OBSTRUCTION
OF THE
THORACIC DUCT.

BY

E. C. EDWARDS.
MB, CM, EDIN.
Obstruction of the thoracic duct is spoken of by Professor Coates in his work on General Pathology as "not an infrequent accident." Nevertheless, it is a lesion about which, apparently, very little is known, if we may judge from the scantiness of the literature on the subject. Hitherto, the examples of it which have been placed on record are comparatively few, and this appears to be owing partly to the secret position of the duct, and partly to the obscurity, and in some cases even the total absence, of the phenomena presented by that lesion. Moreover, even in some of the cases which have been recorded, the existence of the obstruction was only surmised, while the actual state of the duct, mirabile dictu, was not examined. In the majority of the autopsies made at the present day the condition of the thoracic duct is altogether overlooked, and I cannot discover in the literature any account of the results

of a systematic examination of the thoracic duct. Since that published in the early part of this century (1829) by M. Andral (1). He states that he examined the duct in over six hundred bodies, and only very rarely met with any appreciable lesion in it: apparently, from his account, it was the seat of pathological alteration about five times in the six hundred and odd cases examined.

Now it fell to my lot in the early months of last year to have under my care a patient suffering from obstruction to the thoracic duct. As I have not recorded the case elsewhere I shall proceed to describe it here, in the hope that it may throw some further light on the subject, to the general consideration of which I shall have to return later.

The following is the history of the case:

A.C., aged 61, a carpenter, came under my care at the East Suffolk Hospital on March 1, 1875, complaining of swelling of the body and legs, shortness of breath, and troublesome cough and expectoration.

His family history was good and his habits temperate. He had never roved out of England and except for bronchitic attacks during the past few winters had always enjoyed good health.

His present illness began as follows: for many past he had suffered from pains in his body. Six weeks before admission the legs began to swell, and he had sickness every night until one week before admission, when it had ceased, and thenceforward he said, the abdomen first began to swell. Up to that time he had continued to follow his employment, but for the past five days had been obliged to take to his bed, since when the swelling of the legs had diminished somewhat.

On admission he appeared a short, heavy man, grey-haired and old-looking. The face
was pale, the breathing laboured, and the patient was weak and unable to stand. His temperature was normal.

**Alimentary System.**

He complained of constant pain in the abdomen, in the umbilical and epigastric regions. The appetite was bad, the tongue coated, and the bowels would only act with perseverence. The abdomen was very prominent, measuring 48 inches round (at a point 2 inches above the umbilicus): there was no enlargement of the abdominal cutaneous veins. The abdomen was tender and "pitted" on pressure; it was extremely tense and a very distinct thrill of fluctuation could be obtained. The percussion note, tympanitic round the umbilicus, was dull above the symphysis (forehead 5 inches) and in the flanks, the note becoming tympanitic in the upper flank when the patient was turned on his side. The large border of the liver lay one inch below the right costal margin, but the extent of its vertical depth was not obtainable on account of the lung condition.

**Respiratory System.**

Respiration was laboured and wheezing, so
per minute, and there was frequent cough with a considerable amount of muco-pulent 
expectoration.

The chest was markedly "barrel-shaped," and the lungs hyper-resonant and otherwise 
possessing all the ordinary signs of chronic bronchitis and emphysema — those 
signs of pleural effusion at both bases post-

Circulatory System.

There were cyanosis of the lips, dyspnoea, and faintness.

The heart was completely covered by the lungs, could not be seen or palpated, nor 
was there any cardiac dulness obtainable. The sounds were feeble and only with difficulty 
heard through the pulmonary sounds. There was no audible murmur. The pulse was 
regular, 50 per minute, and small in volume and force. There was some thickening of the 
ventricles.

Urinary System.

Micturition was frequent, and difficult 
owing to the constant condition of the bladders.
The quantity of urine was scanty (12 ounces in
the 24 hours); it was turbid and high-coloured, acid, spec. grav. 1032, and containing albumen in small amount and a copious deposit of a mucus-coloured matter. It contained no blood, bile, or sugar, and microscopically one found, besides the mucus, hyaline casts in number and a few granular epithelial casts.

There was no emaciation but rather adiposity.

The long limbs (especially the right), the scrotum, penis, abdominal walls, and loins, were all very oedematous; but elsewhere there was an absence of oedema.

There was no enlargement of lymphatic glands to be made out.

On the following day (March 2nd) paracentesis abdominalis was performed with Soutter's aspirating tube, when 125 ounces of milky fluid were drawn off. The abdominal circumference was thus reduced from 43 to 40 inches, but although a careful examination was made nothing further could be discovered in this region.

