THE HEPATIC VEINS IN MAN AND THEIR SPHINCTER MECHANISMS

By JAMES B. GIBSON

Department of Pathology, Queen's University, Belfast

The structure of the hepatic veins differs from that of most other veins and resembles that of the hepatic portion of the vena cava, for the muscle is relatively well developed and lies chiefly in the adventitia. This arrangement of the muscle may permit the veins to play a part in controlling the hepatic circulation. Folds are present where the ostia join the cava and a throttle mechanism has been postulated at this level. The older views on this ostial sphincter have been reviewed by Franklin (1937). At a lower level in the hepatic-venous tree, Deysach (1941) described in animals a sluice mechanism which altered the venous drainage under certain experimental stimuli. Those who have studied the hepatic circulation in animal livers by transillumination (e.g. Knisely, Bloch & Warner, 1948; Seneviratne, 1949–50) have referred to a contractile power on the part of the sinusoids especially at their terminations in the central venules (outlet sphincters).

This paper deals with the anatomical basis for sphincter action in the hepatic-venous system in man and its possible function. It reports personal observations on the gross and microscopical structure of the veins and their radicles. Useful accounts of the microscopical structure of the hepatic veins are given by Pfuhl (1922), by Miyake (1928–30) and by Tischendorf (1939), but the subject is confused by a diversity of terminology and by some differences in the reported facts. A revised terminology is proposed to conform to the observed facts. The general arrangement of the large veins and their ostia is well known and has been described by Elias & Petty (1952) and by Gans (1955), and the drainage territories of the main hepatic veins have been defined by Knopp (1953).

MATERIAL AND METHODS

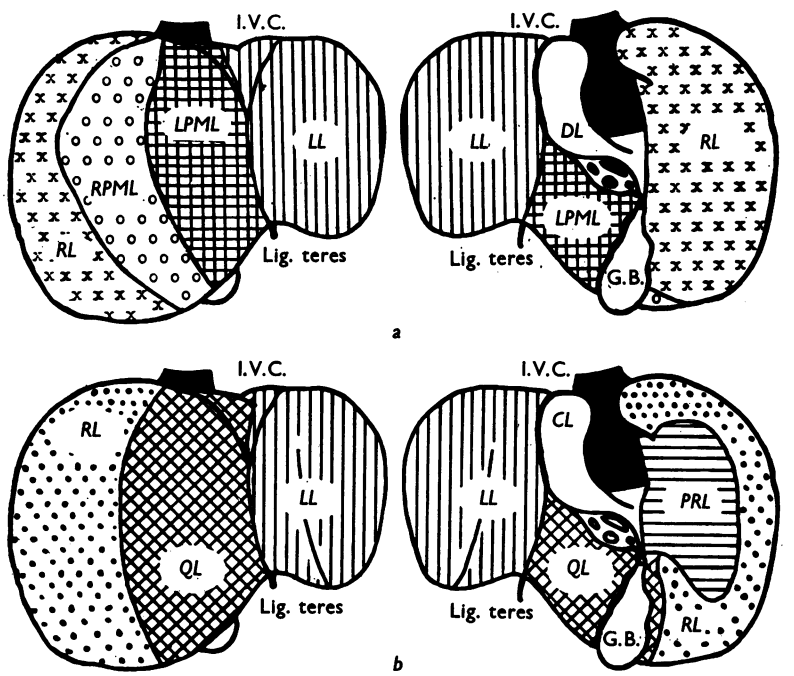
The gross characters of the hepatic veins were studied in approximately 1000 routine autopsies. In addition, in fourteen normal livers obtained at post mortems, retrograde injections were made into the main hepatic veins with plaster of Paris (1), plastic (1), neoprene latex (5) and warm coloured gelatine (7). Injection pressures of 5–22 mm. Hg were used and the livers were washed out through the cannulae for about 15 min. before injection; longer washing produced swelling of the parenchyma. Except in the case of the plaster-of-Paris cast, the relative positions of the veins were preserved by supporting the livers in warm water or saline during injection. The livers were supported and fixed overnight in cold formalin which was acidified for the latex casts. The livers injected with gelatine were sectioned by hand in horizontal planes and the courses and territories of the injected vessels were plotted in relation to the outlines of the livers. For the other preparations liver tissue was removed by dissection and by treatment with acid to leave vascular casts

which were examined under various magnifications. Camera lucida drawings were made of appropriate portions. About 100 blocks selected from normal livers in the autopsy material were studied histologically by a variety of staining methods for elements such as muscle (picro-Mallory method of Lendrum & McFarlane, 1940), elastica, reticulin and nerve. The measurements of the diameters of the veins given in this paper are for the dilated state unless otherwise specified, and were made either directly on casts or on the images of casts projected on the camera lucida. Measurements were made also on paraffin sections and these have been increased by 20 % to allow for shrinkage, a proportion arrived at by a comparison of corresponding vessels in casts and sections.

OBSERVATIONS

Ostia and large hepatic veins

The territories of drainage of these vessels are shown in Text-fig. 1*b*. The right ostium measures about 15 mm. in diameter and the left ostium about 13 mm. The only noteworthy variations in the ostia lie in the extent to which the terminations of the v. hepatica sinistra (*HS*) and the v. hepatica media (*HM*) combine to form the left ostium. Completely separate openings of the veins in the cava were not



Text-fig. 1*a*. The lobation of the liver based on portal-venous supply, according to Gans (1955), but simplified. *RL*, right lobe; *RPML*, right paramedian lobe; *LL*, left lobe; *LPML*, left paramedian lobe; *DL*, dorsal lobes.

