported 51 vein grafts in German casualties in World War I. (Christopher ,1981).

Thrombotic obliteration of the aortic bifurcation was described by Leriche in 1923. Leriche emphasized that the disorder is a chronic process.

In 1929 dos Santos reported the first abdominal aortic arteriography. Great progress has been made then by Howell, after the discovery of heparin. However, it did not come to clinical usage until about 1939.

Thromboendarterectomy was introduced by dos Santos in 1947. This was followed in 1955 & 1967 by the reports of Cannon and Warren.

Successful aortic anastomosis was undertaken by Gross, and Crafoord of Stockholm in 1949. French surgeons were the first to undertake successful homograft replacement of the abdominal

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aortic bifurcation, Oudot (1951), for atherovc-lerotic occlusion, and Dubost (1953) for aortic aneurysm. From that time the clinical application of arterial reconstruction has become widespread by the contribution of Debakey and his colleagues in the U.S.A.(Imparato, 1979).

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PATHOLOGY AND PATHOGENESIS OF AORTOILIAC OCCLUSIVE DISEASE

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PATHOLOGY AND PATHOGENESIS

More than 95% of chronic occlusive arterial disease result from atherosclerosis. Theories regarding pathogenesis are controversial and have been discussed by many authors.

Strandness and associates (1960) described intimal proliferation and hyalinization of the muscular media of arterioles in the skin of diabetic and non diabetic patients with arteriosclerosis.

With the immunofluorescent technique it had been demonstrated by Blumenthal et al (1966) in some patients especially diabetics that the small arteries showing endothelial proliferation bind insulin and rabbit antihuman globulin. This reaction suggests an immunologic basis for the lesion.

Thrombosis is usually responsible for completing occlusion of a narrowed atherosclerotic artery. Wessler (1965) proposed that arterial thrombi are usually initiated by platelet

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aggregation " white thrombus " . They are therefore less likely to be prevented by anticoagulant therapy than are venous thrombi which are usually initiated by deposition of fibrin and erythrocytes " red thrombus".

In evaluating the functional effects of a stenotic lesion, it is mecessary to keep in mind that the degree of arterial occlusion must be quite marked before a reduction in blood pressure, and hence, in volume flow, occurs distal to the lesion. It has been shown experimentally that stenosis in the lumen size up to 60% produces only insignificant blood pressure changes. Stenosis between 60% and 70% of the lumen size is associated with definite alteration of blood flow distally (Haimovici,1954).

ETIOLOGY

A number of views have been proposed regarding the cause of atherosclerosis, but none of them has recieved general acceptance. It is clear however, that more than one factor is involved in the etiology of atherosclerosis. Such factors include :

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A. Alteration in lipid metabolism.

B. Alteration in the blood supply of the arterial wall.

C. Combination of normal and abnormal stress on the arterial wall.

A- ALTERATION IN LIPID METABOLISM

There has been a great amount of experimental work on the hypothesis that the pathologic changes in atherosclerosis are associated with , or caused by , error in the metabolism of fat and other lipids. (Oliver and Boyd 1955).

It has been shown that lesions similar to atheromatous plaques in humans can be produced in rabbits, chickens, rats and dogs, through the use of diet rich in cholesterol.

Stephenson et al.(1962) proposed that, errors of lipid metabolism are not suffecient to explain the atherosclerotic changes , but fatty plaques characteristic of atherosclerosis develop only if the arterial wall is injured. The blood lipoproteins are believed to become trapped in the

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involved segment of the vessel, with subsequent production of inflammation and fibrosis, followed by the formation of plaques.

Another view has been adopted by Barker(1975) who stated that;atherosclerosis does occur in the absence of what is usually recognized as hypercholesterolemia. The abnormally low proportion of cholesterol esters in atheromatous aortas as compared to normal ones was described by Windaus (1910). Thus , aspects other than absolute lipid levels must be considered .

The role of familial hypercholesterolaemia in coronary artery disease has been stressed by Kannell , 1971 and Streja et al.,1978. The relation between familial hypercholesterolaemia and coronary artery disease is well demonstrated in large-population prospective studies such as that in Farmingham. It might be so dramatic as in the case of 3.yr-old child with homozygos familial hypercholesterolaemia who died with a myocardial infarct in the studies of kindereds with familial hypercholesterolaemia.

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There is more certainty about the independent correlation of hypertriglyceridemia and

coronary artery disease.

This relation has been downplayed by the Farmingham data, while it has been considered a strong risk factor in the Stockholm prospective study by Bottiger et al.(1979,1980).

Unfortunately, that study does not provide data with respect to the subject's high density lipoprotein levels (HDL). This would be important to know because of the inverse relationship between very low density lipoproteins VLDL, and HDL,and occurrence of ischaemic heart disease . (Streja et al.,1978, Miller et al.,1977, and Schaefer et al., 1978).

Recently, Schonfeld et al.,1980 , raised some suspicion about the inverse relation between VLDL and HDL. It was realized that HDL levels do not increase when the triglyceride levels fall during treatment of hypertriglyceridemia.

Brunzell et al.(1976) has explained this controversy by suggesting that hypertriglyceridemics should be separated into those with familial hypertriglyceridemia and those with familial combined

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