The patient, after experiencing temporary
Considerable relief from the aspiration, very soon became worse again. The urine continued scanty, ranging from 10-25 ounces per day. Its character remained as before, and frequent examination failed to detect the presence of chyle in it.

The stools, except from being watery by the use of purgatives, presented no abnormality in appearance. The ordure of all the parts was on the increase; and, in order to render nutrition less difficult, incisions were made in the breast, the fluid evacuated being of the ordinary serous type. The abdominal pain continued, and the patient was losing flesh considerably. His temperature remained subnormal. The abdomen showed signs of having rapidly refilled with fluid, and on March 11th (ten days since the previous aspiration) it was again tapped. 35 ounces of fluid were drawn off, very similar in character to that previously obtained but rather more yellow and creamy in appearance.

During the next two days the patient sank rapidly, the heart weakness greatly increasing, and death resulted on March 14th.
Post Mortem Examination - March 15.
The body was swaddled in the upper parts, the abdomen was prominent and tense, and all the lower part of the body were extremely oedematous.

Throat.
The cartilages were fully ossified.

Opening the chest, the pericardium was invisible, being completely overlapped by the lungs whose anterior margins met near the middle line.

The right pleural cavity contained 30 ounces, the left 40 ounces, of turbid reddish fluid. There was old standing pleuritic adhesion of this at the apex of the right lung; and on both sides some difficulty was experienced in removing the lungs owing to their adhesion to the pleura, both at the diaphragm near the mid-line posteriorly, and also along the posterior mediastinal region. This adhesion was evidently of recent origin.

The lungs were deficient in weight (right weighing 49 ounces, left 13 ounces), and both were markedly emphysematous, especially at their anterior margins. The base of the
Lungs were emphysematous in character and contained a considerable amount of mucoid, frothy material. At the anterior surface of the root of the right lung were two or three enlarged lymphatic glands about the size of a pea and containing soft, fluminous material.

The pericardium contained 1 ounce of turbid, yellow fluid.

The heart weighed 11 ounces. Its valves were competent and natural, except for atheromatous changes (thickening and calcareous nodules) in the segments of the aortic valve. The heart walls were natural. There was a considerable amount of pale antemortem clot in all the chambers.

Abdomen.

The abdominal walls were edematous and contained a thick firm layer of fat. The peritoneal cavity contained rather
More than 2 pints of free fluid, milky-like that evacuated during life.

The great aneurism extended well down over the intestines, and its condition was altogether most remarkable. It was hard and firm, varying from half to three-fourths of an inch in thickness, and contained an excessive amount of fat, the fine localization of which rendered the inner surface of the aneurism very uneven.

On turning back the aneurism it was apparent that the small intestines were projected forwards by some deeper-lying mass.

The visceral peritoneum showed definite signs of chronic peritonitis, more especially in the upper part of the abdominal cavity where there were adhesions and matting together of some of the visceru, as will be mentioned a little later on.

The serous lining of the stomach, towards the pyloric end and along the lesser curvature, was much roughened and raised into small papillary prominences. Posteriorly it was bounded down by adhesions in the...
pyloric region. There was no dilatation of the stomach and its intestinal surface presented no abnormality.

The small intestine. The serous coat appeared slender throughout and the blood vessels in parts were congested, but there was no plastic adhesion of the bowels and no deposit of lymph. The appearance of the lacteal vessels throughout the length of the small intestine was most remarkable, as will be seen by the accompanying photographs (taken from a portion...
of the jejunum). They were in a state of extreme chylean engorgement, standing out prominently as white tortuous bladed channels, so that their cause in the wall of the intestine could be admirably seen and traced. They were largest and most numerous towards the side of the attachment of the mesentery. When punctured, a creamy material escaped.

As the internal surface of the intestine the villi appeared very prominent throughout, distended with chyle.

The mesentery, in its entire extent was, like the intestine, abnormally thick, varying from 3/4 - 1 1/2 inch. When held between the fingers it felt peculiarly firm while the enlarged lymphatic glands in it could be readily felt out. An section it was seen to contain an excessive amount of fat and fibrous tissue, and its lymphatic vessels and glands were greatly dilated and distended with creamy chylean material which readily exuded on slight pressure.

