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A CONTRIBUTION TO THE STUDY OF

PORTAL HYPERTENSION

BY

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TO
MY WIFE
TONY

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PREFACE

THE following contribution to the study of Portal Hypertension was submitted for the Jacksonian Prize of the Royal College of Surgeons for the year 1956. Its format was dictated by the requirements of that ancient prize which stipulate that the essay shall not exceed 75,000 words in length. No attempt has, therefore, been made to summarise the vast amount of scientific work which has recently been published on the hepatic circulation and on liver disease, although I have attempted to relate my clinical experience to the scientific findings.

I am fully conscious of the debt that I owe to the many physicians and surgeons who referred patients, and to my colleagues and assistants. To all I wish to record my most sincere thanks for their help. Individual mention must be made of those who have referred the majority and given me special encouragement: Mr Lawrence Abel, Dr G. P. Baker, Dr Kenneth Black, Dr Ronald Bodley Scott (who referred Case 1, among many others, and provided the initial stimulus), Dr Geoffrey Bourne, Sir Stanford Cade, Dr J. Caplan, Dr Edward Cullinan, Sir Daniel Davies, Dr H. K. Goadby, Sir Neil Hamilton Fairley, Dr Alfred Franklin, Dr John Harman, Dr Philip Harvey, Mr Basil Hume, Dr John Hunt, Dr Kenneth Robertson, Mr Norman Tanner, Dr P. G. Todd and Professor A. W. Woodruff.

Dr Ronald Bowen has given practically all the anaesthetics with his usual skill which has always given me great confidence, even during the most difficult operations.

The development of an efficient method of obtaining portal venograms has been of vital importance and depended very largely on the excellent help given to me by Dr R. A. Kemp Harper and on the unstinted co-operation of Dr George du Boulay and Dr Benjamin Green of St Bartholomew's Hospital and Dr J. J. Stevenson at the Royal Marsden Hospital.

Professor W. V. Mayneord and Dr E. H. Belcher of the Royal Marsden Hospital have enabled me to estimate the speed of flow in the portal vein and to Dr Belcher I would like to express my particular thanks for the number of hours that he has waited in the operating theatre in order to be able to estimate the speed of flow after the construction of a venous anastomosis. Mr H. C. Hodt has shown his expected ingenuity in constructing the double scintillation detector. Dr R. A. Allen of the Atomic Energy Research Establishment at Harwell has estimated for me the content of arsenic and gold in certain cases of cirrhosis hepatis by the fascinating method of irradiation spectrometry.

Dr Hermann Lehmann, at the suggestion of Professor Bruno Mendel, has for five years worked on the assessment of liver function by estimating the pseudocholinesterase content of the serum, information which I have found of great value, and Professor J. W. S. Blacklock and the Research Committee of St Bartholomew's Hospital have enabled this work to be done with the aid of a grant from the Medical Research Council.

Mr I. Cull and Miss J. Akister have prepared tables and illustrations for reproduction. I am especially grateful to Mr N. K. Harrison and his staff in the Photographic Department of St Bartholomew's Hospital and to Miss J. Hunt of the Royal Marsden for providing me with the photographic illustrations.

Miss E. C. Hall, Miss J. Lightfoot and Miss M. Rutherford of St Bartholomew's Hospital have supervised the nursing of the majority of the patients with incomparable skill and kindness. The following sisters of St Bartholomew's Hospital, Miss B. Bartlett, Miss J. Christie, Miss V. Jenkins, Miss A. Mallet, Miss M. Rutherford and Miss J. Souttar, and Miss G. Berdach of the Royal Marsden Hospital, have organised the operating theatres and taken the cases in a way which could not have been bettered. My theatre orderly, Mr Henry Dossett, assisted in devising the hornpipe position which I have found most useful.

Lastly, I would like to express my thanks to Miss Bernice Cattle and Miss Audrey Harby for keeping the records with such care and for helping to prepare this book for publication, and my appreciation of the great consideration shown to me by Mr Charles Macmillan of E. & S. Livingstone Ltd.

ALAN H. HUNT.

London, 1957.

ACKNOWLEDGMENTS

A number of the illustrations have been reproduced before, sometimes in more than one journal, in order to demonstrate different aspects of the subject of portal hypertension. For example, Figure 86 was originally reproduced by Dr du Boulay and Dr Green to show the position of the cassette tunnel on the operating table and then used again by Mr Lawrance, Mr Whiteley and myself to show the hornpipe position. I wish to express my most sincere thanks to Dr L. W. Proger of the Royal College of Surgeons for supplying me with Figure 98, and to the editors of the various journals and books concerned for their permission to reproduce these illustrations.

