Effects of Excess Iron on The Performance of Isolated Ischaemic-Reperfused Rat Hearts

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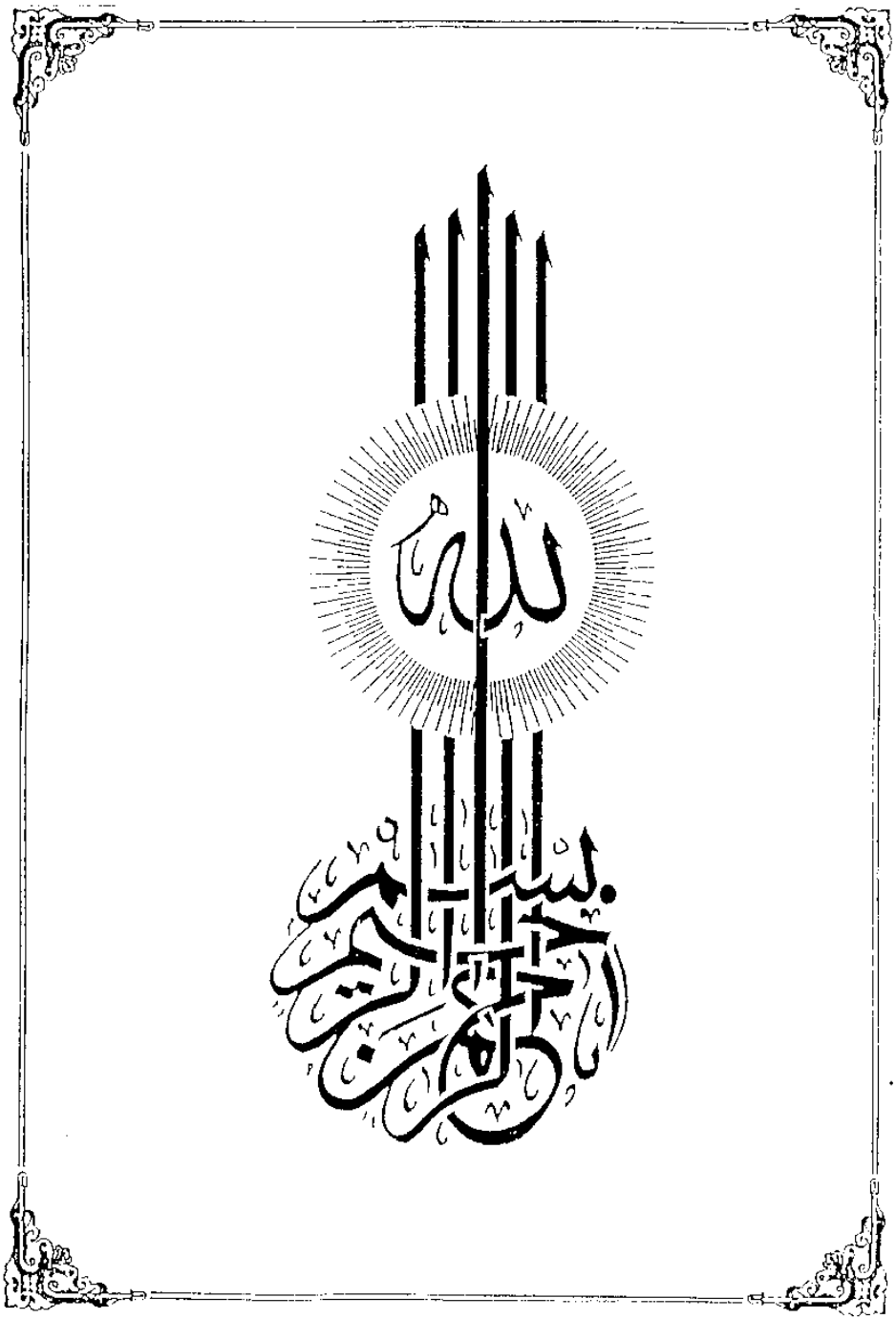
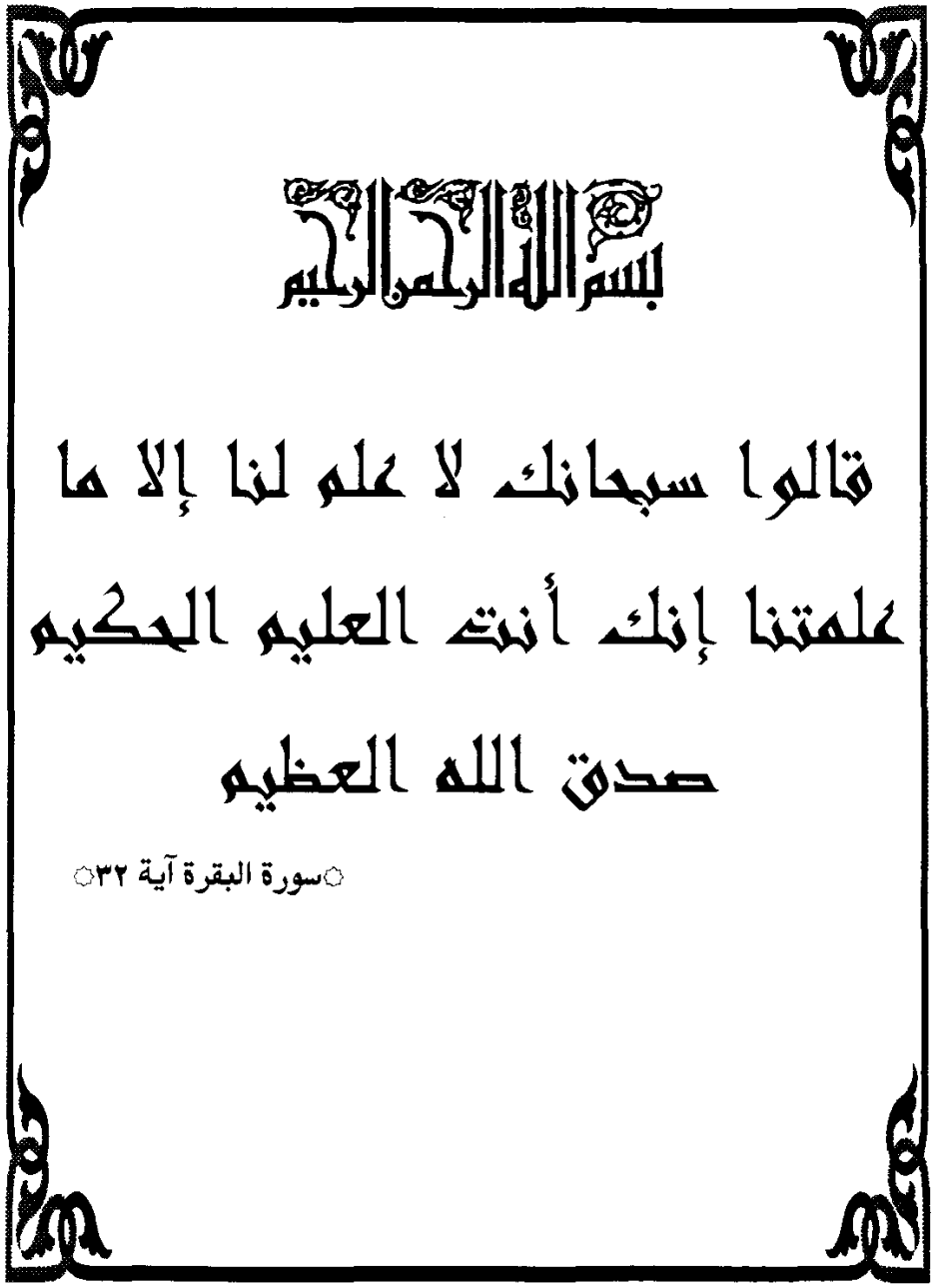
***** 1998 *****