Text-fig. 1*b*. The lobation of the liver based on hepatic-venous drainage as worked out in this study. *RL*, right lobe (vein *HD*); *CL*, caudate lobe and process; *QL*, quadrate lobe (vein *HM*); *PRL*, paracaval portion of right lobe; *LL*, left lobe (vein *HS*).

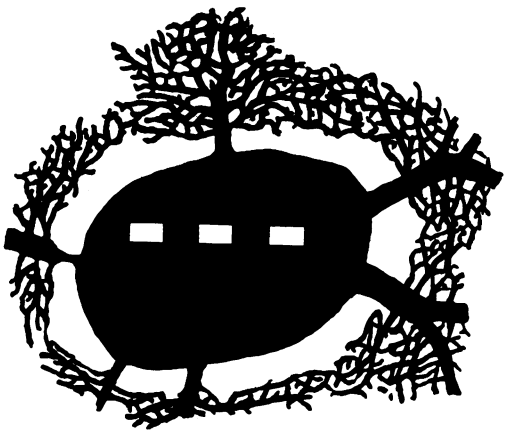
encountered. There is considerable variation in the exact courses and tributaries of the three main hepatic veins, but each of them (*HD*, *HS* and *HM*) regularly drains a certain portion of the liver. The exact boundaries of these portions vary by 1 or 2 cm. from one liver to another. The areas are not the same as those defined by the portal-venous supply. The hepatic-venous radicles alternate with the portal-venous branches at all levels in the liver and with few exceptions, such as the left lobe, the respective territories overlap (Text-figs. 1*a*, *b*). It is noteworthy that the paracaval portion of the right lobe (*PRL* in Text-fig. 1*b*) and the caudate lobe and process are drained by several small ostia located in the cava distal to the two main ostia.

The thickest part of the walls of the ostia and hepatic veins is the muscular adventitia. There is no discrete external elastic lamina or medial coat, but internal to the adventitia there is a zone of moderate thickness which contains fine, circularly arranged smooth-muscle fibres intermingled with elastic and collagenous fibres. In the ostia the elastic fibres of the internal part of this zone are often condensed to form an internal elastic lamina between the circular fibromuscular layer and the endothelial lining, but the lamina is not regularly visible in the hepatic veins. The adventitial muscle is disposed in longitudinal bundles which cause a ridging of the lining of the veins that may be visible to the naked eye. The bundles are bound together by the collagenous and elastic tissue of the internal collagenous layer of the adventitia. External to the longitudinal muscle layer lies a zone of loose texture containing lymphatics, arteries and nerves. The nerve bundles are small and contain fine non-myelinated fibres of autonomic post-ganglionic type. Nerves are most numerous round the ostia but can be found readily in the walls of veins as small as 5 mm. Lymphatics can sometimes be identified in the walls of veins as small as 2 mm. diameter. The outermost layer of the adventitia is composed of collagen and elastic fibres densely adherent to the liver and is in fact a part of the liver capsule. Small bile ducts can sometimes be found in this layer in otherwise normal livers.

Because of the loose texture of the tissue between the internal and external collagenous layers of the adventitia, the inner parts of the ostia and the main hepatic veins are not firmly fixed to the liver substance. This can be appreciated better in fresh than in fixed material. The terminal part of the left hepatic vein lies on the superior surface of the liver and is free to contract, but there is also a considerable degree of play round the lips of both the main ostia. At these lips the muscle coat projects inwards (Pl. 1, figs. 1, 2*a*, *b*) in folds that are better formed at the lower edge of the opening than at the upper. The folds contain two separate layers of muscle, one contributed by the caval and the other by the ostial wall: a few of the muscle fibres are looped round the ostial opening to form a sling (Pl. 1, fig. 2*b*). Between the layers the zone of loose texture is particularly wide, and fat may be present in it. These arrangements constitute the ostial sphincters. Asymmetrical bundles of muscle as in the adrenal and renal throttle veins are not present. The muscle layer of the main ostia is about 0·3 mm. thick, i.e. about half the thickness of that of the caval wall. There is an equivalent abrupt reduction in muscularity where the main radicles join the ostia distally, and folds similar to those at the proximal ends of the ostia are present but they are smaller and less mobile.

The medium-sized and small hepatic veins

The structure of the medium-sized and small hepatic veins is similar to that of the larger vessels, but is progressively simplified in the diminishing orders and the veins are more closely adherent to the liver substance. There are no valves in the hepatic-venous system. Hepatic veins contain more muscle than do intra-hepatic portal branches of similar size. The muscle is progressively reduced, and in veins of of less than 1 mm. diameter the adventitia is composed chiefly of collagenous tissue (Pl. 2, figs. 5, 6). In picro-Mallory stained sections, longitudinal adventitial muscle fibres are rarely found in veins smaller than 1 mm., but circular fibres are often visible in the inner layer at that level and can be distinguished in the smallest of the hepatic veins (400 μ). The hepatic veins increase gradually in calibre and join each other and larger trunks in many different ways. The angles of junction tend to



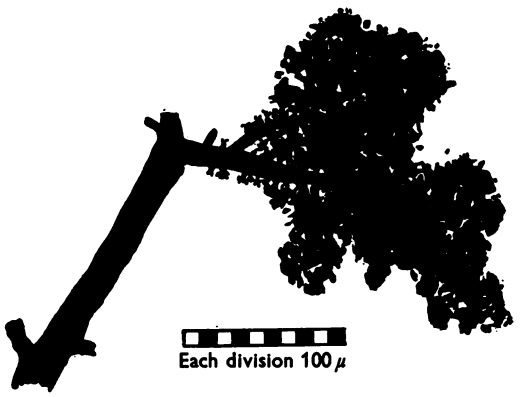
Text-fig. 2. Camera-lucida drawing of part of a thick section of a neoprene-latex cast from an adult human liver. Central venules of various shapes and sizes join an hepatic vein of the seventh order at approximately right angles. Sinusoids do not enter the hepatic vein directly. The central venule on the left is constricted where it enters the larger vessel. Each division of scale (centre) represents 100 μ.

