Postnatal Changes in the Portal Circulation*

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Immediately after birth the liver is deprived of the large flow of blood supplied during foetal development via the umbilical vein and portal sinus. Simultaneously the blood pressure in the portal sinus, previously as high as in the umbilical vein, falls.

How the loss of oxygenated umbilical blood is compensated and what consequences, if any, result from the blood pressure drop in the branches of the portal vein, is not yet known. However, as intestinal digestion begins, the blood flow through the portal vein, and hence through the liver sinusoids, probably increases within a few days after birth, the nutritional function of the umbilical blood flow being taken over by the portal circulation. Some accommodative structural changes in the trunk of the portal vein can therefore be anticipated, since in the foetus the portal vein probably transports only a small amount of blood from the as yet inactive gut.

It has long been observed that the postnatal changes in the liver circulation may sometimes be followed by lesions affecting each ‘half’ in a different manner (Gruenwald, 1949, 1955; Emery, 1952). More recently data about both the functional and structural adaptive changes of the portal vein immediately after birth have become available, and these complement each other in an interesting way (Arcilla, Oh, Lind, and Blankenship, 1966; Meyer and Kliebsch, 1963; Meyer, 1964).

Foetal Liver Circulation and Blood Pressure in Umbilical and Portal Veins

In the foetal liver, the umbilical vein ends with an intrahepatic umbilical recess, from the convex portion of which the ductus venosus originates (Fig. 1). Followed laterally the umbilical recess becomes the left branch of the portal vein: thus, a large amount of the oxygenated umbilical blood flows directly into the left portal branch. The left ‘half’ of the liver in this way receives oxygenated umbilical blood. On the other hand, the right ‘half’ of the liver, i.e. the area supplied by the right main branch of the portal vein, receives not only oxygenated umbilical blood, but also venous blood that flows to it via the trunk of the portal vein. This trunk opens into the portal sinus at a sharp angle (Fig. 1) and continues directly into the right main portal branch. Thus, it can be supposed that the blood from the main trunk of the portal vein is directed mainly toward the right. Accordingly, before birth the right liver ‘half’ is at a disadvantage with respect to oxygen supply, and some degenerative changes have been observed in it (Gruenwald, 1949, 1955).

In comparison with the wide main portal branches which represent a direct continuation of the umbilical vein, the trunk of the portal vein has at birth a relatively small diameter, confirming the supposition that it transports only a small amount of blood from the as yet inactive gut.

The direct communications between the umbilical vein and portal sinus suggest that, in the foetus, the blood pressure in the trunk of the portal vein must correspond approximately to that in the umbilical vein.

The problem of umbilical venous pressure in the infant at term, before initiation of breathing, was studied by Haselhorst (1929) who recorded and compared pressure in three term infants at caesarean section with infants still in utero. The mean pressure in the vein of the umbilical cord was 26 mm. Hg (range 22 to 34 mm. Hg), figures that were confirmed some 30 years later by Margolis and Orcutt (1960) who, under the same circumstances, found a mean value of 24 mm. Hg (range 17 to 32 mm. Hg). In 5 human foetuses studied at midterm we have found values between 10 and 15 mm. Hg (T. Mohr, W. Blankenship, and J. Lind, unpublished data; see Fig. 2).

Changes in Liver Circulation after Birth

Immediately after birth the pressure in the umbilical vein falls. Waligren, Karlberg, and Lind (1960) recorded pressure in the umbilical vein before and after the first breath of the newborn infant.
delivered normally at term. The range of umbilical vein pressure in six infants at 10 seconds after delivery was 15 to 50 mm. Hg, with a rapid fall to 10 to 20 mm. Hg at 100 seconds. Nyberg and Westin (1958) similarly reported a mean value of 26 mm. Hg in 17 newborn infants.

The cessation of the umbilical blood flow can hardly be compensated for by an increase in the portal circulation, as the gut only begins to function many hours after birth. Since the blood pressure in the greater circulation rises postnatally, the hepatic artery might compensate for the loss of the
umbilical blood flow but its role in this context remains unknown.

The portal pressure obtained at 4-hour of age or later is about one-quarter of the umbilical venous pressure recorded at or immediately after birth, i.e. about 6 mm. Hg (Arcilla et al., 1966). This pressure is comparable to that in adults (Sherlock, 1955), as is also the pressure difference between the portal vein and the inferior vena cava, i.e. the perfusion pressure gradient largely determines liver blood flow, since most of the liver blood supply originates from the portal vein.