The large intestine - There was the same
dry appearance of the mammary coat and the absence of engorgement of its lymphatics contrasted strongly with the condition seen in the small gut. The lining mucous membrane appeared normal. The mesocolon like the intestines was thickened and firm and contained a large quantity of fat, but an injection there was no exudation of chyloous material. The liver weighed 4 lbs. 12 oz., and was enlarged hard and firm, feeling almost like a waxy liver but giving no reaction with iodine. The right lobe was adherent to the diaphragm along its upper surface posteriorly, while below there were adhesions as will be noted immediately. The surface had a slightly pitted appearance. On section there were no cancerous nodules found, and no chyloous material was seen or could be expressed from the cut surface. The gall-bladder was distended, containing thick yellow bile. The spleen weighed 3 ounces. It was decidedly small and atrophied. There
were no cancerous deposits in it, nor could chyle be expressed.
There was an excessive quantity of peri nephritic fat. The left kidney weighed 7 oz.; the right 6 oz., and both were natural in size. The capsules were a little protruded and did not ship readily. Auscultation, the only abnormality was the presence of small cream-coloured points in the vicinity of the blood-vessels of the renal pelvis. These little points when cut across exuded, a slight pressure, thick creamy material, and evidently represented lymphatic vessels impaled with chyle.

The stomach and intestines were next removed from the abdomen, but not without considerable difficulty, as the duodenum was found imbedded in a mass of new growth.
This mass was hard, and irregular in surface, and extended across the upper posterior part of the abdomen, from the vicinity of the spleen on the one side
to the neighbourhood of the right kidney, and suprarenal body, and the hepatic flexure of the colon, as the other — this latter being doubled on itself at an acute angle owing to impaction of its mesocolon in the contraction set up by the Neoplasia — The mass of Neoplasia occupied the position of the pancreas; it was adherent to the under surface of the right lobe of the liver and the pylorus, and deeply involved the duodenum with the termination of the common bile duct. The main portion of the mass consisted, apparently, of fibrous tissue, but in its substance portions of pancreatic tissue could be distinguished from which clear fluid escaped on section, and it was noticeable that no chylous material could be expressed from the cut surface of the tumour mass. Posteriorly, the trunks of the abdominal aorta and inferior vena cava were involved in the Neoplasia; while the portal vein and its tributaries, - which could not be made out, and no doubt were seriously interfered with in their course.

Extending from the lower part of this
Tumour was another large dusky mass, consisting of lymphatic tissue and glands, and lying deeply situated at the horse of the lumbar vertebrae as far as the bifurcation of the aorta. It occupied the whole space between the kidneys, excluding the large vessels from view, and measuring 2-2½ inches in antero-posterior thickness. It was greyish white in colour and presented at its anterior surface four or five rounded prominences soft and pulpy to the touch. These on dissection proved to be oval in shape, varying in size from a walnut to a hen's egg in size; and the greater part of the mass was found to be made up of about a dozen of these swellings, arranged irregularly about the middle line. They represented the lumbar lymphatic glands, which, from their extreme engorgement and dilatation, had become transformed into mere thin-walled sacs. They were filled with an inspissated greasy yellowish white material, in character almost identical with thick cream. There

* Vide the accompanying sketch (over).
This sketch (made after removal from the body) will give a rough idea of the appearance presented by the second mass referred to, consisting of the lumbar glands (a. a. a. a.) encased and projecting on the anterior surface of the general mass of lymphatic tissue (b. b. b. b.) surrounding the abdominal aorta and inferior vena cava.
was no rupture to be made out at any part of these related lymphatic structures. Further dissection in the mass revealed the trunk of the abdominal aorta, with the inferior mesenteric artery passing forwards from it to emerge anteriorly at about 1 inch above the lower margin of the mass. Higher up the renal arteries were discovered running out to the lateral margins of the mass; while on the anterior surface, about half an inch from its upper end, the superior mesenteric artery made its appearance.

The inferior vena cava was dissected out and was apparently peculiar, but deviated somewhat to the right in its course through the mass.

Attention was next directed to the discovering of the thoracic duct. The recognized position (opposite the 1st and 2nd lumbar vertebrae) of the commencement of the duct in the vasa praevaenum chyli was occupied by the mass of neoplastic and chronic inflammatory material, producing such a confused matting together of all the tissues in these parts as to render the discovery
of the duct on this side of the diaphragm would, in my opinion, be impossible. I proceeded therefore to the thorax, in the hope of being able to trace it from thence into the abdomen, and found the tissues of the posterior mediastinum (to which the lungs had been adherent) filled with dense fibrous material following the course of the aorta and closely adhering to that vessel.

After some careful dissection the duct was traced lying between the aorta and vena azygos, and in connection with one or two lymphatic glands at the root of the lungs. It is noteworthy that either in these glands or anywhere in the surrounding tissues there was no trace of chylous matter; nor was there any of that chylous injection of the subpleural lymphatics which has sometimes been observed in these cases.