بسم الله الرحمن الرحيم

قالوا سبحانك لا علم لنا إلا ما
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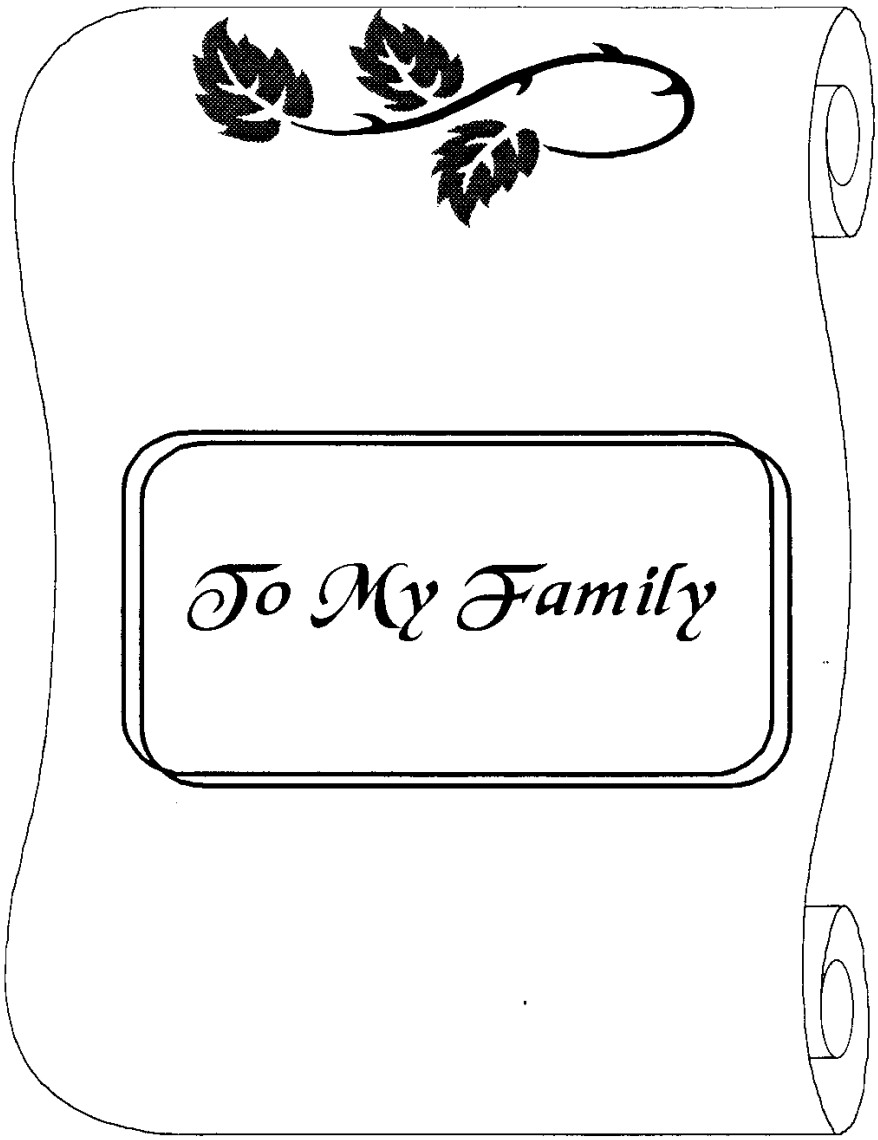

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Introduction

Introduction

Iron overload is encountered in chronically anemic patients on regular blood transfusion regimens, e.g. thalassemics, as well as in individuals in whom intestinal control of iron absorption is ineffective ***(Charlton et al., 1973).*** The eventual result is extensive iron – induced injury to the liver, pancreas, heart and other organs ***(Brittenham et al., 1994).***

On the other hand, cardiac insult was observed in hearts subjected to reperfusion following transient ischemia, the reperfusion being associated with more damage than improvement. In fact, it has been reported that timely reperfusion limits ischemic injury and myocardial infarct size ***(Reimer et al., 1977).*** Yet, ***Lucchesi, 1990,*** stated that although reperfusion halts the progression of ischemic injury, it predisposes to reperfusion process which may cause additional myocardial necrosis leading to suboptimal myocardial salvage.

The pathogenesis of reperfusion injury is multifactorial. Iron has been implicated in reperfusion – induced myocardial injury by playing an important role in free radical generation during ischemia reperfusion ***(Floyd and Lewis, 1983).***

It was, thus, intriguing to study the performance of iron – overloaded hearts subjected to ischemia – reperfusion, and to demonstrate a possible cardioprotective effect of the iron chelator desferrioxamine, proposed earlier to reduce the myocardial ischemia – reperfusion injury ***(Menasche et al, 1990).*** This might add more light on such important, yet still incompletely defined, issue.