F A T E M B O L I S M

Thesis submitted for the partial fulfilment of the Master degree in orthopaedic surgery .

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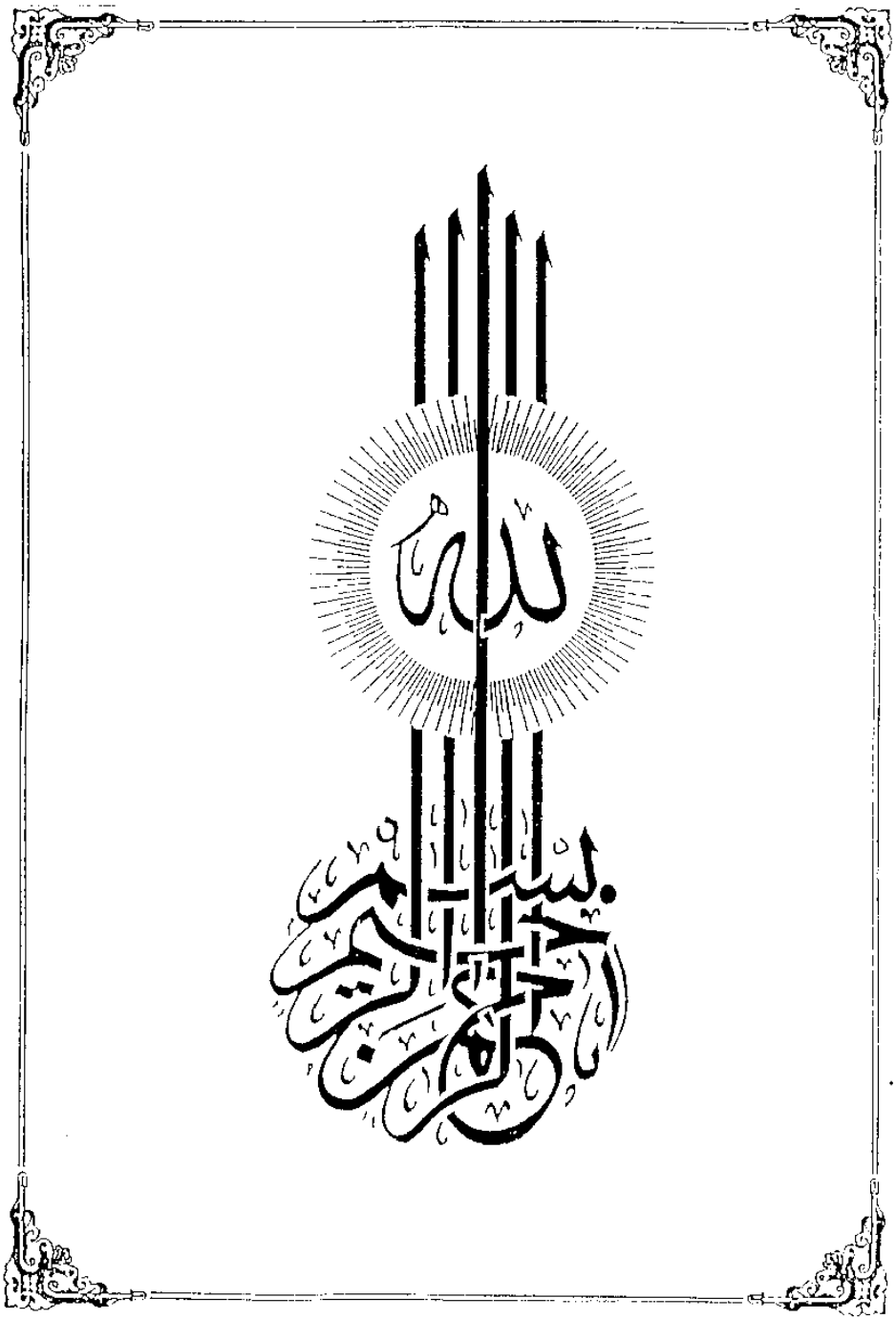

A C K N O W L E D G E M E N T
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CONTENTS

INTRODUCTION

(Fig. 1)

Photomicrograph using fluorescence microscopy demonstrating fat emboli in a dog sacrificed after multiple fractures.