The condition of the duct was as follows: it was irregularly dilated, having a nodular or varicose appearance, and the largest dilatation reached the size of a filbert nut. The duct-lumen was filled with creamy material, similar to that found in the
Lumbar glands, which showed no signs of adhesion to the walls. The vessels walls were thicker opposite the Nanaoco portions than elsewhere, while in some of the wider portions they were much attenuated—The interior of the duct was of a white colour with here and there a few reddish streaks.

An attempt was next made to introduce a Syring from the thoracic into the abdominal part of the duct, but it failed to pass further than the level of the diaphragm, whether owing to occlusion of the duct at this level or to a deviation in its course from contraction of the surrounding tissues, I was unable to discover.

The proximal end of the duct passed naturally into the subclavian vein, which vessel appeared quite natural; and, entering the duct near its proximal end, were several lymphatic trunks, no doubt concerned in the return of lymph from the upper body portions of the body. They contained no chyle.
Microscopical examination of the chylous material removed from the duodenum, small bowel, and mesentery, in each case, showed it to consist mainly of finely molecular fat, with a few large nucleated epithelial cells.

Accompanying are sections of some of the abdominal contents:—

1. Section of portion of pancreas from tumour mass (dogwood) shows much interstitial cirrhosis of the gland, and what appears to be broken down scirrhous malignant tissue.

2. Section of omentum shows cancerous deposit and much cirrhosis in parts: in some places there is loss of increase of fibrous tissue, and in some, adipose tissue predominates.

3. Section of mesentery (dogwood) shows typical scirrhous carcinoma in a mesenteric gland.

4. Section of liver (carcinoma) shows increase of fibrous tissue chiefly of portal canals.

5. Section of small (lutein). shows largely dilated lacteal vessels.

* Owing to an unfortunate error in the hardening fluid employed a good deal of decomposition occurred in the parts before the sections were made.
engaged with chyle, lying in the submucous layer, in the submucous layer, and some also between the two layers of the muscular coat.

The 3 remaining sections of kidney and suprarenal body— all show considerable cirrhosis.

The chylous ascitic fluid, drawn off on two occasions during life, gave the following results on examination:

- Of the first specimen there were 125 ounces, alkaline in reaction and of spec: grav: 1028.5
- Of the second specimen there were 85 ounces, neutral, and of spec: grav: 1022. Both were milky in appearance and remained of the same consistence after standing for many days. There was a slight deposit of red blood corpuscles, and a distinct creamy layer formed on the surface of each. They coagulated heavily on boiling or on the addition of acetic acid. They contained no sugar. In water they readily diffused reddening it phalescent.

Microscopically, there were a few red blood
corpuscles and some large nucleated epithelial cells, but the main part of the fluid consisted of minute molecular matter which did not stain with ordinary nuclear dyes and represented fat in a state of fine emulsion.

Submitted a specimen of the former fluid to the Clinical Research Association to be analysed quantitatively, with the following results:

The fluid contained:

<table>
<thead>
<tr>
<th>Component</th>
<th>Amount</th>
</tr>
</thead>
<tbody>
<tr>
<td>Water</td>
<td>94.24%</td>
</tr>
<tr>
<td>Solids</td>
<td>5.76%</td>
</tr>
<tr>
<td>Albumin</td>
<td>4.05%</td>
</tr>
<tr>
<td>Fat</td>
<td>0.21%</td>
</tr>
</tbody>
</table>

I might add that a specimen of the fluid which has now been kept for more than 12 months remains very much the same in appearance as when first drawn off. Microscopically, the only difference is that there are now in it a number of acicular crystals, arranged in bundles, which I take to be crystals of the fatty acids.

Before concluding the account of this

* Treated with a solution of Osmic Acid (1 per cent), the fluid became of a brown colour.
case there remain a few points which call for further comment.

First, as regards the diagnosis arrived at during life.

At first examination, the patient was evidently suffering from chronic bronchitis and emphysema and presented besides the signs of anaæmia, ascitic and pleuritic effusion, scantiness of urine, and an enlargement of the liver which was regular and not in any way nodular.

From the condition of the heart and of the urine, and from the limitation of the oedema to the lower half of the body, it seemed right to exclude organic disease of the kidneys.

The heart was apparently free from mural lesion but was decidedly restless in action.

As a whole, therefore, prior to the abdominal tapping, the case was considered as one of gradual failure of the right ventricle from the insufficiency caused to the pulmonary circulation by the emphysematous condition of the lungs.

After the evacuation of the ascitic fluid, however, it was evident that there was
an escape into the abdominal cavity from some portion of the Chyliferous System - a condition which is recognized as most frequently brought about by an obstruction to the thoracic duct.

