

## Greater/Lesser Arterial Blood Supply Causing Hyperoxia/Hypoxia Producing Hyperplasia/Hypoplasia of Developing Organ -- Discovery of New Teratogen With Creation of Hyperoxia Hypothesis



### Medical Science

**KEYWORDS :** Teratogenicity, Arterial blood supply, stomach, Liver.

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#### ABSTRACT

*Abdomen of 216 cadavers was dissected in order to study the teratogenic effect of greater and lesser blood supply on development of stomach and liver with gall bladder respectively. Only in one cadaver stomach and left gastric as well as right gastroepiploic arteries were larger in size while liver with gall bladder and cystic as well as hepatic arteries were smaller in size. Gastric hyperplasia and cystohepatic hypoplasia were teratogenic effects of greater blood supply to stomach and lesser arterial blood supply to liver with gall bladder due to hyperoxia and hypoxia respectively because during ontogeny arteries develop prior to organogenesis. Greater or lesser arterial blood supply is a new teratogen discovered by Dr. Keshaw Kumar with creation of hyperoxia hypothesis.*

#### INTRODUCTION

Brent and Franklin (1960)<sup>1</sup>, Franklin and Brent (1964)<sup>2</sup>, Leist and Grauwiler (1972, 1974)<sup>3,8</sup> reported the effects of uterine vessel clamping on the development of rat embryos. Danielsson et al. (1989, 1990)<sup>4,7</sup> described digital defects induced by vasodilating agents and its relationship to decreased uteroplacental blood flow. Caplan and Koutrouspas (1973)<sup>5</sup> described the role of differential vascularization in control of muscle and cartilage development in chick limb. Grabowski (1970)<sup>6</sup> showed embryogenic oxygen deficiency as a physiological approach to analysis of teratological mechanism. Keshaw Kumar (2002)<sup>9</sup> observed teratogenicity of greater and lesser blood supply on gastric and cystohepatic development. However effects of hyperoxia or hypoxia on a developing organ due to which hyperplasia or hypoplasia is caused in a developing organ is not available in literature.

Present study was conducted to create hyperoxia hypothesis and to discover a new teratogen if any.