Proceedings of the Staff Meetings of the Mayo Clinic and Drs Douglas, Baggenstoss and Hollinshead for Fig. 1.

British Journal of Clinical Practice for Figs. 2, 3, 15, 16, 31A, 38A, 48.

American Journal of Roentgenology for Figs. 7, 10, 22A and B.

British Journal of Radiology for Fig. 9.

Lancet for Figs. 14, 20, 86.

Proceedings of the Royal Society of Medicine for Figs. 17, 53, 112.

British Medical Journal for Figs. 37, 66.

Messrs J. & A. Churchill for Fig. 40.

Masson et Cie, Paris, for Figs. 49B, 89, 92.

Journal de l'anatomie et physiologie for Fig. 52.

Appleton-Century-Crofts, New York, for Figs. 81, 97.

CONTENTS

CHAPTER I

INTRODUCTION

THE portal vein exists to carry blood from the stomach, intestines, spleen, and pancreas to the liver. An obstruction to this flow is necessarily unphysiological, whatever it may be. It leads to stagnation of portal blood, a rise of portal venous pressure, and congestion of the organs which drain into the portal tree, particularly the spleen. The body's reaction is to attempt to outflank the obstruction by diverting portal blood into the systemic circulation through communicating vessels. In the cardiac end of the stomach and in the oesophagus these develop into a mass of varicose channels from which devastating haemorrhages are liable to occur. The relief mechanism suddenly thus becomes a serious threat to life.

This, briefly stated, is the condition that has come to be known as portal hypertension. It is not a disease but a state, most commonly caused by a cirrhotic liver. The surgeon is becoming interested in operations which can relieve or palliate this state, and if in so doing he can succeed in improving upon nature by diverting all the portal blood directly into the systemic circulation, it is necessary to find out what effect such apparently unnatural measures have upon the body.

The circulatory state of the portal tree can become so altered that accepted physiological conceptions may have to be modified or discarded in considering the problem. It is necessary for new ideas to be subjected 'to fair trial lest they be dissipated in an atmosphere of tradition and authority' (of Wangensteen). The present report, which analyses 250 personal cases of portal hypertension, has therefore been written with the purpose of examining the new findings and assessing the new methods of treatment. I hope it will help to clarify certain issues on which there is at present no general agreement. I intend that it shall be a statement and demonstration of what has been encountered and what has resulted from treatment, unencumbered by speculative theorems.

The patients were in no way selected. They constitute a consecutive personal series, but there has, of course, been a sifting of material in the mode of reference. For example, cirrhotic patients who had responded favourably to medical treatment alone were not often referred because there was no need for a surgical opinion in this group. It was the more complicated cases with haemorrhage, ascites, or jaundice that were sent for investigation or treatment. Many were desperately ill, some in the very terminal stages of their disease, and they had been sent in the hope that surgery might possibly have something new to offer. None has been omitted from the

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analysis. The follow-up has been complete, thanks to the co-operation of the surviving patients themselves and their doctors, physicians and surgeons. The first patient was seen early in 1947 and the last to be included in the series in June 1956, so that the results have been assessed on an appreciable follow-up, in some cases of many years and never less than six months. Relevant material from patients seen during the latter part of 1956 has also been included in the discussion without in any way prejudicing the analysis.

> 'When animal existence is supported by any other than the usual admirably contrived means, it cannot fail to excite the attention of the philosopher, since it shows to him the powers and resources of nature.'—ABERNETHY, 1789.

CHAPTER II

ANATOMICAL CONSIDERATIONS

THE PORTAL VEIN

KNOWLEDGE of the normal anatomy and of the variants of the portal vein and its tributaries is essential to the proper understanding of portal obstruction, particularly of the extrahepatic type. Excellent recent studies have been published by Gilfillan; Douglass, Baggenstoss, and Hollinshead; Falconer and Griffiths; and Child. The accompanying diagram of the normal anatomy (Fig. 1) is generally accepted as the usual arrangement. All workers

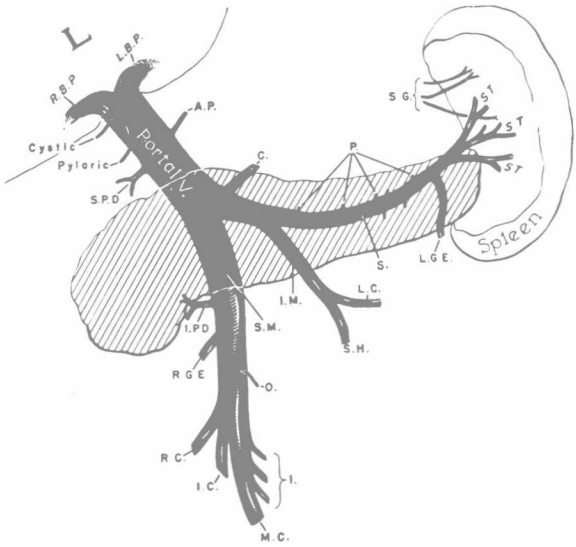


FIG. 1
The portal vein and its tributaries.
(From Douglas, Baggenstoss & Hollinshead.)