The most careful examination of the abdomen led to only indefinite results. Owing to its obscurity (which was nevertheless characteristic), the pancreatic newgrowth was not made out; and finally the obstruction to the thoracic duct was regarded as dependent, probably, on thrombosis of the great veins of the neck following or the cardiac failure.

The results of the post-mortem examination left no doubt as to the obstruction of the thoracic duct, which, from the morbid appearances, seemed to have been produced at the level of the peritoneum chylolytic duct pressure from the pancreatic newgrowth.

The actual point from which the chyle escaped into the abdominal cavity was not apparent. It might conceivably...
have been due to rupture of the receptaculum chyli or alteration into it by the tumour growth; but this would hardly have eluded notice, and probably, had it occurred, the fluid evacuated would have been much richer in chyle. In the whole I am inclined to think that the escape took place from rupture of a number of the smaller dilated lacteals.

From the signs of chronic "lymphatic oedema" (i.e., increased formation of fibrous tissue and fat) seen in most of the abdominal contents, it seemed certain that the obstruction to the duct had been partial, although the absence of signs of development of a collateral circulation for the chyle was somewhat against this assumption.

The cannula bile duct, although much involved in the tumour growth, apparently remained pervious, as witnessed by the absence of jaundice and also by the fact that chyle was produced, in spite of the diseased condition of the pancreas. Too much stress, however, must not be placed on the latter point, for, according to Professor
Cohnheim\textsuperscript{11}, the presence of the pancreatic juice in the intestine does not seem to be absolutely essential to the production of chyle, and "we meet," he says "with express statements in the literature that, in spite of extreme atrophy of the pancreas, the chyle vessels were found distended by white chyle."

Lastly, the atrophied condition of the spleen was remarkable, not merely from its contrast with the enlargement and chronic oedema of the other abdominal viscera (notably the liver), but also from the fact that its very proximity to the seat of new growth would presumably have rendered it all the more liable to an interference with its lymph return. This condition is, apparently, only to be explained by the found that in this case the interference affected not the lymph return, but the arterial supply of the gland.

This concludes the account of my case of obstruction to the thoracic duct, and I shall now pass, without further introduction, to the general consideration of that subject.

\textsuperscript{11} Leckie in Sir: Pathology (Soc. Soc. 1876) Vol. III p. 945.
Obstruction of the Bronchial Duct.

The aetiology of this condition may be classified as follows:

Obstruction due to:
1. External pressure on the duct,
2. Changes arising in its walls, and
3. Changes arising within its lumen.

1. Obstruction by external pressure is produced by:
   i. Aortic Aneurism.

   Two cases have been recorded by Sir W. Turner, who also refers to cases recorded by Valsalva, Santorinio, Baunicke, and Bennett. In all, the aneurism was either of the descending part of the aorta or of the thoracic aorta, with both of which the duct is in close anatomical relation.

   (i) Tubercular glands in the posterior mediastinum.

   Cases recorded by Otto (2) and Mortier (3).

   (ii) Carcinoma Tumours.

   Cases by Virchow (4) and Kepple-Seyler (5).

   (iv) Cicatricial Contraction in posterior mediastinum.

   Cheyne (6) gives this as a cause of obstruction but without stating his authority.

---

(3) Philos. Trans. 1841, p. 21.
In all four of these conditions the presence sets up inflammation of the duct walls and thrombus; and these apparently are more accountable for the actual obstruction of the duct than is the obliteration of its lumen by compression, which, in every case (from the nature of the disease), can only be of transient aspect.

2. Obstruction from changes arising in the duct walls.

Owing to the fact that marked changes in the duct walls are nearly always found co-existent with thrombosis of its contents, and that each may be either cause or effect of the other, it follows that in many cases no little difficulty is encountered in determining whether they should be classified under this or the following heading. It seems, however, proper to place here:

(i) Congenital Malformations of the duct, of which cases are recorded by Broschlet (1), Dr. Cayley (2), and Dr. Cusworth (3).

(1). Du Syst. Lymphat. Nîmes de concours 1836.
(3). Cited from Tanner's Pract. of Medec. 1865. p. 795. The case was recorded in Cusworth's Pathology. Anat. and Pathology, Plate.
(i) Tuberculosis of the Duct.

This condition, starting in the duct wall, has been seen by Ponfick (1) and Stilling (2), and by Dr. Whitley (3) who gives a very full account of one such case.

(ii) Cancer of the Duct.

According to Vinncke (4), may sometimes originate in its walls.

(iii) Simple Inflammation of the Duct walls is probably of very rare occurrence as a primary condition. Andral (5), however, regards it as a cause of obstruction, and Vinncke (6) quotes the case of Woman as an example.