The aim of this thesis is to review the literature dealing with the condition , its pathogenesis , diagnosis and treatment .

HISTORICAL REVIEW

noted the toxic effects of free fatty acids (Peltier, 1956). The result was a description of the respiratory theory (Peltier , 1969) which is still valid and is the basis of current therapeutic regimes .

Sevitt (1962) reported a 70% incidence in childhood fatalities from injuries . Szabo , Serenyi and Kocsar (1963) reported the post mortem presence of pulmonary emboli in 100% of a series of 10,000 victims of major accidental injuries . Sproule et al (1964) were the first to report severe arterial hypoxaemia in three patients with FES . Ashbaugh and Petty (1966) described the respiratory distress syndrome and emphasized the use of corticosteroids in the treatment of the respiratory complications of FES . Gurd's (1974) criteria for positive diagnosis is still in common usage . Renne (1978) described FES evidenced by fat macroglobulinaemia after total hip replacement Lachiewicz (1981) reported cases of FES following total knee arthroplasty using the hinge type prosthesis and two cases with the use of total condylar prosthesis . Lindque et al (1987)

suggested the routine determination of arterial blood gases on admission of any patient with a long bone fracture . They proposed new criteria for a positive diagnosis of FES and suggested the use of methylprednisolone as a prophylactic measure .

PATHOGENESIS

alter the physical state of lipids in the blood and enhance coalescence of the normally small chylomicrons into larger fat droplets with subsequent embolization and thrombus formation .

THEORIES EXPLAINING THE SOURCE OF EMBOLIC FAT :

1- The classical or mechanical theory :

It states that the cause of fat embolism is always trauma . The mechanism of fat embolism always results from mechanical factors and the three distinct stages by which the pathogenesis must proceed are :

(1) The injury of adipose tissue , sufficient to produce a disorganization of the supporting fibrous tissue stroma to liberate free fat into an injured area .

(2) The rupturing of a certain number of blood vessels specially the veins within the abraded area .

(3) The establishing of some mechanism which will cause the passage of free fat into the open ends of the blood vessels . Due to the presence of blood vessels within calcified tubules in the bones

these osseous tubules , lying within the bones and holding the blood vessels fast to their walls , cannot collapse . When a bone is broken sufficiently to open some of these intraosseous blood vessels , it is also broken sufficiently to cause a liberation of medullary fat , and , under these conditions , when the ends of the blood vessels are held open by their rigid perivascular sheaths , the first two stages of the pathogenesis are fulfilled . Then the same physiologic forces that maintain the venous circulation operate to draw free fat into the wide open ends of the blood vessels ,namely ,the regional muscular movements , the negative venous pressure and other forces .In this manner , considerable free fat enters the venous circulation , this is carried to the heart and thence to the lungs , where it lodges in the capillaries forming effective emboli , completely blocking many of the smaller vessels , setting up a train of pathologic changes including hyperaemia , oedma , occlusion of the arterioles , petechial haemorrhages and latter , round cell infiltration, (GAUSS , 1924) .

Evidence in favour of the mechanical theory :

The bone marrow cavities are extremely vascular, (Harrison , 1955) ; and the marrow fat is free after fractures and appears in the haematoma , (Bergentz , 1961). This is suggested by increasing the incidence of fat embolism after intramedullary nailing that has been carried out soon after injury , (Campbell , 1980) ; and after using acrylic cement ,that has been used since 1962 for the fixation of metal implants in bone after total hip arthroplasty pressing the marrow fat and causing rise in the medullary pressure, (Harris et al , 1975 and Campbell , 1980) . The observation of marrow and methacrylate emboli in the lungs of patients after total hip replacement have demonstrated the easy access of marrow contents to the fragile medullary sinuses, (Renne and Wurthier, 1978) .

Fatty emboli have been demonstrated in the blood by means of phosphin 3 , immediately after injury , and this can be experimentally prevented by prior vein ligation or application of a tourniquet , (Peltier , 1956) .