The Function of the Gallbladder as a Lesser Biliary Circulation

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IT is a traditional teaching method to regard the gallbladder (GB) as a reservoir for bile, whereby the bile secreted from the liver is stored and concentrated in this viscus until required for further digestive purposes. The GB is believed then to contract and discharge its contents to the cystic duct (CD), common bile duct (CBD) and then to the duodenum. The hypothesis questioned in this paper is concerned with the function of the GB. Accordingly, all the bile which enter the GB is believed to be totally absorbed back to the liver through the tributaries of the cystic vein (CV) (which drain the GB to the hepatic vein) and possibly through the ducts of Luschka (DL) and Rokitansky-Aschoff sinuses (RAS). In this way, none of the bile contained in the GB can flow to the CD, CBD or duodenum and the bile required for digestion will therefore come from the liver. The contraction of the GB (cholecystokinesis) are believed to increase and maintain certain gradients of intravesical pressure which in turn will increase the filtration force necessary for the absorption of GB bile. The GB may act therefore as a lesser biliary circulation (LBC) to absorb all the GB bile to the greater biliary circulation, i.e. the liver.

This may seems a telelogical hypothesis especially with the lack of experimental evidences which may be taken against it. It is however, based on many relevent clinical observation collected from the well documented literature as well as from the limited practice of the author.

The walls of the GB are made of smooth muscle mixed with fibrous tissue and are lined by mucosa. The mucosal surface which is designed for absorption is divided into polygonal spaces by delicate mucosal fold. These fold are best appreciated if a fresh GB is examined under water using a dissecting miscroscope and will be seen to float up like a water plant in a clear pool. Around the neck of the GB, the mucosa is thrown into series of short ridges arranged spirally around the neck and are known as the "spiral valve" (Valve of Heister). Being made of mucosal folds, this valve may only allow the flow of bile from the extrahepatic biliary ducts (EBD) to the GB but prevent its flow from the GB to these ducts. There is no known valve which would allow the flow of fluids in both directions unless if it is controlled manually or automatically.

Normally, the GB does not lie obliquely with the common hepatic and biliary ducts as it is usually illustrated. It rests on the transverse colon and curves on itself so that its body may reach as low as the opening of sphincter of Oddi. This position does not help evacuation of bile to the CD but its retention inside the GB and thus its absorption.

Histologically, the wall of the GB consists of mucosal, muscular and serous coats (Bloom and Fawcett 1975). The mucosa is made of tall columnar epithelium and lamina propria. Goblet cells are lacking and glands are absent except near the neck of the GB where few tubuloalveolar mucous-secreting glands are uncommon. The muscle coat is composed of few interlacing bundles of smooth muscle fibres of irregular pattern in stroma made of fibrocollagenous tissue. The serous coat is formed of dense connective tissue and is continuous with the interlobular connective tissue of the liver. The blood vessels of the muscular coat communicate freely with those of the mucosal and serosal coats (Bloom and Fawcett 1975). Again, the tall lining are designed for absorption rather than secretion and the various droplets commonly seen in sections of normal GB may indicate its absorptive role including that of lipids. These droplets must not be confused with those of cholesterolosis in which the droplets are not only confined to the epithelial cells but also in the phagocytic histiocytes in the deeper parts of the GB. On the other hand, the ultrastructure of the lining epithelium of the GB shows great absorptive similarity to that of the intestinal mucosa (Bloom and Fawcett 1975).

The absence of submucosa from the GB indicate that the contraction of its muscles are not "true peristalsis" as those of the stomach or intestine where the contraction of the well developed muscle coat permit the mucosal folds to move and slide over a large surface of submucosa thus aid in absorption. As mentioned earlier, the contraction of the randomly oriented (i.e. weak) smooth muscle bundles of the GB may principally be aimed at maintaining a certain gradient of intravesical pressure necessary for the total absorption of bile.

Another possible route for the absorption of bile from the GB is through the DL and RAS. The DL are present on the hepatic side of the GB and communicate freely with each other in the serosal coat of the GB as well as with the intraphepatic biliary ducts (Mis-

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sion 1969). These ducts were reported not to open into the lumen of the GB, (Mission 1969; Bloom and Fawcett 1975) but they may possibly communicate in the serosal coat with the RAS which are themselves a mucosal extrusions into this coat. The presence of RAS in sections submitted for histological examination is not an indication of a diseased GB because they are commonly encountered in sections of normal GB (Hoffbauer 1947). It follows that during chole-cystokinesis the GB bile may flow directly through RAS and DL to the liver. This route may account for the absorption of such bile components as cholesterol and bile salts and pigments which are believed not to be absorbed from the GB mucosa (Dietschy 1966; Lester and Troxler 1969; Ostrow 1969; Brobeck 1973).

