was withdrawn. The pathologist's report indicated that we were dealing with a case of chylothorax.

The initial aspiration produced a temporary improvement, and energetic steps were then taken to restrict the intake of fats and to rely on rectal feeding. It was impossible to control the outflow of chyle, however, and on August 21, against our better judgment, we again felt obliged to aspirate. On this occasion five pints of similar fluid were removed and relief proved dramatic. Although obviously wasted, he became cheerful and was quite free from pain. Continuous intravenous glucose-saline was now administered, but again and again chyle accumulated, and in all twenty-seven pints had been aspirated by August 27, when the patient died in a state of exhaustion. Dr. A. F. Sladden, who undertook a post-mortem examination, has been kind enough to furnish the following notes:

"During life a sample of fluid aspirated from the right pleural cavity was examined and found to be sterile and free from excess of leucocytic cells, but milky in consistency and appearance. Further analysis showed the presence of fat to the extent of 0.83%, and the effusion was accordingly regarded as chylous in nature. At the post-mortem examination the right lung was found collapsed, with some chylous effusion around it. No effusion was present on the left side. The parietal pleura as it lay along the spinal column was torn to form a diamond-shaped gap 3 inches long and 3/4 inch wide, the greater length of the 'diamond' being in the line of the spine on its right lateral aspect. The centre of this lesion lay across the level of the ninth and tenth thoracic vertebrae, and the junction of these two vertebral bodies was completely fractured though there was no displacement. The anterior ligaments had been completely torn across, and this injury had also severed the thoracic duct as it passed upwards along the spine. From this torn duct chyle had leaked into the right pleural cavity and had caused profuse effusion of fluid around the right lung."

### Discussion

McNab and Scarlett (1932) in an authoritative article on traumatic chylothorax mention that rupture of the thoracic duct most frequently causes an effusion into the right pleural cavity. They suggest that this occurs because the duct is on the right side for two-thirds of its thoracic course, the aorta forming a barrier to the left. In the experience of these clinicians there is seldom a history of crushing violence, and, excluding penetrating wounds, trauma is usually due to a sudden and exaggerated lordosis of the spine, causing excessive stretching of the duct. They hold that rupture is more likely to occur shortly after the ingestion of a large meal, when the duct is distended by chyle. Usually a silent post-accident period of three or four days is followed by the abrupt onset of dyspnoea, rapid thready pulse, pallor, cold sweats, and profound shock. The injury has proved fatal in 50% of the recorded instances.

Clearly the case we have described does not in any way upset the general conclusions of McNab and Scarlett, but it may provide a means of accounting for the "latent period," which is so mystifying. It is quite conceivable that after rupture of the thoracic duct the chyle, which is not under any marked pressure, first accumulates behind the fairly resistant pleura. When a portion of the latter membrane becomes tense enough for its blood supply to be depleted erosion would be simplified and subsequent outpouring of chyle would precipitate symptoms of intense shock. Our only evidence in support of this theory is the diamond-shaped pleural tear described by Dr. Sladden, which might have resulted as readily from erosion as from trauma.

### Treatment

The methods of treatment which have been recommended in traumatic chylothorax are varied and in some instances contradictory. They include: (1) abundant administration of fluids by mouth; (2) curtailment of fluids and the institution of rectal feeding; (3) a fat-free diet; (4) a diet rich in fats (one practitioner actually administered chyle by mouth!); (5) whole-blood transfusions and continuous intravenous glucose-saline (since treating our case we have learned that glucose causes a rise in lymph pressure, so that in all probability this measure is harmful); (6) the creation of a positive intrathoracic pressure by means of artificial pneumothorax—apparently this cannot possibly compress the ducts enough to prevent the outflow of chyle; (7) thoracotomy, with removal of large clots—the aim of this more heroic measure appears to be the production of adhesions around the leaking duct; (8) reintroduction of extravasated chyle.

The last-mentioned treatment strongly appeals to us, and we regret our ignorance of the technique when our case was under observation. Bauersfeld (1937) describes a patient suffering from chylothorax whose condition steadily retrogressed until tubing from a needle in the chest was connected with a direct transfusion set and the fluid was reintroduced into the right median basilic vein. The general condition then steadily improved and weight increased rapidly. It may be, as Bauersfeld modestly suggests, that sealing of the tear in the duct occurred in the period during which intravenous chyle was administered; but even if this be so it does not detract from the value of his novel treatment.

Unduly impressed, perhaps, by our recent experience, we feel bound to advise that even in trivial injuries to the neck, chest, and spine the possibility of damage to the thoracic duct must always be envisaged. Moreover, in these days of falling masonry and of stabbing and bullet wounds it is not unlikely that chylothorax may be met with more frequently than in the past.


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# COMMON BILE DUCT OBSTRUCTION

## IMPORTANCE OF SURGICAL DECOMPRESSION


BY

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In spite of the growth of hepato-biliary surgery the patient with jaundice as a result of common bile duct obstruction still represents a complex surgical problem. Many investigations of this pathological state are being carried out to-day, and it is only by examining the physiological, clinical, and surgical aspects of the question that new advances can be made.

In the past decade the importance of the mechanical alterations in the biliary system has come to be recognized. This is of value because not infrequently they are the sole cause of the severe damage to the liver and are also often associated with other inflammatory conditions. It is necessary to study these mechanical alterations in the biliary system in two sets of circumstances: (1) during the obstruction of the common bile duct; and (2) during the decompression of the biliary tree.