Probably the cessation of the umbilical circulation does not affect to the same degree both 'halves' of the liver, i.e. its parts supplied by the left and right branches of the portal vein. In view of the anatomical peculiarities mentioned above, when the umbilical circulation stops, this would affect first the left 'half', which antenatally gets oxygenated blood via the wide umbilical vein and its immediate continuation, the left main branch of the portal vein (Fig. 3). On the other hand, immediately after birth, the ramifications of the same left main branch of the portal vein are supplied exclusively with venous blood from the trunk of the portal vein. Moreover, this change is associated with a simultaneous reversal of the blood stream in the left main branch of the portal vein. Before birth, the umbilical blood flows from left to right; after birth, the portal blood—which comes from the trunk of the portal vein—flows from right to left. Since the opening of the trunk of the portal vein is directed somewhat towards the right, and since the left main branch of the portal vein arises from the trunk of the port vein at a sharp angle, the blood supply to the left half of the liver after birth is relatively unfavour-

**Fig. 2.—Continuous pressure tracings of umbilical vein and umbilical artery of human foetus 40 g. (on the left) and 360 g. (on the right) body weight obtained at the time of caesarean section, with the placenta still in situ.**

**Fig. 3.—(Left) The distribution of the umbilical and portal blood flow in the foetal liver. The left 'half' of the liver, i.e. the part supplied by the umbilical recess and the left part of the portal sinus (up to the opening of the trunk of the portal vein) receives only oxygenated umbilical blood (hatched). The right 'half' of the liver, supplied by the right part of the portal sinus (beyond the opening of portal trunk) receives not only the umbilical but also the venous blood from the portal vein (solid black). (Right) In the adult liver all the portal branches are supplied exclusively by the venous blood of the portal vein. Note the difference in the shape of the foetal and adult liver, caused by the relative diminution of the left 'half' of the liver after birth.**
able. The consequence is that after birth there is a continuous relative diminution in the size of the left 'lobe', i.e. 'half' of the liver, followed by a marked change in the shape of the liver, which soon assumes the adult configuration (Fig. 3).

If the ductus venosus should stay open after birth, short-circuiting of the portal blood through it might further impair the postnatal nutrition of the left side of the liver. Some lesions resembling a minor degree of atrophic red infarction may occur on this basis, as noted by Emery (1952).

**Structural Adaptive Postnatal Changes of Trunk of Portal Vein**

As soon as the digestion begins, there is presumably a marked rise in the blood flow from the peripheral branches of the portal vein. The quantity of milk which the infant ingests daily in the first days of life is equal to about one-eighth of its amount. By 2 weeks of age, the infant has to deal with 500-600 ml. and sometimes with up to twice this amount. In terms of relative body surfaces, this is equivalent to an intake of 10-20 litres of milk by an adult. This rapid activation of the gut and the consequent increase in portal circulation cause a marked widening of the trunk of the portal vein in the first weeks of life. We have found that the diameter of the trunk of the portal vein—measured on the post-mortem retracted vessels—increases between 1 and 2½ times during the first 2-3 weeks of life (Meyer and Kliebsch, 1963). This striking postnatal widening of the portal vein is associated with some structural changes in its wall. The portal vein of a newborn infant often represents a contracted, thick-walled vessel with a relatively narrow lumen (Fig. 4). Its wall consists of an inner media-like layer, which is rich in elastic networks, and a broad outer layer of longitudinal smooth muscle bundles (Colour plate, Fig. A). The two layers are connected by an intermediate layer of loose connective tissue but are clearly demarcated from each other throughout. In the dorsal and lateral sectors of the cross-section, the layer of longitudinal musculature often appears distinctly folded, whereas the inner circular layer shows a regularly round contour (Colour plate, Fig. B). In the portal vein of a 19-day-old infant such folding can no longer be discerned; the muscle bundles now having the appearance of narrow flat bands (Colour plate, Fig. B). The significantly wider lumen is now surrounded by a thinner wall.

A comparison of both cross-sections shows that the postnatal widening of the trunk of the portal vein is associated with unfolding of the vessel wall, mainly of its longitudinal musculature, which has been developed antenatally and can be spread now over the expanding vessel wall.

