

**DIFFERENT LINES OF TREATMENT
IN CASES OF OESOPHAGEAL VARICES**

Essay

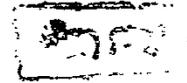
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TABLE OF CONTENTS

INTRODUCTION	1
HISTORICAL REVIEW	3
ANATOMY OF THE OESOPHAGUS AND PORTAL CIRCULATION	6
PHYSIOLOGY AND HAEMODYNAMIC OF PORTAL CIRCULATION	25
PATHOLOGY OF LIVER CIRRHOSIS AND PORTAL HYPERTENSION	42
DIAGNOSIS OF PORTAL HYPERTENSION AND OESOPHAGEAL VARICES	56
DIFFERENT LINES OF TREATMENT IN CASES OF PORTAL HYPERTENSION AND OESOPHAGEAL VARICES	75
DISCUSSION	209
SUMMARY AND CONCLUSION	219
REFERENCES	221
ARABIC SUMMARY	

INTRODUCTION

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Upper gastrointestinal bleeding from oesophageal varices is not considered only a medical problem but also it is a national one, because the mortality rates for medical and/or surgical management of these conditions are still very high.

Precipitation of bleeding episode has been related to two factors; increased pressure within the varix and ulceration due to oesophagitis. 90% of adult patients demonstrate intrahepatic disease (liver cirrhosis) and variceal bleeding is the cause of death in those patients (Grace et al., 1966). It is important to establish diagnosis on an emergency bases by the use of oesophagoscopy or selective angiography (Conn et al., 1966). The therapeutic regimen to control bleeding site without further disturbance of diseased liver cells could be achieved by blood replacement, arrest of the bleeding and supportive measures (Warren et al., 1974). Surgical therapy of portal hypertension is directed primarily toward its complications i.e. haemorrhage from oesophageal varices and to patients with known cirrhosis and with varices have fallen into disrepute and it has further become clear that

(2)

shunt operations while controlling haemorrhage have been followed by disappointingly low long term survival, thus encouraging the development of procedures designed to preserve hepatic blood flow. It is also clear that more work needs to be done correlating the measurements of portal pressure with the selection of an appropriate operation to preserve hepatic blood flow, such as the Warren shunt or selective portasystemic shunting (Maingot, 1979).

HISTORICAL REVIEW

HISTORICAL REVIEW

Oesophageal varices are the important site for the portasystemic collateral pathways as they are liable to catastrophic bleeding that may lead to death. They are usually permanent although fluctuating in size (Sherlock S., 1975).

Before transthoracic operations were possible, only the cervical oesophagus could be treated surgically, and so far as is known. The earliest operations on this organ were limited to cervical oesophagotomy for removal of foreign bodies. By the later part of the 19th century, malignant lesions of the cervical oesophagus become amenable to surgical ablation largely through the efforts of Billroths and Czerny. A pharyngo-oesophageal diverticulum was first resected in 1886, and transabdominal procedures for the relief of oesophageal achalasia were performed in the early 1900s, as were staged reconstructive operations for corrosive stricture and malignant lesions. A successful one stage, transpleural oesophageal resection and oesophago-gastrostomy for carcinoma remained an unattainable goal until Ohawa's report in 1933. In the United States, the first successful oesophago-gastrectomy

(4)

for cancer was performed by Marshall of the Lahey clinic in 1937. There after all aspects of thoracic surgery progressed rapidly. Advances in anaesthesia, blood replacement, and surgical technique, lowered the mortality and morbidity rates of oesophageal surgery to acceptable levels. Despite these major advances it was not until after the world War II, that oesophageal surgeons become aware of the many complexities of oesophageal functions, which are so often distributed by disease and by surgical intervention. Proper patient selection has been facilitated, and the surgeons can now base their operative efforts on sound physiologic grounds. A prerequisite for safe and effective oesophageal surgery is a clear understanding of normal and abnormal oesophageal physiology (Davis Christopher, 1981).

Oesophageal varices are dilated veins in the submucosal layer of the wall of the oesophagus and they are nearly always caused by obstruction of the portal or splenic veins resulting in increased pressure within the portal system. The obstruction may be extrahepatic as occurs with thrombosis of the splenic vein or portal vein (Bickham-Callander, 1955).

(5)

We have to notice that portal hypertension is not an entity in itself rather it is the reflection of a pathologic lesion causing haemodynamic changes in the portal system. The oesophageal varices result from the decompensated cirrhosis in which the blood has been found to pass through the collateral circulation resulting in gradual dilatation and tortuosity of the oesophageal veins, and the rupture of the varices may occur for multiple reasons, resulting in hematemesis which may be fatal (Maingot, 1979).

ANATOMY OF THE OESOPHAGUS
&
PORTAL CIRCULATION

ANATOMY

Anatomy of the oesophagus:

The oesophagus extends from the pharynx above the level of the transverse process of the sixth cervical vertebrae to its entry into the stomach in the abdomen, at the level of the 10th thoracic vertebra. The oesophagus is 23-25 cm. long and its entrance into the stomach is 38-42 cm. from the upper alveolar margin. It is described by the anastomosis as consisting of cervical, thoracic and abdominal part (Last, 1978).

But for surgical purposes, it is enough to divide it into cervical and thoracic segments (Postlethwait, 1979). Physiologically, it is better considered as consisting of an upper $\frac{1}{3}$ and lower $\frac{2}{3}$. These two segments are differently constructed and have different nerve supply (Allison, 1949).

(1) The cervical part of the oesophagus:

It lies in the front of the vertebral column, the longus coli muscle from which it is separated by loose connective tissue of the prevertebral fascia. In front of it, lies the trachea and the thyroid gland with the recurrent laryngeal nerve

(7)

between the oesophagus and trachea. The carotid sheath, containing the common carotid artery and internal jugular vein, lies on each side of the oesophagus (Last, 1978).

(2) The thoracic part:

The anterior relation of the thoracic part of the oesophagus from above downwards are: The trachea (through its entire length), the left main bronchus, the inferior trachibronchial lymph nodes (in front of which lies the right pulmonary artery), the pericardium (in front of which lies the oblique pericardial sinus and the left atrium of the heart), and finally the diaphragm. In the superior mediastinum, the oesophagus is in relation on each side to the mediastinum, the pleura from which it is separated on the left side by the left subclavian artery, the thoracic duct, left vagus nerve and the arch of the aorta, while on the right side of the oesophagus lies the arch of the vena azygos. In the posterior mediastinum, the descending thoracic aorta lies on the left side of the oesophagus then become posterior to it near the diaphragm. The thoracic duct lies at first on the right side of the oesophagus in the posterior mediastinum, then

(8)

it passes behind it to become on the left side in the superior mediastinum. The right intercostal arteries pass between the oesophagus and the vertebral column. The mediastinal pleura touches the oesophagus in places particularly on the right side, where, however, there is a pouch of pleura between the oesophagus and aorta, but nowhere the pleura attached to the oesophagus (Last, 1978).

(3) Anatomy of the lower end of the oesophagus:

It passes through an elliptical oesophageal hiatus which is formed of the skeletal muscles of the diaphragm. There is a separation in the anterior-posterior plane of muscle fibres composing the right crus of the diaphragm. This separation forms a sling anterior to the oesophagus. But there is a less definite reunion of the muscle fibres, posteriorly leaving a V-shaped defect. The hiatus is normally just larger enough to comfortably permit the passage of oesophagus i.e. approximately 2.5 cm. in diameter. The oesophagus passes through the crural tunnel obliquely being directly anterior to the aorta just above the hiatus and lying to the left of the aorta just below the hiatus. Although the oesophageal hiatus is generally made up of the