SURGICAL MANAGEMENT OF NEONATAL NECROTIZING ENTEROCOLITIS

Essay

Submitted for the Partial Fulfillment for the Master Degree in General Surgery

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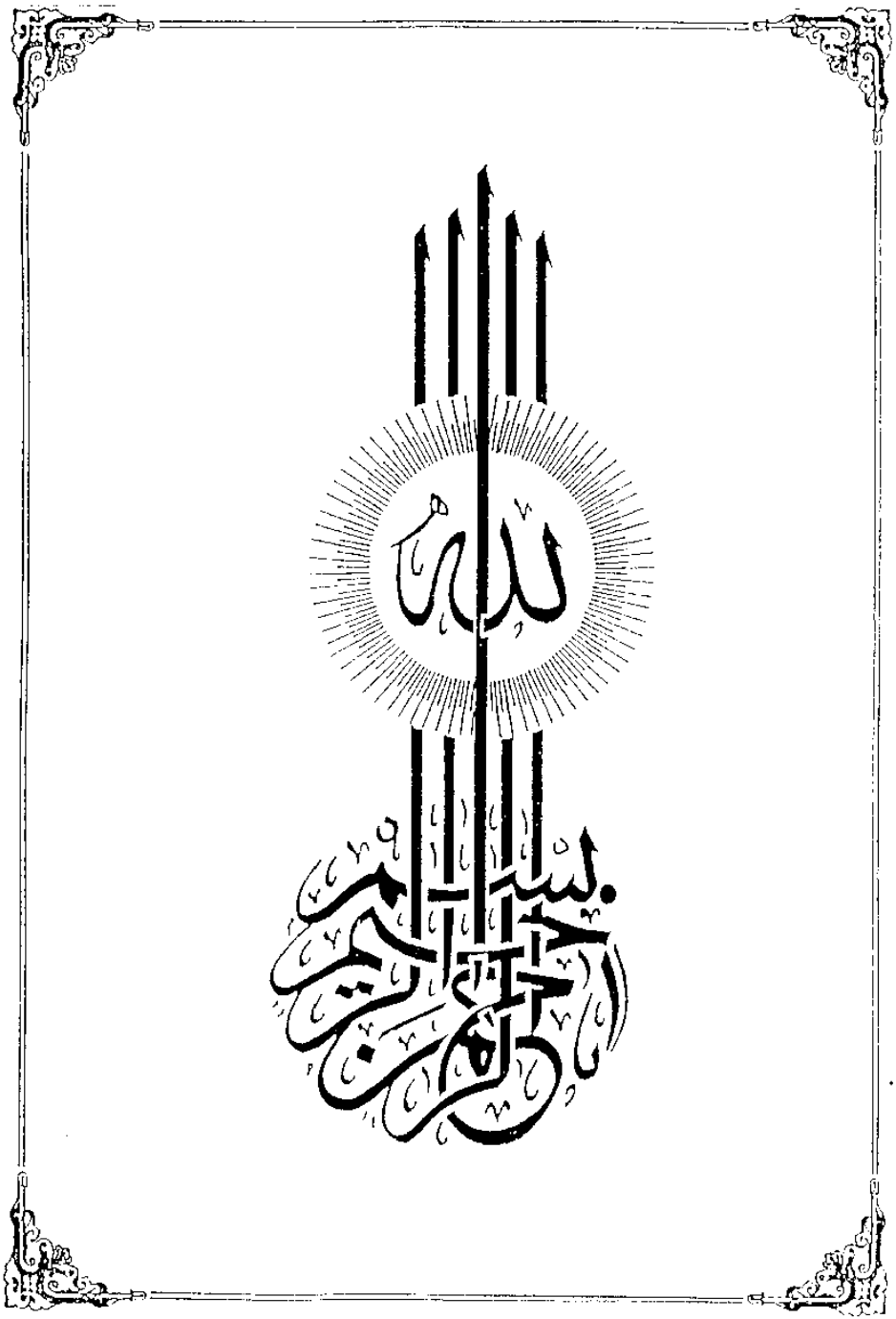
بسم الله الرحمن الرحيم

قالوا سبحانك لا علم لنا إلا ما علمتنا إنك

أنت العليم الحكيم

صدق الله العظيم

سورة البقرة، آية ٣٢



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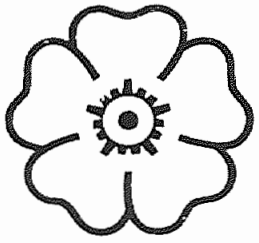