become less acute as the vessels are traced down; they approach 90° in the case of the smallest hepatic veins. A feature of the ramification that can readily be appreciated in casts is the fact that many small tributaries—intercalated veins and central venules—join the hepatic veins directly. These junctions are frequent in the case of the smaller orders of hepatic veins, but are also found in medium-sized vessels (Pl. 1, fig. 3). The small vessels enter the larger ones at right angles in a radial fashion (Text-fig. 2). Some are narrowed at their junctions, apparently because their thin walls have been compressed there by contraction of the muscle in the walls of the hepatic veins. These narrowings, termed 'junctional constrictions' in this paper, affect the central venules more than the intercalated veins. They can be seen readily in paraffin sections (Pl. 2, figs. 4–6).

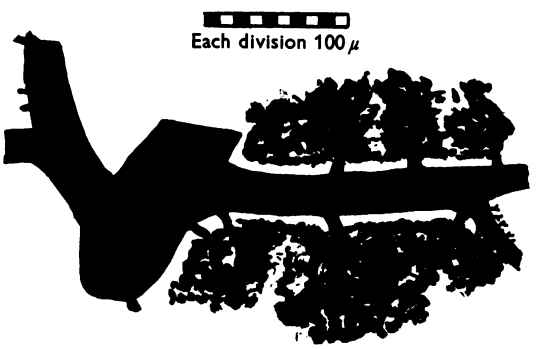
The small veins

For reasons discussed below, the term 'intercalated vein' is applied here to all veins of 100–350 μ diameter. By ordinary histological methods muscle cannot be

demonstrated in the intercalated veins. The vein wall is collagenous and may appear asymmetrical in cross-section; it contains small numbers of elastic fibres arranged circularly. Central venules (40–100 μ) have thin collagenous walls, and elastic fibres are rarely found in them in normal livers. Muscle fibres cannot be demonstrated histologically at any point in the central venules and in particular not at the entries of sinusoids where Knisely *et al.* (1948) located their outlet sphincters in animals.



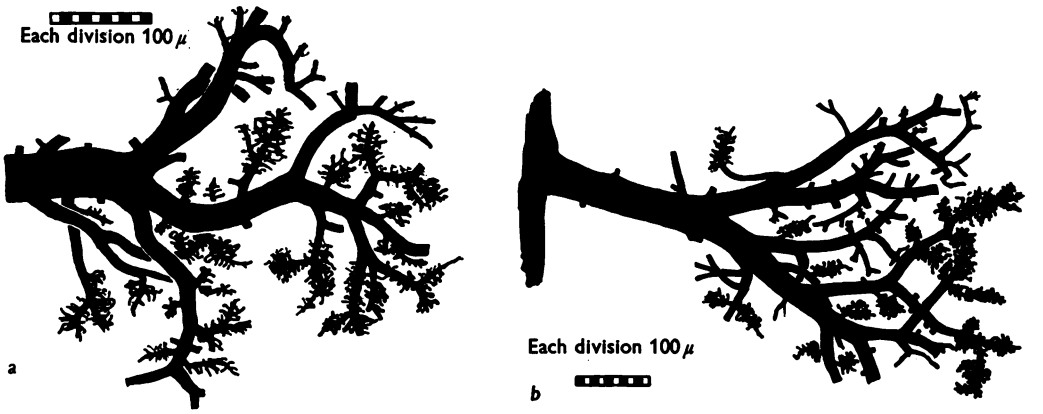
Text-fig. 3. Camera-lucida drawing of a 'massive lobule' of Pfuhl (1922) and its central venule joining a long intercalated vein. Sinusoids do not enter the latter. From a peripheral area of a neoprene cast of the hepatic veins of a 3-day-old infant.



Text-fig. 4. Camera-lucida drawing of central venules draining lobules of various sizes directly into an intercalated vein which does not receive any sinusoids. Two central venules join an hepatic vein of the seventh order which is the largest vessel shown. From a central area of the same cast as Text-fig. 3.

In some portions of the neoprene casts the injection mass flowed into the ends of the sinusoids. The sinusoids, which were identified microscopically in tease preparations and in thick frozen sections of the casts, terminated only in central venules (Text-figs. 2, 5*a*, *b*). The sinusoids in the casts formed a continuous spongework which was separated from the intercalated and hepatic veins by a space about 100 μ wide. The veins were entirely free of sinusoidal insertions although joined to the anastomosing mass of sinusoids at frequent intervals by hair-like central venules (Text-figs. 2, 4). Examination of paraffin sections confirms that sinusoids terminate exclusively in central venules. There is no channel which can be interpreted as a

vessel intermediate between sinusoids and central venules, but the composition of the latter is diverse and irregular and the largest venules are made up by the junction of smaller ones (Text-fig. 3). These united venules continue to receive sinusoids till close to their terminations in hepatic or in intercalated veins. Many intercalated veins throughout the liver drain into other and larger intercalated veins (Text-figs. 5*a*, *b*) and not directly into hepatic veins, and such large intercalated veins probably correspond to the 'collecting veins' of other authors. They are not, however, characterized by any difference in structure from the smaller intercalated veins which might justify the use of a separate name.



Text-fig. 5. Tease preparations of portions of a neoprene cast of the hepatic veins of an adult. In some instances the sinusoids have been traced in round the central venules. In (*a*) from a central area, an hepatic vein of the eighth order is made up chiefly of intercalated veins of different sizes. Central venules join both types of vein. In (*b*) from a peripheral portion of the same cast, two intercalated veins unite to form an hepatic vein of the eighth order, which in turn joins a larger vein. The intercalated veins are made up by smaller intercalated veins and by central venules. The intercalated veins tend to be longer in this part of the cast and the junctions are more acute.