I should also mention here that Dr. F. J. Roberts (7) remarks incidentally that “obstruction of the thoracic duct is said to arise from disease of its valves.”

3. Obstruction from changes arising within the lumen of the duct.

I have already alluded to the close connection of thrombosis of the duct contents with changes in its walls. Independently, however, of changes, thrombosis and occlusion may originate in the duct.

(3) J. of Anatomy, p. 523.
(4) J. of Anatomy, p. 523.
(5) J. of Anatomy, p. 523.
(6) J. of Anatomy, p. 523.
from the presence of purulent, tubercular, or cancerous material, or more rarely of the filaria sanquinis:
or, as the result of thrombosis in the large veins at the root of the neck in cases of uncompensated heart-failure.
Examples of the latter have been recorded by Oppolzer, 
Pettenkofer, and Rokitansky, 
and the case recorded by Sir John Stewart, 
is probably to be regarded as another example of the former class, cases were recorded by Andral, Sir A. Cooper, 
Humphry, Vilpando, 
Warms, and 
Dr. Stephen Mackenzie.

So much for the etiology of thoracic duct obstruction; next as regards its:

Pathology and Morbid Anatomy.

The morbid appearances vary considerably according to the mode of production of the occlusion. Thus, as the result of congenital Malf ormation, Dr. Cayley found Strictures.

(8) 1863. 1859. p. 220.
of the duct near its entry into the Subclavian vein. Here the duct was occluded by a thrombus, while below the level it was much dilated resulting in rupture of the Receptaculum chylus. (1)

On the other hand, dilatation and thrombosis in the whole extent of the duct, was observed due to congenital malformation, was observed by Carrell. (2)

Again, if obstruction be dependent on inflammation, the following changes take place in the duct. The external coat becomes thickened and infiltrated with cells, the intima becomes opaque and is stripped of its endothelium, while the lymph congeals and blocks up the channel. The thrombi thus formed may become organised and permanently obliterate the lumen of the duct; but, apparently, they may occasionally disappear or suppurate, and their products entering the circulation may produce pyaemia (as in the case of worms). (3) As the result of inflammation therefore, it may find the occluded duct the seat of

(2) K. Tanner's Practice of Medicine 1865. p. 798.
Narrowly circumscribed narrowing, or contraction throughout the whole or part of its extent, and splaying, converted into an un没有什么 fibers cord (Lundel (1)).

In cases due to external pressure, narrowing and occlusion of the duct is observed above the point of obliteration up to the entry of the first collateral branch. Above that level the duct may be found dilated, but this is not constant, and Junculea says (2) is usually absent when the obstruction has taken place gradually, allowing time for the development of collateral channels. Nor frequently but, apparently for the same reason, not constantly, dilatation occurs below the contracted area.

In cases where obstruction is dependent on Venous thrombosis in Heart Disease, the duct is found dilated throughout, and its walls may be thickened, but there are no other signs of change in them.

Where thrombosis occurs from the presence of filariae sanquirinis, as of prominent, cancerous or tubercular material, there is considerable irregularity in the

(1) Précis d'Anat. pathol. p. 441. Vol II.
condition found. The duct may be dilated throughout the whole or part of its extent, the dilatation being nodular or varicose; and in some cases this is associated with areas of contraction and obliteration of the lumen, due to inflammation of the vesicle walls secondary set up by the nature of the duct's contents.

Thus in Dr. Mackenzie's case (1) which was due to fibrosis surrounding the duct was simply punched and dilated in its lower portion, hitherto it became unprevis and could not be faced, while above this level to its termination it resumed its normal calibre.

Again, in the cases recorded by Anval(2), in which the duct was filled with prominent and cancerous material, its dilatation is described as being of a nodulated or "bosselé" character; and this he attributes partly to the accumulation and adhesion of thromboid material around the valves, and partly to thickening of the walls between the valves. It seems, however, to be more properly accounted for by the normal structure of all the lymphatic vessels (including therefore

---

(2) ibid. p. 440. Vol. II.
to the lacteals as well as the thoracic duct), the walls of which, according to Gray (1), expand above the attachment of each valve-segment into a pouch or sinus, and so, when distended, assume a knotted or beaded appearance.

A similar irregularly dilated condition of the duct has been several times observed as a sequence to plugging of the duct by tubercular caseous material. Two cases were recorded by Sir A. Cooper (2) in 1798, and in 1826 M. Landrin (3) gave a description of the condition.