To My Family

LIST OF CONTENTS

	Page
Introduction	1
Pathophysiology of necrotizing enterocolitis	3
Pathogenetic mechanisms in the development of necrotizing enterocolitis	11
Predisposing factors	14
Infectious agents	26
Pathology	30
Diagnosis and classification	32
Non operative treatment	47
Operative treatment	54
Colonic stricture following successful management of necrotizing enterocolitis	63
Summary	67
References	69
Arabic summary	

Introduction

INTRODUCTION

Necrotizing enterocolitis (NEC) is the most common gastrointestinal emergency in the human neonate ***(Kleigman, 1984)***. The incidence increases markedly at lower gestational ages ***(Crissinger, 1987).*** Various factors have been associated with the development of necrotizing enterocolitis, including infectious agents, hypertonic feedings, asphyxia, hypovolemia, hypothermia, and exaggerated shunting reflexes, resulting in irreversible ischemic GIT injury ***(Crissinger, 1987).***

Although the accepted mode of surgical treatment is resection of all obviously necrotic and perforated bowel, exteriorization of the ends, peritoneal lavage and subsequent closure, in many cases the ends of bowel are perfectly viable and may be primary anastomosed in a well-resuscitated baby ***(Kolsloske, 1985).***

Incidence

The incidence of necrotizing enterocolitis (NEC) varies from one nstitution to another and depends on several variables including the iumber and survival of low birth weight infants, the source of patient :eferral and inborn transfer, etc.

The average incidence was 24 cases per 1000 admissions and a case :ate of 1.2 per 1000 live births as estimated by Sweet ***(Sweet, 1980).***

Ryder et al conducted a prospective, 12 centre investigation of ecrotizing enterocolitis and the estimated incidence was 2.4 per 1000 live irths ***(Ryder et al., 1980).***

Kleigman and Fanaroff reported an incidence of necrotizing enterocolitis among infants weighing less than 1.500 gm of 7.9% ***(Kleigman et al., 1981).***

Pathophysiology of necrotizing enterocolitis

PATHOPHYSIOLOHGY OF NECROTIZING ENTEROCOLITIS

Physiology of the gut:

Introduction:

The ultimate function of the gut is to assimilate ingested nutrients and water and make them available for cells through the body by the way of the blood and lymph circulation. The energy necessary to support the digestive, propulsive and transport activities of the bowel is also provided by the circulation.

The physiological process of assimilation and the rapid growth of the fetal and neonatal intestines increases the demands for energy and require an adequate supply of oxygen for optimum aerobic metabolism.

Regulation of circulatory homeostasis in he developing intestine:

Fig. 1 illustrates the possible intrinsic and extrinsic regulation contributing to the control of intestinal blood flow.

Intrinsic regulation:

Normally intestinal blood flow is maintained above a critical limit and changes in response to various functional stimuli ***(Crissinger et al., 1989).***

Metabolic and myogenic mechanisms are considered of highly physiologic significance in explaining the mechanistic basis for intrinsic vascular regulation ***(Banks et al., 1985).***