In the casts, certain small differences between the superficial and the central portions of the liver were noted in the arrangement of the small veins (Text-figs. 5*a*, *b*). In the central parts, a large proportion of the central venules, probably the majority, terminated in hepatic veins, whereas in superficial areas a higher proportion ended in intercalated veins. In the superficial zones, the central venules were more irregular in size and the intercalated veins were often longer and their junctions more acute (Text-figs. 5*a*, *b*). From the above descriptions it is clear that blood leaving a central venule often reaches an hepatic vein, and sometimes an hepatic vein of large size, without having traversed an intercalated vein. In other instances, especially in the periphery, it passes through an intercalated vein before reaching an hepatic vein.

Anastomoses between different venous territories

When warm gelatine of a different colour was injected into each of the major ostia, the mass often entered the sinusoids. The colours sometimes mingled at the limits of the venous territories shown in Text-fig. 1*b* over as much as 2–3 cm. This spread

apparently took place through the sinusoids which form a continuous anastomotic network throughout the liver, and not through larger vessels. Spread was limited because of the small size of the vessels involved but was usually sufficient to fill the sinusoids in the paracaval portion of the right lobe (*PRL* in Text-fig. 1*b*); the proper veins of this area were usually not injected. Evidence was not found of any regular anastomoses between central venules, intercalated veins or hepatic veins within the liver. After full maceration the neoprene casts separated easily into discrete shrublike masses comprising the smaller tributaries of larger veins (Pl. 2, fig. 7). In the occasional instances in which anastomoses were found deep in the liver between small hepatic or intercalated veins, they were associated with other minor irregularities and so are thought to be due to pathological processes. In the earlier stages of cleaning the casts, however, and on the surfaces of livers injected with coloured gelatine, scanty and irregular but definite networks of veins of the size of small hepatic or intercalated veins were usually visible. This capsular network was probably accentuated by the pathological thickening of the capsule which is common particularly on the anterior surface of the liver, but it appeared to be a normal structure and it was found in the single instance of a neonatal liver used in the series. No material difference in respect of the features described in this paper was noted between this cast and the others made from adult livers.

DISCUSSION

Terminology

The *hepatic veins* comprise the section of the venous system between the intercalated veins and the ostia and their structure is uniform in plan. The veins increase in calibre gradually by the junction of radicles, but are conventionally divided into orders. If we suppose that each order measures 1·6 times the diameter of the one below, we find that there are eight orders from the smallest (400 μ) to the largest hepatic veins (1 cm.). There is a sharp transition at the 400 μ level from the hepatic veins which are partially muscular to the small veins below them which have walls without muscle fibres. These small veins are of two sorts, intercalated veins and central venules. No important alteration in structure or arrangement was noted in the present study at any level between the central venules and the hepatic veins, and all veins at this level have been described as *intercalated veins* in this paper and the term 'collecting vein' has not been used. The intercalated veins are characterized by the absence from their walls of muscle demonstrable by histological techniques and by the absence of sinusoidal tributaries. The alternative term, 'sublobular vein', seems less apt and has been used by Deysach (1941) to describe vessels in his experimental animals which are clearly hepatic veins. Deysach (1941) reported what appeared to be new channels of drainage for the sinusoids, simple endothelial tubes which he called 'small sluice channels' emptying into sublobular veins. These channels were widely opened by adrenalin and closed by parasympathomimetic drugs. By measurement of the vessels he illustrated, however, it can be seen that the 'small sluice channels' are central venules which can be contracted at their points of entry into hepatic veins (Deysach's muscular sublobulars) in the same way as the junctional constrictions described in this paper. Deysach's 'large sluice channels' were intercalated veins.

The *central venule* is clearly a distinct type of vessel and the present study confirms the findings of Elias & Popper (1955) that they are the only vessels in which sinusoids terminate in man. They are not, however, uniform in size and vary from 40 to 100 μ, the largest venules being made up of smaller ones. Pfuhl (1922) also noted this irregularity in the outflow tracts of the 3-dimensional sinusoidal network. It tends to compensate for inequalities in the venous drainage of a lobular system deriving its blood supply from portal tracts. In parallel with this variation in the central venules, the terminations of the sinusoids are also irregular, for sinusoids enter the venules sometimes singly and sometimes in groups.

Structural basis for the distribution of some hepatic lesions

In cases of hepatic-venous occlusions, areas of liver tissue which correspond to the territories shown in Text-fig. 1*b* are found to be congested or atrophied, while the remainder of the liver is spared or has undergone hyperplasia. For practical purposes the lobes shown in Text-fig. 1*b* are independent in their venous drainage. In the present study, anastomoses other than sinusoidal and subcapsular anastomoses were found only rarely and irregularly between individual hepatic veins (Pl. 2, fig. 7), and this is in keeping with the findings of Tori (1955) and of Goldsmith & Woodburne (1957), though at variance with those of Mall (1906), Elias & Petty (1952) and Gans (1955) who reported frequent anastomoses between small hepatic veins. Structural differences between the superficial and central portions of the liver can often be seen in morbid processes (Walker, 1958) and they may be due, at least in part, to the differences in venous drainage between these areas (Text-figs. 5*a*, *b*) and to the existence of venous anastomoses in the liver capsule. A difference in reaction between the superficial and deep parts of the liver cannot be ascribed in man to the operation of the vascular-shunt system described by Daniel & Pritchard (1951) in animals. This short-circuit route for blood flow through the liver depends on the close spatial relationship of large portal to large hepatic veins throughout their courses and on their inter-communication through small branches and radicles located at the proximal ends of the large vessels. Although small radicles join hepatic veins of various orders in man (Pl. 1, fig. 3), the courses of the large portal and hepatic veins are not parallel in the unitary human liver as they are in the multilobed livers of animals. The two sets of vessels run at approximately right angles (Elias & Petty, 1952) and the ostia are widely separated from the porta so that there is no common vascular hilum in man.