### Effects of Obstruction of the Common Bile Duct

In reference to the former, the most important consideration is the phenomenon of biliary hypertension. It is well known that the bile pressure increases progressively after obstruction of the common bile duct, and this increase in pressure is responsible for the dilatation of the intrahepatic and extrahepatic bile channels. This

hypertension, occurring in a viscus in which further distension is limited by its capsule, has repercussions on the more delicate elements of the parenchyma—the liver cells and the blood vessels. Following upon this we see:

(*a*) *The Mechanical Effect on the Epithelial Cells.*—In the lobules the dilated bile capillary causes compression and disruption of the columnar cells. The intercellular fissures allow the escape of bile into the blood capillaries and lymphatic spaces, and thus bring about jaundice.

(*b*) *The Effect of this Biliary Hypertension on the Blood Circulation.*—Interference with portal circulation is brought about through compression of the interlobular veins by the distended bile ducts. Often the sinusoids also are compressed by the dilated bile capillaries.

The extension and intensification of these two groups of alterations are proportionate to the duration and the degree of common bile duct obstruction.

The above facts have been observed by different workers and also by me in experimental animals (thirty-two dogs) and in man. These histological changes are of clinical importance because they show that a patient with biliary obstruction suffers a progressive loss of the vital activity of the liver, and therefore the risk becomes increased with imprudent waiting before draining the biliary tracts. The importance of this is apparent when it is realized that cellular regeneration is markedly inhibited while the obstruction is present (Bollman and Mann, 1936).

### Decompression of the Biliary System

More recently the second aspect of the problem—the decompression of the biliary system—has been the chief object of consideration. It is on this question that I wish to lay particular stress.

A general pathological principle to consider first is that *the delicate elements of an organ exposed for some time to an increased internal pressure do not reassert themselves in the same manner when the decompression is carried out suddenly as when it is carried out gradually.* In connexion with this principle, what are the physiological and pathological modifications in the liver during biliary decompression? There are three factors to be considered in relation to this: (1) the rate of the decompression; (2) the degree of the obstruction; and (3) the duration of the obstruction.

Regarding the first of these factors it is recognized that rapid drainage of the bile brings about an important alteration in structure of the hepatic lobule, whereas slow drainage does not cause as substantial a change in the parenchyma of the liver. I carried out a series of experiments the object of which was to study this question (Canonico, 1938). In fifteen dogs I produced a mechanical obstruction of the common bile duct. After a period of obstruction varying from one to fifteen days the biliary system was decompressed by removal of the ligature (slow decompression), by indirect cholecystostomy (rapid decompression), and by two-stage cholecystostomy (rapid and slow decompression). The histological changes were as follows: In the case of rapid decompression destruction of the normal lobular architecture occurred; the blood capillaries immediately became congested, resulting in many cases in rupture of the walls, with ensuing haemorrhage. The columnar cells were disrupted and the cells underwent necrosis and autolysis. These changes were more intensive the longer the duration and the greater the degree of the obstruction. On the contrary, with slow experimental decompression there was no marked change in the lobular structure.

What is the pathogenesis of the observed phenomena resulting from sudden decompression? I believe these are a result of the physical changes in the parenchyma. As previously established the biliary hypertension during the obstructive period is responsible for the collapse of the sinusoids and the interlobular branches of the portal vein, with the ensuing interference with the blood circulation. In these circumstances if an extensive bile drainage is performed a sudden fall in pressure is produced in the bile system with a corresponding sudden increase in the vascular system. Consequent upon this rapid increase of pressure in the blood vessels there is possible rupture of the capillary walls (haemorrhage *ex vacuo*) and intensive disruption of the columnar cells. On the other hand, the gradual lowering of the bile pressure allows the progressive re-establishment of the balance between the biliary and the circulatory systems without substantial damage to the lobular structure.

In accordance with the above concept it is quite simple to appreciate the importance of the second and third factors. The greater the duration and the degree of obstruction of the common bile duct the more favourable are the conditions for the production of the pathological changes, for the cellular lesions and the circulatory interference are increased. In the experimentally produced total obstructions lasting only twenty-four to forty-eight hours, or in the partial obstructions, sudden decompression resulted in less damage. In other words, these facts are in keeping with the known phenomena of rapid decompression in any hollow viscus, as seen particularly in rapid emptying of a markedly distended bladder with the resulting congestion and haemorrhage into the mucous membrane. McLaughlin and Levering have obtained similar results in their studies of rapid lowering of gastric pressure.

I feel that these findings are of practical importance in the surgery of obstruction of the common bile duct, and believe that the same phenomena observed in the experimental animals occur in the human and are responsible in some cases for two post-operative complications—haemorrhage and "hepatic insufficiency."

### Commentary

From the above follow two principal conclusions: first, aspiration must be avoided in any bile drainage; secondly, gradual decompression of the obstructed biliary system must always be the procedure of choice.

To obtain slow decompression one should not use a large-bore drainage tube. It is also essential to regulate the flow of the bile so that emptying occurs very slowly. To procure this it is sufficient to compress the drainage tube partially with the ordinary clamp or to raise the tube to a level above the bile duct that will neutralize the biliary pressure. Ravdin and Frazier use for this a simple apparatus. In general, during the first twenty-four hours there should not be collected more than 250 to 300 c.cm. of bile. After this the rate of drainage is regulated to increase the flow of bile. Our experience with slow decompression in man is very heartening. Among British surgeons Sir John Fraser (1938) suggests: "When operating on cases which are profoundly jaundiced adopt a two-stage method, for by this means the risks of a sudden biliary decompression are diminished."

In conclusion may I repeat that "hepato-biliary surgery bases its progress on the increased knowledge of the physiopathology of the liver and its excretory ducts" (Canonico, 1937).

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