emphasise the great variations that may be encountered within the 'normal' range and how these variations may affect the development and treatment of the abnormal state. As examples, the left gastric, or coronary, vein may enter splenic or portal and its position may have a significant bearing on the development of oesophageal varices; radicles from gallbladder, duodenum, pancreas, and stomach may or may not enter the trunk of the portal vein itself. The position, length, diameter, tortuosity, and lie of the portal vein vary from case to case (Rousselot, 1953). Abernethy in 1789 even described a portal vein opening directly into the inferior vena cava in a case of trans-

position of viscera. Duplication and anterior placement of the portal vein have also been described.

The operating surgeon must be able to translate academic anatomical knowledge into a demonstration of projection anatomy so that he may know

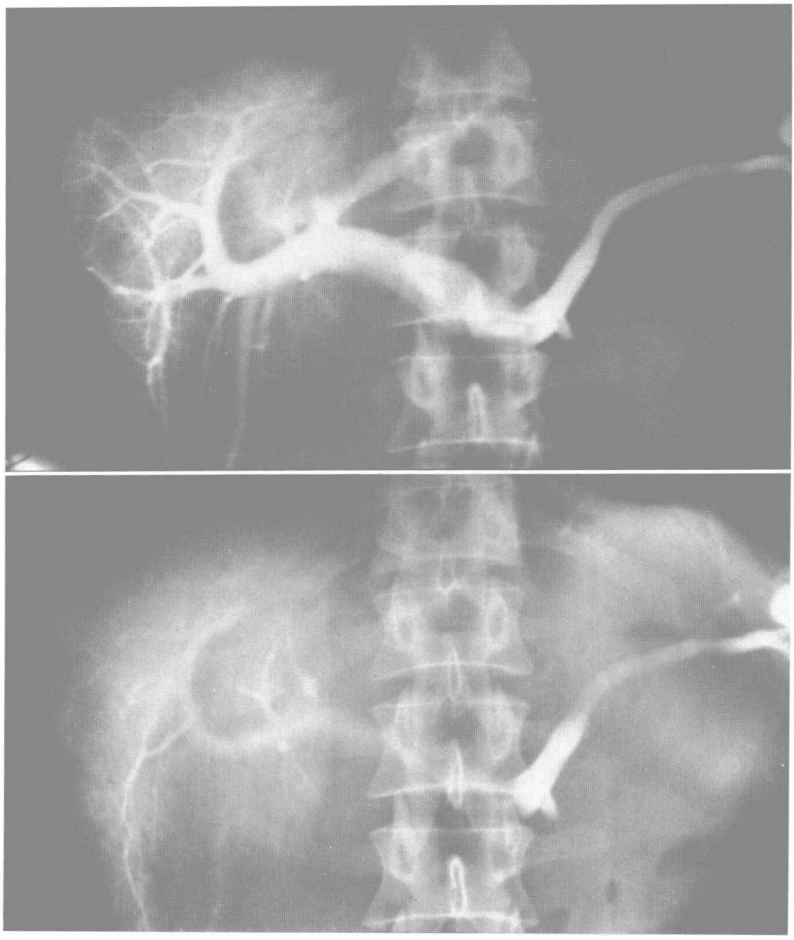


FIG. 2

Normal splenic venogram. Thrombocytopenic purpura. Portal venous pressure 80 mm. water. Diffuse distribution of blood throughout liver.

the exact state of the portal vein in each case. To him only the anatomy of the individual is important, and he should know it if possible before beginning the dissection. Portal and splenic venography are the radiographic methods employed, prior to palpation and dissection. The accompanying figures (2, 3) are two examples of unimpeded normal portal circulation and provide the standard by which subsequent illustrations may be judged. The

blood flows directly and without diversion from splenic or superior mesenteric through the portal vein into the liver.