Pathological patterns on a finer scale can also be explained on an anatomical basis. Elias & Popper (1955) have shown how the regular pattern of chronic venous congestion in man depends on the fact that sinusoids terminate exclusively in central venules in the human liver. In cardiac cirrhosis a significant amount of the new fibrous tissue is formed in the parenchyma in the immediate neighbourhood of the hepatic veins (Moschcowitz, 1952) and this seems to be due to the fact that many central venules join hepatic veins directly (Pl. 1, fig. 3). The lobules served by these venules are exposed more directly than other lobules to the elevated pressure in the larger veins because the junctional constrictions of their central venules are held open in the walls of the dilated hepatic veins.

Hepatic-venous sphincters

Experiments on animals, which have been reviewed by Andrews, Hecker, Maegraith & Ritchie (1955, 1956) strongly suggest that hepatic-venous barriers exist in several species and that they can act under physiological conditions, at least as a variable element of a general response by the hepatic vasculature. Blood flow in such experiments has generally not been facilitated beyond what normally prevails, and the hepatic-venous sphincters would appear usually to be in a state of tonic rather than of active contraction. The vasomotor action is probably under nervous control (Banfai, Kubik & Somogyi, 1953). Hepatic-venous spasm may be important under pathological conditions as in Chiari's disease and in some anaphylactic reactions, but there is no direct evidence for a physiological action on the part of the hepatic veins in man. There are three levels at which the hepatic-venous tree might control the outflow of blood: (i) at the ostia, (ii) at the level of sinusoids, and (iii) at junctional constrictions in the walls of hepatic veins.

(i) Venous spasm has been reported at the ostia (Bradley, Inglefinger, Bradley & Curry, 1945) during hepatic-venous catheterization and has been produced post-mortem by Elias & Feller (1931), but the amounts of muscle present in this site (Pl. 1, fig. 2*a*, *b*) seem smaller than would be expected if their action played a part in the daily economy of the body.

(ii) Adjustments of the venous outflow at multiple points in small vessels would seem more effective than control at a few sites in the larger veins, but there are no contractile elements in the sinusoidal walls that are demonstrable by histological methods. The anatomical basis for the outlet sphincters described by Knisely *et al.* (1948), Wakim & Mann (1942) and Bloch (1955) in transillumination studies on animals remains obscure. The sinusoidal circulation of the liver, however, operates at pressures under 40 mm. Hg so that the critical opening pressure is probably above the critical closing pressure (Burton, 1954). This factor will tend to maintain any closure of the sinusoids that follows a reduction in the entry of blood, and the latter is probably the primary factor causing narrowing of the sinusoids (Andrews, 1957). The possibility cannot be excluded that changes in the shape or alignment of the liver cells themselves can occur under physiological conditions in such a way as to alter the diameter of the sinusoids to which they are so closely applied.

(iii) The experiments of Deysach (1941), of Maegraith, Andrews & Wenyon (1949) and of Thomas & Essex (1949) have shown that the venous drainage can be altered round individual hepatic veins in animals. In man, Popper (1931) described junctions of central venules with hepatic veins as 'funnel-like' and later with Elias (1955) illustrated thin-walled venules constricted on entering a thick-walled vessel. In the neoprene casts made in the present study central venules were found commonly to enter hepatic veins (Text-fig. 2; Pl. 1, fig. 3) and these junctions were sometimes contracted (Text-fig. 2). Both dilated and contracted junctions were seen in sections (Pl. 2, figs. 4–6). The junctions are so frequent that junctional constriction is probably the chief venous-sphincter mechanism in the human liver. The free sinusoidal anastomosis will tend to equalize the effects among lobules in the immediate vicinity of any single constriction. Constrictions are absent where thin-walled central venules or intercalated veins unite, and thus the arrangement of the

finer radicles may influence the effectiveness of the sphincter mechanism in different parts of the liver. Circulation through the peripheral portions can be affected less by junctional constrictions than that in the central portions, because towards the surface a greater proportion of the central venules joins intercalated veins (Text-fig. 5*a*, *b*).

Possible physiological action of the hepatic veins

The special distribution of the muscle of the hepatic veins of man in longitudinal bundles seems to suit the vessels for a more or less automatic action concerning respiration. There are no valves in the hepatic veins and they are exposed to respiratory variations in thoracic pressure. Brecher, Mixter & Share (1952) have pointed out the importance of collapse of extra-thoracic veins such as the axillary and jugular veins, but this is not possible in the case of the hepatic veins, which are firmly attached to the liver substance. When blood is sucked out of the hepatic veins in inspiration, contraction doubtless occurs in the vessel walls to preserve tone. In this way, the central venules are narrowed at their junctional constrictions and the negative pressure is damped down before affecting the sinusoids. At rest, hepatic-venous constriction is probably not essential, but on deep breathing the barrier action of the hepatic-venous muscle may play a valuable part in protecting the liver from the greatest fluctuations in caval venous pressure and, to a lesser extent, in preserving the autonomy of the portal circulation.

SUMMARY

The hepatic veins and their sphincter mechanisms are described as seen in naked-eye and microscopical studies on autopsy material supplemented by casts of the veins and their small radicles and by gelatine-injection preparations.