It is noteworthy that in the unfolded portal vein the dorsal wall always appears thinner and poorer in smooth musculature than the ventral. The same differences in the thickness of the wall can be seen if the vessel is distended after death by hydrostatic pressure (Fig. 5).
Thus, the dorsal wall of the vein shows greater distensibility than does its ventral wall. The significance of these differences becomes evident when the topographical relations are taken into account (Colour plate, Fig. C), for the more distensible dorsal wall lies directly under the serosa, and here the adjacent dorsal surface of the liver forms a wide groove, the incisura venae portae. Consequently, there are no anatomical structures to oppose distension of the dorsal wall of the portal vein. The thicker ventral wall, on the contrary, is more firmly fixed to its surroundings, the bile-ducts, hepatic artery, and nerves. Moreover, the ventral wall is on the inside of the curve, which forms the trunk of the portal vein. The walls of the inside curves of blood vessels are known to be regularly thicker and less distensible (Meyer and Henschel, 1958). Therefore, it seems likely that the postnatal widening of the portal vein and its continuous adjustment to the increasing blood flow after birth occurs at the expense of its more distensible, unfixed dorsal sector. Thus, the dynamic function, i.e. adaptation to variations in blood flow, and the static function (fixation of the vessel) seem to be carried out by different segments of the vascular tube.

The difference in the thickness and structure of the ventral and dorsal walls of the portal vein persists into adult life (Krenz and Meyer, 1966). Probably, therefore, in the adult adjustment of the portal vein to varying blood volume also takes place at the expense of its dorsal segment.

Some further peculiarities of the newborn portal vein seem to be important for its continuous and rapid adjustment to the increasing blood flow after birth. The portal vein in the newborn shows a well-differentiated and clearly demarcated inner circular layer (Colour plate, Fig. D, left), which is rich in elastic network and smooth muscle cells. Remarkably, with advancing age the collagen networks, which increase in mass, gradually dissociate the elastic networks, blur the boundary between this layer and the longitudinal smooth musculature, and fuse them together (Colour plate, Fig. D, bottom). Thus, in its original form the inner circular layer appears to possess a special function after birth and during the first phase of growth, probably by ensuring a high degree of elasticity of the venous tube. Later in life it seems that this layer is not needed to the same extent.

The longitudinal smooth musculature of the portal vein also seems to be important for its adaptation to the increasing blood flow after birth. The elastic fibrils, which run on the surface of the muscle bundles, connect them with the elastic and collagenous networks of the whole vessel wall, including the circularly oriented layers. Thus, the longitudinal musculature, besides varying the tension of the parallel running fibrils, can also influence the status of circular networks and in this way adjust the cross-sectional area of the portal vein trunk. During the forthcoming growth, differentiated elastic tendons appear on the ends of some smooth muscle bundles and connect them directly with the circularly arranged elastic networks (Meyer and Kliebsch, 1964).

**Summary**

During foetal development a large portion of the oxygenated umbilical blood flows through the umbilical recess directly into both main portal branches. Accordingly, these are exposed to the umbilical blood pressure and have a wide lumen. By contrast, the trunk of the portal vein is narrow, and there is probably only a small blood flow through it from the inactive digestive tract, though the pressure in it is approximately the same as in the umbilical vein or perhaps somewhat higher.

Immediately after birth the liver is deprived of the substantial umbilical blood flow. The portal venous pressure falls to about one-quarter of the umbilical venous pressure at birth. However, the pressure gradient between the portal vein and inferior vena cava remains constant.
With the cessation of the umbilical circulation the preferential blood supply of the left liver half, consisting exclusively of oxygenated umbilical blood during foetal development, ceases. This causes a lower rate of postnatal growth of this half of the liver. 

With the beginning of intestinal function, the blood volume flow through the portal vein increases considerably, causing a marked postnatal dilatation, the diameter of the vessel approximately doubling in the first 3 weeks. This dilatation is associated with an unfolding of the originally thick wall of the portal trunk, especially of its longitudinal musculature which spreads now over its thinning wall.

The increased portal circulation enhances primarily the blood supply to the right half of the liver, since the opening of the portal trunk is directed toward the right. The resulting relatively higher rate of growth of this liver half favours the marked postnatal change in liver shape.

Sometimes acute pathological lesions can be observed as a consequence of the postnatal change in liver circulation in either its right or left half.

References