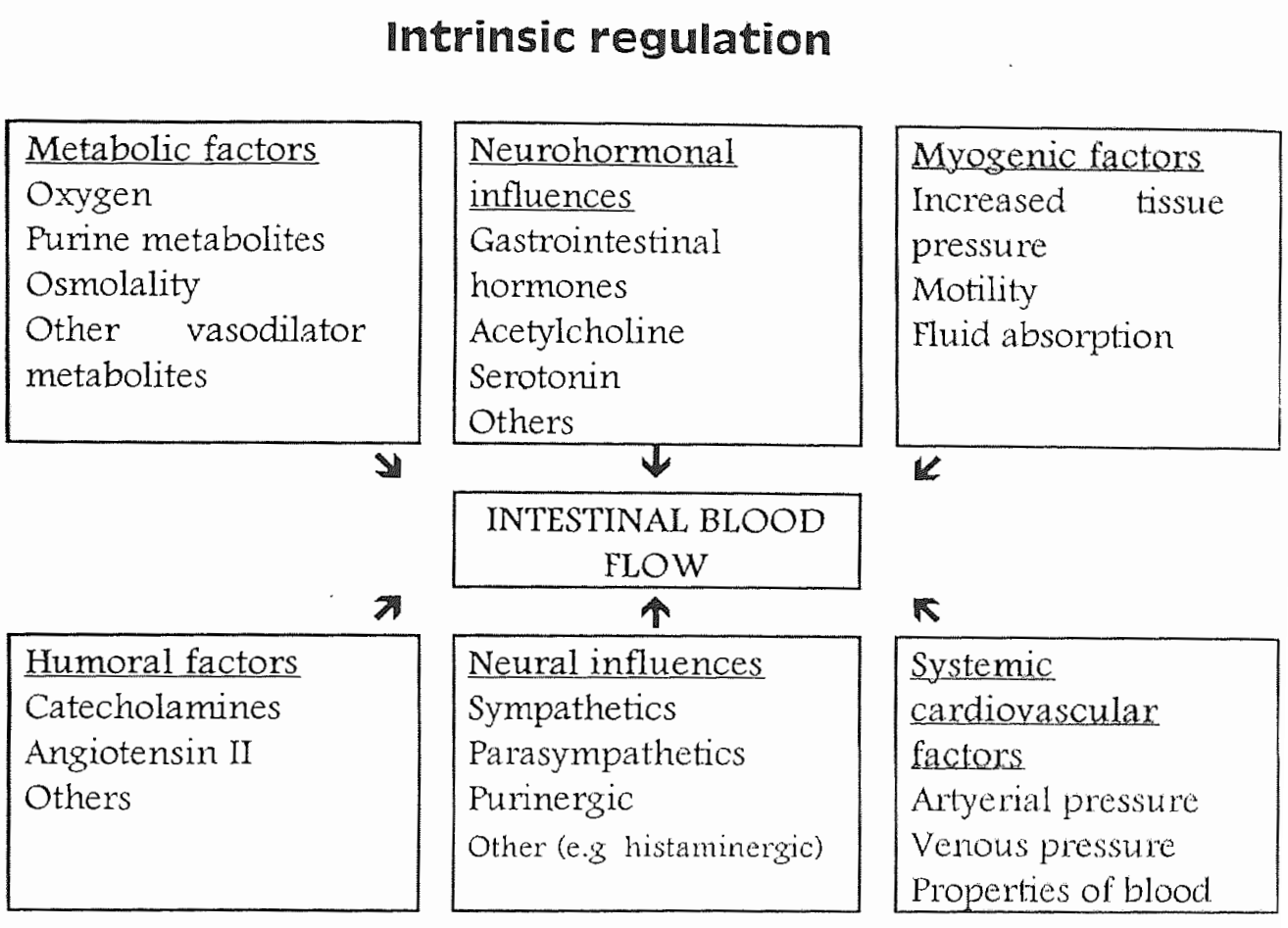

Fig. 1:

Intrinsic and extrinsic mechanisms contributing to the control of intestinal blood flow

The metabolic hypothesis is based on the assumption that vascular resistance and precapillary sphincter tone are linked to the metabolic status of the tissue ***(Crissinger and Granger, 1982).***

A reduction in blood flow causes a decline in tissue oxygen tension (PO_2) and accumulation of metabolites both of which dilate arterioles and relaxed precapillary sphincters ***(Shephered et al., 1984).*** These effects return blood flow to normal.

The myogenic hypothesis is based on the inherent property of vascular smooth muscle to contract in response to a stretch stimulus ***(Folkow et al., 1964).***

Vascular response to arterial pressure alteration:

Pressure flow autoregulation is defined as the ability of the intestine to maintain a constant blood flow during alteration in arterial pressure ***(Johnson et al., 1960).***

The neonatal intestine, despite its high basal O_2 uptake, does not show a correlation between metabolic rate and autoregulatory ability, and pressure flow autoregulation is less intense (or absent) when compared to adult intestine ***(Buckley et al., 1985).***

The efficacy of vascular control of intestinal circulation improves with advancing post natal age. These results indicate that the more immature intestinal circulation is less capable of vasodilatation in response to arterial occlusion.

Vascular response to venous pressure elevation:

Studies revealed that vascular resistance rises in response to venous pressure elevation ***(Johnson et al., 1984).***

Findings that are consistent with a myogenic mechanism, this typical myogenic response can be abolished or reversed after increasing the O_2 demands in intestine of adult animals suggesting that the response to venous pressure elevation is influenced by the metabolic state of the intestine ***(Granger et al., 1980).***

Vascular response to alteration in arterial oxygen concentration:

As metabolic theory predicts arterial hypoxemia increases blood flow and elicits capillary recruitment in adult cats ***(Samvik et al., 1968).***

In the fetal lamb the primary intestinal response to reduction in O_2 supply is an increase in O_2 extraction rather than a change in blood flow ***(Edelestone et al., 1982).***