The only normal anastomoses between the hepatic-venous territories are the continuous sinusoidal network and an irregular network of small veins in the external capsule of the liver. The hepatic veins maintain a uniform pattern of structure throughout and muscle is present chiefly in longitudinal bands in the adventitia. The smallest hepatic veins measure 400 μ in diameter in the dilated state.

The smaller veins are of two kinds—the larger or intercalated veins, 100–350 μ in diameter and the central venules, 40–100 μ in diameter. Muscle cannot be demonstrated in their walls by ordinary histological techniques. Central venules are the only veins which receive sinusoids in the human liver. They vary considerably in form and this permits an even drainage of the sinusoidal network deriving its blood from the portal tracts.

Some structural peculiarities which may determine morphological features of pathological processes in the liver are discussed. On anatomical grounds, it is considered that the short-circuit routes of Daniel & Pritchard (1951) cannot be developed sufficiently in the human liver to be of practical importance.

Structures which might act as venous sphincters are present at the ostia and at innumerable points of junction of central venules with small and medium-sized hepatic veins (junctional constrictions). An anatomical basis was not observed for outlet sphincters at the terminations of sinusoids in central venules. The evidence for physiological sphincters at the ostia is unsatisfactory, but junctional

constrictions can probably act in this way. Their action may protect the sinusoidal portion of the vascular bed from the fluctuations in the caval blood pressure caused by respiration.

REFERENCES

ANDREWS, W. H. H. (1957). The blood flow of the liver. *Brit. med. Bull.* **13**, 82–86.

ANDREWS, W. H. H., HECKER, R., MAEGRAITH, B. G. & RITCHIE, H. D. (1955). The action of adrenaline, l-noradrenaline, acetylcholine and other substances on the blood vessels of the perfused canine liver. *J. Physiol.* **128**, 413–434.

ANDREWS, W. H. H., HECKER, R. & MAEGRAITH, B. G. (1956). The action of adrenaline, noradrenaline, acetylcholine and histamine on the perfused liver of the monkey, cat and rabbit. *J. Physiol.* **132**, 509–521.

BANFAI, I., KUBIK, I. & SOMOGYI, E. (1953). The morphology and role of the phrenic nerve in the hepatic sphincter. *Acta morph.* **3**, 413–426.

BLOCH, E. H. (1955). The *in vivo* microscopic vascular anatomy and physiology of the liver as determined with the quartz rod method of transillumination. *Angiology*, **6**, 340–349.

BRADLEY, S. E., INGLEFINGER, F. J., BRADLEY, G. P. & CURRY, J. J. (1945). The estimation of hepatic blood flow in man. *J. clin. Invest.* **24**, 890–897.

BRECHER, G. A., MIXTER, G. & SHARE, L. (1952). Dynamics of venous collapse in the superior vena cava system. *Amer. J. Physiol.* **171**, 194–203.

BURTON, A. C. (1954). Relation of structure to function of the tissues of the wall of blood vessels. *Physiol. Rev.* **34**, 619–642.

DANIEL, P. M. & PRITCHARD, M. M. L. (1951). Variations in the circulation of the portal venous blood within the liver. *J. Physiol.* **114**, 521–537.

DEYSACH, L. J. (1941). The nature and location of the 'sphincter mechanism' in the liver as determined by drug actions and vascular injections. *Amer. J. Physiol.* **132**, 713–724.

ELIAS, H. & FELLER, A. (1931) Über einen muskulären Drosselmechanismus an den Lebervenenmündungen. *Z. ges. exp. Med.* **77**, 538–550.

ELIAS, H. & PETTY, D. (1952). The gross anatomy of the blood vessels and ducts within the human liver. *Amer. J. Anat.* **90**, 59–111.

ELIAS, H. & POPPER, H. (1955). Venous distribution in livers. *Arch. Path. Lab. Med.* **59**, 332–340.

FRANKLIN, K. J. (1937). *A Monograph on Veins*, pp. 55–57. London: Baillière, Tindall and Cox.

GANS, H. (1955). *Introduction to Hepatic Surgery.* Amsterdam: Elsevier Publ. Co.

GOLDSMITH, N. A. & WOODBURNE, R. T. (1957). The surgical anatomy pertaining to liver resection. *Surg. Gynec. Obstet.* **105**, 310–318.

KNISELY, M. H., BLOCH, E. H. & WARNER, L. (1948). Microscopic observations concerning the regulation of the blood flow through the liver and other organs and the removal of particles from the blood. *K. danske vidensk. Selsk. (Biol. Skrift.)* **4**, 7.

KNOPP, J. (1953). Ein Verfahren zur Abgrenzung der Stromgebiete grosser intrahepatischer Gefässe. *Virchows Arch.* **323**, 563–590.

LENDRUM, A. C. & MCFARLANE, D. (1940). A controllable modification of Mallory's trichrome staining method. *J. Path. Bact.* **50**, 381–384.

MALL, F. P. (1906). A study of the structural unit of the liver. *Amer. J. Anat.* **5**, 227–308.

MAEGRAITH, B. G., ANDREWS, W. H. H. & WENYON, C. E. M. (1949). Active constriction of hepatic venous tree in anaphylactic shock; relation to centrilobular lesions. *Lancet*, **2**, 56–57.

MIYAKE, M. (1928–30). Vergleichende Studien über den feinen Bau der Pfortader und Lebervenen. *Arb. med. Univ. Okayama*, **1**, 166–172.