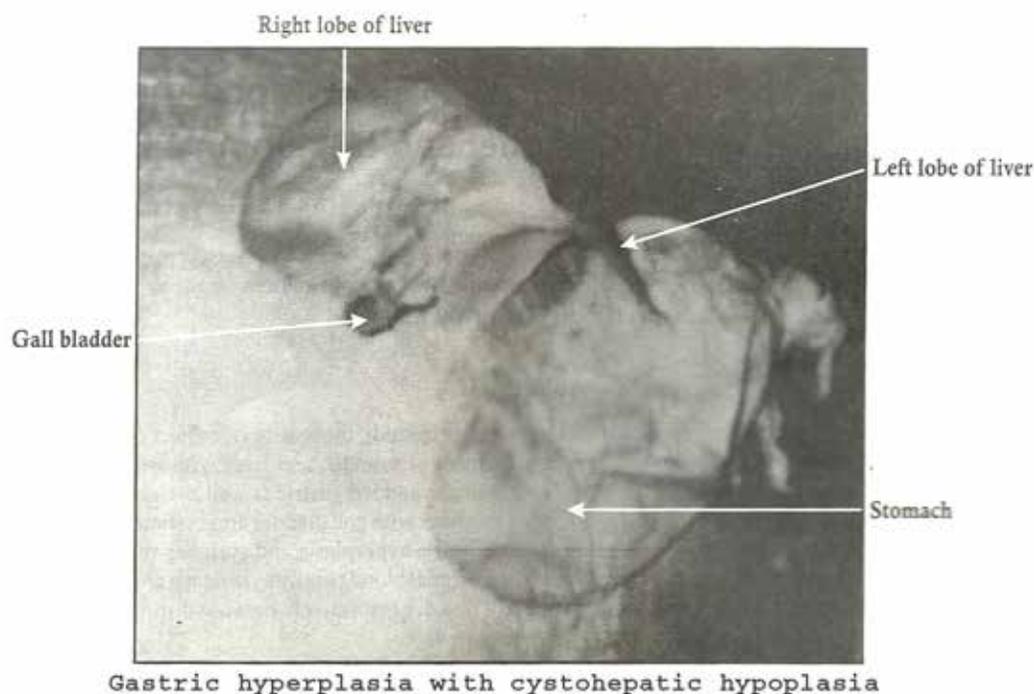
#### MATERIAL AND METHODS

Abdomen of 216 cadavers was dissected to find out anomalies in the arteries supplying stomach, liver and gall bladder and also to observe the teratogenic effects of greater arterial blood supply on gastric development with lesser arterial blood supply on cystohepatic development if any.

#### OBSERVATIONS

While dissecting abdomen in cadavers only in one out of 216 cadavers it was found that stomach was enlarged twice to its normal size having quadrangular shape and lying in left as well as right hypochondriac regions of abdomen. The position and shape of liver was normal but it was reduced to half of its normal size and its left lobe was represented by a fibrous band. Size of quadrate lobe, caudate lobe and gall bladder was also reduced according to reduction in size of right lobe of liver. Histological study of these did not reveal any abnormality.

Superior mesenteric artery gave common hepatic artery which divided into a large gastroduodenal and a small hepatic branch to supply right lobe of liver which was reduced to half of its normal size. From hepatic artery a smaller cystic artery arose to supply gall bladder which was also reduced to half of its normal size. Right gastroepiploic branch arising from gastroduodenal artery was larger sized and superior pancreaticoduodenal artery was normal sized. After giving normal sized splenic artery coeliac trunk continued as a larger left gastric artery which gave a slender branch to fibrosed rudimentary left lobe of liver before reaching the lesser curvature of stomach.



Gastric hyperplasia with cystohepatic hypoplasia

## DISCUSSION

Findings of Grabowski (1970)<sup>6</sup> in chick embryo that hypoxia caused disturbance of fluid and electrolyte balance and an increase in the body fluid was associated with oedema and haemorrhage produces maldevelopment, support the observations made in the present study where under nutrition due to lesser arterial blood supply of the gall bladder and right lobe of liver and least arterial blood supply to left lobe of liver resulted into cystohepatic hypoxia causing hypoplasia of gall bladder as well as right lobe of liver and fibrosis of left lobe of liver proving the oedema syndrome described by Danielsson (1990)<sup>7</sup> and Hypoxia hypothesis put forth by Leist and Grauwiler (1974)<sup>8</sup> who maintained that foetal hypoxia led to a dramatic increase of the diameter and rupture of thin walled vessels of the limb plate, localized oedema, haemorrhage (producing further hypoxia) and finally necrosis of the most distal digital phalanges. Clamping experiments performed by Leist and Grauwiler (1972)<sup>3</sup> in the rat have shown that effects on foetus depend not only on degree of blood flow decrease but also on the duration of the block in blood flow which further support the findings in the present study where persistently and permanently lesser arterial blood supply to gall bladder as well as right lobe of liver and least arterial blood supply to left lobe of liver have caused the hypoplasia of gall bladder as well as right lobe of liver and fibrosis of left lobe of liver because during ontogeny arteries develop prior to organogenesis.

Although the mechanism by which greater arterial blood supply to developing stomach caused gastric hyperplasia is not studied in the present work but it must be related with gastric over nutrition and gastric hyperoxia which is just reverse to foetal hypoxia described by Leist and Grauwiler (1974)<sup>8</sup>. Cystohepatic hypoplasia due to lesser arterial blood supply to developing liver and gall bladder supports hypoxia hypothesis while gastric hyperplasia due to greater arterial blood supply to developing stomach gives rise to hyperoxia hypothesis created by Dr. Keshaw Kumar for the first time with discovery of a new teratogen i.e. greater or lesser arterial blood supply.

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