But besides this secondary effect of the tubercular contents on the duct walls, it is now recognised that in some cases, the tubercular condition commences in the duct walls. Phipps (4) first drew attention to this fact and, from observing it in a case of acute general tuberculous, suggested it as the probable cause of that condition. In 1887 Stilling (5) placed six cases on record. In all six the abdominal organs were extensively infiltrated with tubercles and the internal surface of the duct.

(1) Gray's Anatomy. 11th Ed. p. 50.
(4) Vide: Coates' Manual of Pathology (1877) p. 207.
was studded with milky tubercles: and in five of these cases death took place from acute general tuberculosi.

In Dr. Whitley's "case the disease was propagated from tubercular enteritis in a phthisical patient and death resulted from tubercular meningitis. In this case the internal surface of the duct was studded with milky tubercles and in places giant cells were found. This tissue was all to the inside part of the duct, and more externally was fibrous tissue formation; and "from these appearances" he says "one is forced to conclude that the primary tubercular deposit in the wall of the duct led to its closure and finally to complete obliteration": but he adds, "the part played by the inflammatory action excited by the deposit which led to the complete fibrous plugging must not be overlooked." The central portion of the duct was converted into an impervious fibrous cord, below which the duct was dilated and its walls thickened except in the reseptaculum chyli, where from attrition, spontaneous rupture occurred "some months

before death. Above the obliterated portion, the duct gradually returned to its normal condition, and joined naturally into the subclavian vein.

Artenuation of the duct walls has never been observed in its entirety, but only occurs in limited areas, following an extreme dilatation or possibly also, suppuration. It may lead to spontaneous rupture of the duct, as in the case just mentioned, or in that described by Lévy, to which I have already alluded (p. 31).

In both these cases, the rupture took place in the receptaculum chyli. In one case recorded by Bassiers which is quoted by Dr. Cumming, rupture took place in the thorax opposite the third dorsal vertebra.

Most often, however, the duct remains intact and rupture occurs in the cervical region.

Thus far I have spoken chiefly of the histology of the walls of the duct and made but slight reference to the state of its contents. Where not completely obliterated, the lumen of the duct is usually

found plugged with a fibrinous thrombus, or with partially coagulated lymph which may be altogether undetached but is generally adherent about the valves and sometimes to the intervening portions of the walls also. From its admixture with chyle the coagulated lymph is usually of thick, soapy consistence: it may contain the filaria, or purulent, cancerous, or caseous tubercular material.

So much for the morbid anatomy of the duct itself, in the lesion of obstruction. I shall pass next to the consideration of the morbid effects to which that lesion gives rise.

At the outset it would seem obvious that an interpolation of abnormal resistance to the lymph-stream, such as the lesion involves, must be followed by marked effects upon the return, not of the chyle only, but also of the lymph from the greater part of the body. Such, however, is by no means always the case, and indeed experience goes to show that in many
the lesion & may be unattended by any
grave effect upon the lymph stream, and may
even present no signs whatever during life,
due to the establishment of compensation.
This, as a rule, is effected mainly by the
provision of some other channel along which
the lymph and chyle may find their way
to the venous system, but partly also by
an increase in the lymph-absorbing action
of the bloodvessels. As regards the former,
Sir WT Turner (whose paper discusses
the subject very fully) says that such
channels are provided in two ways:
(1) by the establishment of a collateral
circulation,
and (2) by communication of the duct with
other veins besides the left subclavian.
He shows that (1) collateral circulation
may be established by:
(1) splitting of the duct into or near branches
running parallel and reuniting at some
point higher up in the thorax;
or (2) by the existence of a collateral channel
separated from the main duct by a
considerable interval, (of which one


* This duplication occurs in the natural state with sufficient
frequency to be regarded by anatomists as a normal variation
in the duct.—vide Gray's Anatomy (1858) p. 582.
of Andral's (1) cases forms another example in addition to those given by Sir W. Turner; (2) for (i.) by anastomosis between the vessels of the thoracic duct and those of the right lymphatic duct, in cases where the thoracic duct is obliterated throughout.

The vessels involved in this anastomosis are the lymphatics of the liver, upper abdominal wall, thoracic wall (intercostal, pleural and anterior mediastinal vessels), and neck.

Thus in Röhrig's experiment (quoted by Zucchi (2)) of ligation of the duct in dogs at its entry into the subclavian vein, there was no stagnation of chyle and it was seen flowing through the lymph-vessels of the neck and thoracic walls to reach the right lymphatic duct.

Sir W. Turner mentions Cruikshanks' disease in this connection and I would add also Rokitansky's (3) case of obstruction of the duct, in which the subpleural lymphatics on the upper surface of the diaphragm were formed encased with chyle.