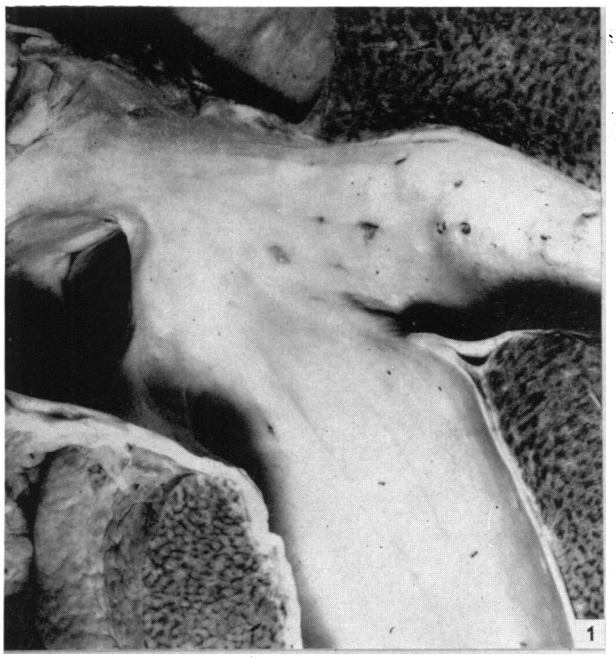
MOSCHCOWITZ, E. (1952). The morphology and pathogenesis of cardiac fibrosis of the liver. *Ann. int. Med.* **36**, 933–955.

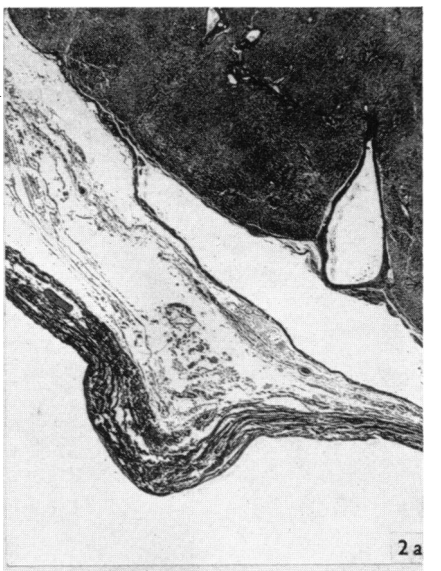
PFUHL, W. (1922). Form und Gefässbeziehungen der Leberläppchen beim Menschen. *Z. Anat. EntwGesch.* **66**, 361–384.

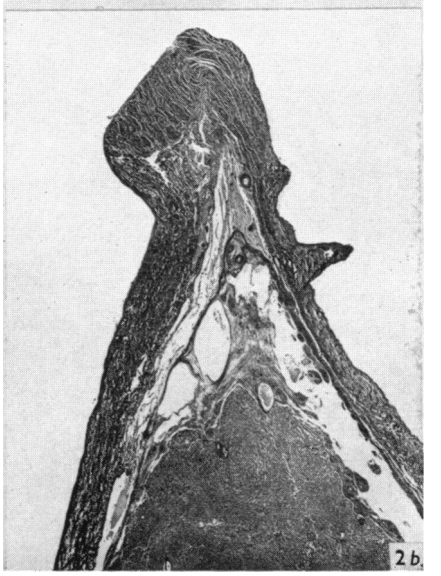
POPPER, H. (1931). Über Drosselvorrichtungen an Lebervenen. *Klin. Wschr.* **10**, 2129–2131.

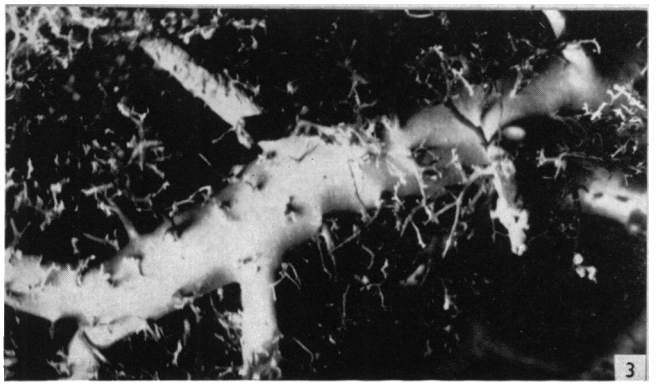
SENEVIRATNE, R. D. (1949–50). Physiological and pathological responses in the blood vessels of the liver. *Quart. J. exp. Physiol.* **35**, 77–110.

THOMAS, W. D. & ESSEX, H. E. (1949). Observations on the hepatic venous circulation with especial reference to the sphincteric mechanism. *Amer. J. Physiol.* **158**, 303–310.