Definition of the portal vein is not necessarily complete by either method owing to displacement of blood containing the radio-opaque diodone by blood from another part of the portal tree. This normal ' streamlining ' or laminar flow (Helps and McDonald, 1954 ; Dreyer, 1954) is well shown in Fig. 3. The speed of flow, about 9 cm. per second, is too slow for there to be more than partial mixture, so the splenic blood stream is either pushed aside, split, or rotated by the blood coming from the superior mesenteric vein. Laminar

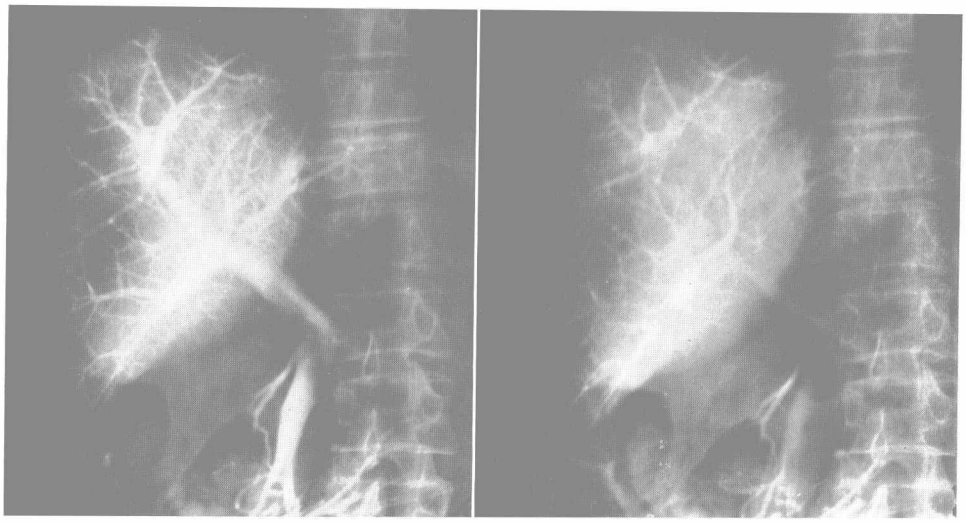


FIG. 3

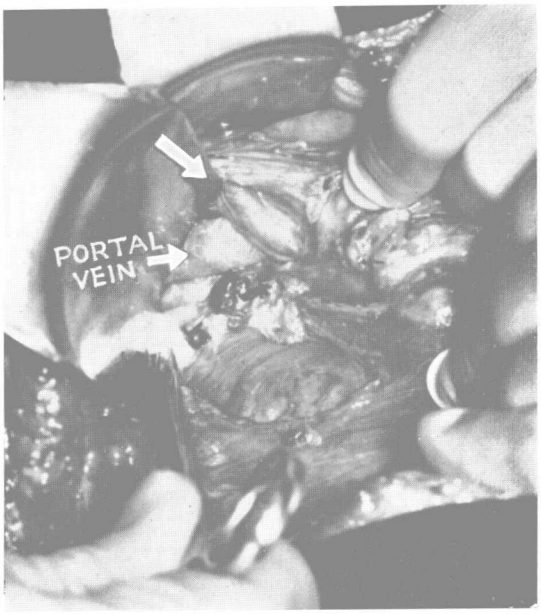
Portal venogram in a case of gallstones. To show the normal liver pattern and a rapid though laminated or streamlined flow through the portal vein. Speed of flow 8·0 cm. per second. Portal venous pressure 55 mm. water.

flow can easily be confused with thrombosis, but the composite picture obtained by both methods and by serial radiographs gives accurate information. Within the liver the blood is usually evenly distributed, and selective flow from one group of portal radicles to any particular segment of the liver has not been demonstrated. Raven came to the same conclusion using slightly different methods.

THE HEPATIC ARTERY

The hepatic artery normally arises from the coeliac axis and divides into two branches which lie in front of the corresponding branches of the portal vein as they pass up to the liver. The unusual must again be anticipated. An abnormality which may seriously interfere with the dissection of the portal

vein has been encountered four times. The hepatic artery itself, or a large branch, has been found hooking round the right border of the vein (Fig. 4). On one occasion the operation had to be abandoned. On another it made the placing of the anastomosis faulty so that the portal vein was kinked over the artery, the shunt became thrombosed and the patient died (Case No. 108). On the third it was just possible to separate hepatic artery and portal vein completely so that an excellent and uninterrupted shunt could be constructed. Unfortunately the man died post-operatively of liver failure (Case No. 245).