Bile is excreted by the liver throughout life. Many food substances namely lipids and proteins are known to stimulate its secretion. Bile (bile salts) itself on reaching the small intestine has a powerful choleretic effect. This is probably through the release of hormone cholecystokinin (Banfield 1975) and possibly secretin which has no cholecystokinetic effect. (Grossman et al 1949; Brobeck 1973) Carbohydrates and increased pressure within the biliary ducts are known to depress bile secretion (Brobeck 1973). During the fasting periods, when there is no food in the duodenum, the tonic contractions of the sphincter of Oddi can raise the pressure in those ducts to as much as 15—25 cm of water. At this range bile can flow through the CD to the GB, but at pressure levels above 25 cm of water the secretion of bile by the liver ceases. (Brobeck 1973). The rhythmic contraction of the GB can however raise the pressure in this viscus to 25—30 cm of water without affecting the secretion or the flow of bile from the liver (Brobeck 1973). It seems very probable that this high intraluminal pressure is the essential factor in raising the filtration pressure required for the absorption of bile from the GB. Another inference would be that the function of the GB is not only its role as a LBC but also its control of the pressure within the biliary ducts thus preventing its rise about the physiological limits of bile secretion.

The bile which enters the GB is composed of water (98%) and solids (2%) which include bile salts, bile pigment, cholesterol, mucine and inorganic salts (Brobeck 1973). Given the capacity to hold about 50—60 ml. this organ would soon be filled with bile beyond its storage capability were it not for the remarkably rapid ability to reabsorb water and inorganic salts (Bicarbonate and chloride salts of sodium and potassium) (Dietschy 1966; Brobeck 1973; Banfield 1975). For example, the canine GB can absorb 10—16% of its volume per hour and almost 90% of the GB bile water is absorbed during this period (Rous and McMaster 1921; Ravdin et al 1932; Grim and Smith 1957; Dietschy 1966). Thus the organic contents of the 50—60 ml. GB bile represent the organic contents of 500—600 ml. liver bile. This concentrating power of the GB to absorb water and inorganic salts leaves the GB with concentrated bile made namely of bile salts, bile pigments and cholesterol which are reported as being a non-absorbable materials (Dietschy 1966; Lester & Troxler 1969; Ostrow 1969; Brobeck 1973). But this is debatable and there are many evidences that even these substances are normally entirely absorbed by the GB mucosa (Wheeler 1971). For example, investigation into the absorption of bile salts reveals that the normal GB can absorb these salts by passive diffusion (Ostrow 1969). Again it seems probable that the absorption of these substances can only take place when a certain level of "absorptive intraluminal pressure" is attained and when certain other bile components are absorbed into the intrahepatic (or biliary duct) circulation.

Bilirubin is the chief bile pigment present in the human bile and this must be conjugated with glucuronic acid prior to its excretion by the liver (Lester and Troxler 1969; Brobeck 1973). Again, there are evidences to indicate that the unconjugated forms of bilirubin are readily absorbed by the GB whereas the conjugated bilirubins are also absorbed, but at a slower rate (Ostrow 1967). It is interesting to note that the conjugated forms are water soluble and thus may diffuse easily, possibly alongside with the bile salts. The unconjugated forms are on the other hand lipid-soluble and may possibly be absorbed alongside with cholesterol which the GB can also absorb (Wheeler 1971). The absorption from the GB of bile and possibly the secretion of other substances including salts, mucine (from the mucous glands which are normally present in the neck of GB) and cholesterol is kept under constant equilibrium so that a disturbance of either mechanisms may result in the appearence of various pathological conditions of the GB including gallstones. Although, the pathogenesis of cholelithiasis is not fully understood (Cardell 1978) their formation around a nucleus of desquamated epithelial cells following cholecystitis is undisputed. A diseased or damaged GB has however, more capacity than normal to concentrate bile (Ostrow 1971) and this may further produces the viscous circle of cholecystitis - gallstones (Cardell 1978).

During routine postmortem, which is normally performed 4-6

hours after death the pressure applied to the GB which causes the flow of bile to the EBD is used to identify the choledochoduodenal papilla. This does not object with the present hypothesis of the GB as LBC because of the rapid autolysis of the GB (and gastroduodenal) mucosa, including the valve of Hiester which occur in less than 30 minutes.

In cholecystography, the dye salts of phenolphthalein are given either orally or intravenously but the first radiograph whch normally shows the shadow of the GB is taken 2-14 hours depending on the route of administration of the salt. A second radiograph showing the "emptying" (i.e. contraction) of the GB is taken 1-5 hours after the ingestion of fatty meal. These observations may again question the contractions of the GB because if they are meant to evacuate its contents to the CD and CBD, then the radiographs should be taken immediately after the administration of the dye or the fatty meal. It appears that these contractions are like those of the splenic capsule in response to shock conditions. Thus whereas the splenic capsule may take several hours to undergo gradual contractions to evacuate its contents into the general circulation and alter the peripheral haematological indices, the GB may also take several hours in raising its intraluminal filtration pressure for the absorption of its bile.

It is interesting to note that after cholecystectomy, the biliary ducts become dilated not only to accommodate the bile which is continuously secreted by the liver, but also to "re-establish" to some extent the lost role of the GB as a LBC. Similarly, the tributaries of the retroduodenal vein (which drain the EBD to the gastroduodenal vein and then to the portal vein) become dilated and tortuous. Likewise, this is to compensate for the role of the tributaries of the CV. There is some evidences to indicate that cholecystectomy is followed by increased biliary duct pressure and this may cause the liver to secrete less bile than normal (Menguy et al 1958; Brobeck 1973).

Finally, it should also be noted that patients with biliary dyskenesia who are advised to avoid fatty meals will continue to complain of fatty dyspepsia even after cholecystectomy. This is not only due to the cholecystokinectic effect of these meals but also to their choleretic effect on the liver to secrete more bile than what the EBD can accommodate. □

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