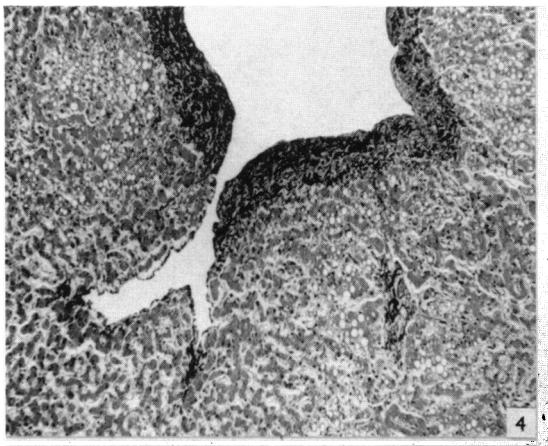


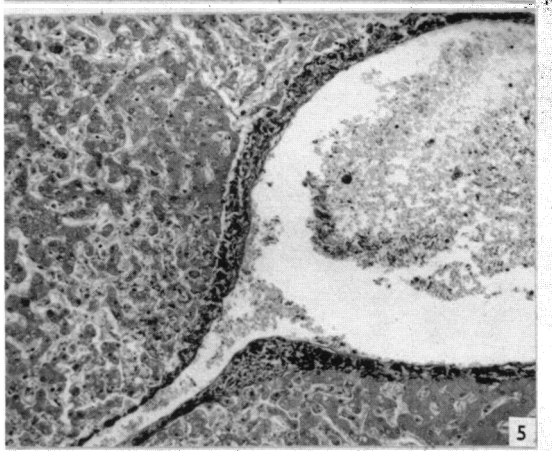


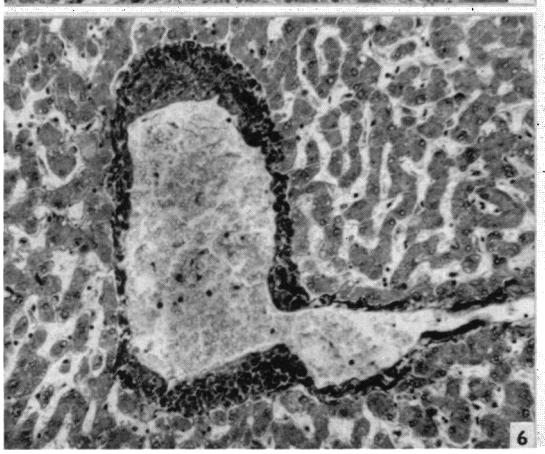


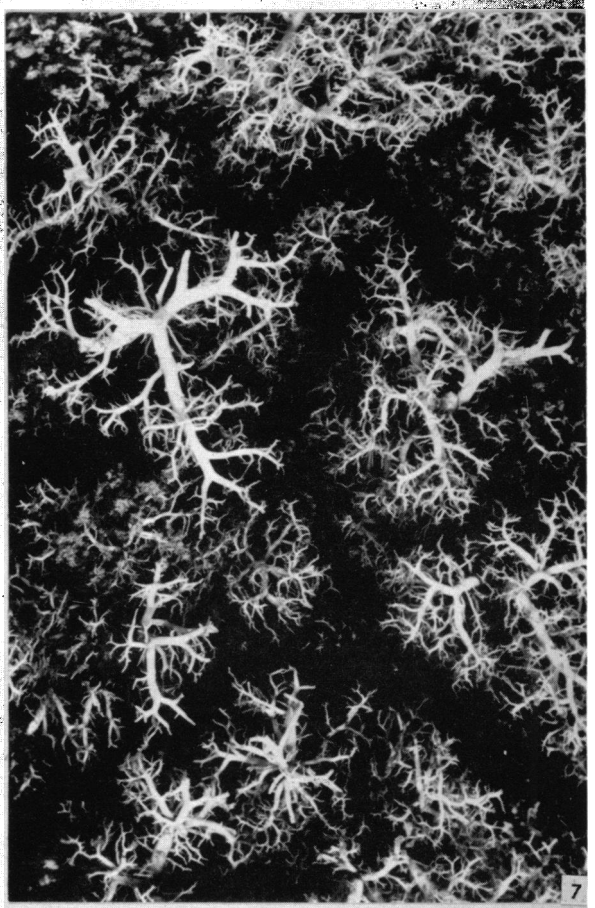
GIBSON—The hepatic veins in man and their sphincter mechanisms

(*Facing p.* 378)


4


5


6


7

TISCHENDORF, F. (1939). Histologische Beiträge zur Kenntnis der venösen Lebersperre. *Z. mikr.-anat. Forsch.* **45**, 266–290.

TORI, G. (1955). Hepatic venography. *Sci. med. ital.* **4**, 306–320.

WAKIM, K. G. & MANN, F. C. (1942). The intrahepatic circulation of the blood. *Anat. Rec.* **82**, 233–253.

WALKER, R. M. (1958). Pathology and treatment of portal hypertension. In *Circulation, a symposium*, ed. J. McMichael, pp. 379–385. Oxford: Blackwell Scientific Publications.

EXPLANATION OF PLATES

PLATE 1

Fig. 1. Junction of the right hepatic ostium (right) with the hepatic cava, dilated and seen from behind. Parts of the left ostium and of *HM* are visible (left). The distal lip of the right ostium projects as a fold. ×1·5.

Fig. 2. Longitudinal section of the right hepatic ostium showing folds formed by the junction of the ostial wall (right) with the caval wall. Picro-Mallory stain, ×9. At the upper lip (*a*) the external collagenous layer of the vessel, normally firmly adherent to the liver tissue, has been separated by artefact. The fold at the lower lip (*b*) is larger and contains lymphatics in the loose zone of the vessel wall.

Fig. 3. A portion of a neoprene-latex cast of hepatic veins of the left lobe. The largest vein (2 mm. in diameter) is an hepatic vein of the fifth order. It receives hepatic veins of smaller size and many intercalated veins and central venules. The central venules are the smallest vessels visible. ×4·5.

PLATE 2

Fig. 4. A contracted hepatic vein of the sixth order (approximately 0·5 mm. in diameter in this state) which is joined by a dilated central venule of about 60μ in diameter. The latter is constricted at its junction. Picro-Mallory stain, ×65.

Fig. 5. Dilated seventh-order vein (800μ) and a central venule (50μ). The junction is widely patent. There is slight centrilobular congestion. Picro-Mallory, ×80.

Fig. 6. Contracted eighth-order hepatic vein (300μ diameter in this state). It is joined by a large central venule which is constricted at its mouth. This is a funnel-like *junctional constriction*. The muscle which is irregularly distributed in the wall of the hepatic vein is restricted to the intima. Picro-Mallory, ×110.

Fig. 7. Anterior surface of the left lobe of the liver. The hepatic veins have been injected with neoprene-latex and the parenchyma digested down to a depth of about 3 cm. The field is composed of discrete clumps each centred round an hepatic vein of about the sixth order (2 mm.). The radicles form a medusa-head pattern and do not anastomose. The finest vessels clearly visible are central venules. ×2.