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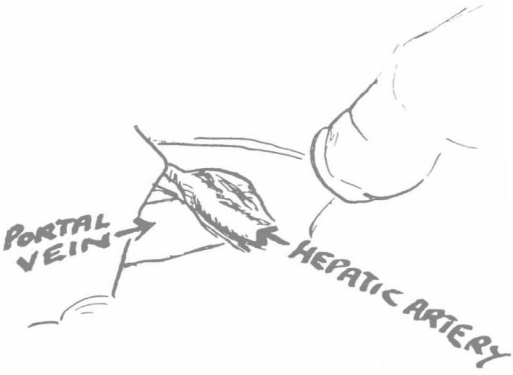

B

FIG. 4

Patient No. 108. Abnormally placed hepatic artery which can be seen (arrowed) hooking round the portal vein on its posterior, right, and then anterior aspects. This and the proximity of the pancreas rendered porta-caval anastomosis impossible.

At postmortem the shunt was found to be satisfactory. The artery was the common hepatic, intact and undamaged, which arose from the superior mesenteric (an anomaly which has been described by Henle, 1872, and by Browne, 1940). The fourth patient was similar, but he fortunately made an excellent recovery from his porta-caval anastomosis and will, I hope, give us no opportunity for further dissections.

Other anomalous origins (see Henle, 1872) are encountered from time to time, such as that of the left hepatic artery arising from the left gastric, which becomes of special importance in considering the operation of hepatic arterial ligature. The purpose of this operation is to cut off the whole arterial supply to the liver, and this will mean an extensive search to be sure that no major vessel is missed.

THE INFERIOR VENA CAVA

The successful construction of a direct porta-caval anastomosis depends to some extent on clean dissection of the vena cava right up to and even behind the caudate lobe of the liver. At and just below this point there are two, three, or even four veins draining into the vena cava from the liver itself, the capsule of liver, or other surrounding structures. If they are torn they cause troublesome bleeding; if they are recognised they can easily and bloodlessly be divided between ligatures.

On one occasion the inferior vena cava was found to be double. It compelled a wider dissection but did not interfere with the construction of an anastomosis.

THE RENAL ARTERY AND VEIN

The single renal artery usually lies behind the vein and allows the successful completion of an end-to-side lieno-renal anastomosis. The artery, however, is often multiple (from the segmental origin of the organ) and important branches may lie in front of the renal vein, sometimes making smooth coaptation with the splenic impossible. If they are small they can occasionally be sacrificed, but only after trial clamping for about ten minutes with a Blalock clamp. If they are large it is better to sacrifice the kidney, strip the vein of its enclosing arteries, and make an end-to-end anastomosis with the splenic vein.

Anomalies in the renal vein, though much less common than those of the artery, are sometimes found and can be circumvented with a little ingenuity (Case No. 219).

CHAPTER III

THE CAUSES OF EXTRA AND INTRAHEPATIC PORTAL OBSTRUCTION

GENERAL CONSIDERATIONS

ANATOMISTS have been aware for more than 120 years of the vascular communications that exist between the portal and systemic venous circulations and they have, so far as the restricted scope of the dissection of a few cadavers allowed, recognised a condition similar to what we now term portal hypertension. The establishment during the present century of special centres for the study and treatment of difficult diseases has enabled physicians, surgeons, pathologists, physiologists, anatomists, and radiologists to co-operate in elucidating facts on an altogether more expansive scale. Allen O. Whipple's Spleen Clinic at the Presbyterian Hospital, New York, is such a centre, and from there have originated many of the current ideas on portal hypertension. Rousselot, for example, measured the pressure in the splenic vein on the operating table and was able to define, in 1936 and 1940, the causes of extrahepatic portal obstruction. Out of this grew the classification (Whipple, 1945) that has served well during the short period of ten years that has been available for most of us in this country for the scientific study of ' civilian ' surgery.

There are two main types of obstruction : the extrahepatic, in which the block is beyond the liver and interferes with the flow of blood to that organ (which is normal in other respects) ; and the intrahepatic, in which the fibrosis of the liver itself constitutes the obstruction. In addition, portal hypertension can be caused by an interference in the return of blood from the liver to the heart, as in obstructions to the hepatic veins and the thoracic part of the inferior vena cava (the Budd-Chiari syndrome) and in constrictive pericarditis.

How was it that the early anatomical studies did not lead sooner to the classification propounded by Whipple? One explanation is that the subject is of such complexity, involving so many different organs in a jumble of striking and inconstant symptoms and signs, that a false theory put clinicians and pathologists on to a wrong idea. In 1894 Guido Banti of Florence, deceived by the enormous spleen which made itself evident as the initial abnormality in certain cases of progressive cirrhosis hepatis, described a disease which was named after him. The spleen, according to Banti, was primarily at fault, and the infection, whatever it was, spread from spleen to liver and gave rise to a disease which ' progresses slowly but it inexorably