(6) Communication of the duct with other veins besides the left subclavian, namely, with the three azygos major or one of its

---

(1) cited by Cruveilhier's Anatomie Pathologique, T. 1, p. 226.
branches, has been observed a few times (by Wurtzer and others) according to Sir
Wm Turner.\(^1\)

These then are the provisions towards compensation of which the lymphatic system
is capable; there remains yet the other provision, namely, that in the part of the bloodvessels.

Experiment has shown that in a limb the total lymphostasis by the lymphatics may
be cut off without ordnma resulting, the bloodvessels taking up the functions of the
lymphatics until new lymphatic channels are formed. No doubt the tendency to
ordnma in these cases is to a great extent
overcome, as the late Professor Colinhi\(^2\)
has shown, by the fact that the pressure thus set up in the bloodvessels is directly
opposed to the normal pressure of lymph-transudation.

It seems probable, however, that this
vicarious action of the bloodvessels is but rarely
required, for in the majority of cases the
obstruction of the duct comes on gradually,
and thereby allows ample time for the
development of collateral channels
for the lymph.


\(^2\) Colinhi's Lect. at the Pathology - Sup Soc. Edit.

Vol 7 - p. 510.
Thus, then, by the establishment of compensation, we are able to explain the fact that obstruction of the duct may occur without any resulting accumulation of lymph or chyle: we have next to consider the question, what are the effects produced when compensation fails to be established?

At this point some difference of opinion prevails.

There is no doubt that stagnation of chyle always occurs and, by rupture of the duct or chyliferous vessels, may lead to chylous ascites or chylous pleural effusion, or again the chyle may run back into the renal tracts and give rise to chyluria—apparently also ascites is always found; but

"edema of the skin and extremities is not of constant occurrence in this lesion. Professor Columbus," whose work on Pathology discussed this subject at length, states that not only the edema, but also the ascites (except where due to ruptured chyle-vessels), are produced not by the abnormal resistance to the lymph stream but

by increased lymph-transudation from the blood-vessels. 

Now so far as the ascites is concerned, an element of uncertainty is introduced in almost every case by the existence of some such complication as tuberculosis or malignant peritonitis, uncompensated heart-failure, or interference with the inferior vena cava or other large abdominal veins. But with regard to the oedema there can be no uncertainty: for, as Professor Cohnheim states, it has been clearly shown, both by physiological experiment and pathological observation, that total occlusion of all the lymphatics of a part is never followed by oedema, provided the secretion of lymph within it is normal.

The final conclusion to which Professor Cohnheim comes, and in which Professor Coats evidently concurs, is that:

Stoppage of the lymph-flow cannot of itself lead to oedema or dropsy (with the one exception of ascites due to rupture of chyle vessels), although it is clear it may aggravate an oedema whose cause is to be found in

(11. Manual of Pathology. 1889. p. 87.)
Some other condition. From this, then, it would appear that the only marked effect to which uncompensated obstruction of the thoracic duct can of itself give rise, is stagnation of chyle with its consequences. These latter I shall directly consider in detail; but first the question arises—under what circumstances does compensation fail to be established?

Sir Wm. Turner (1) says that he is inclined to believe that compensation is established in all cases in which the obstruction causes no gradually. Thus however seems doubtful. For in many of the uncompensated cases recorded it seems evident, both from their history and their etiology, that the onset of the obstruction must have been gradual; and probably, (as certainly in those cases dependent on heart lesion) the condition of the great veins at the root of the neck has more to do with the want of compensation than has the rate of onset of the obstruction.

I shall next consider in detail—

The marked effects of uncomplicated obstruction of the duct.

1. Dilatation and chylous engorgement of the lacteal vessels takes place, from their termination in the receptaculum chyli back to their commencement in the lacteal radicles of the intestinal villi. It has been observed in several cases (Morton, Brouchet, Senturinio, Valsalva, Rotenstein), and I have already alluded to the remarkable beaded appearance of the lacteals in this condition allowing them to be readily traced in their course in the intestinal walls and up to the nearest range of mesenteric glands. In the mesentery dilated lymph channels and glands are easily discerned on the cut surface, white in the intestine the villi appear specially prominent. These dilated vessels are one and all filled with a white soapy material, which readily disperses in water and renders it foetid. Seen under the microscope, it consists mainly of finely granular matter (representing fat in a finely emulsified condition), and there are besides, a few nucleated epithelial cells and fatty crystals.

(1) The references to these cases have been given above. pp. 27, 28, 130.
Quincke "asserts that in some cases the mesenteric lymphatics alone are dilated, and only in certain parts of the mesentery; and in support of this assertion he quotes the cases of Langhans" (1) and Marshall-Stevens (2).

It should be noted however that in neither of these cases was the thoracic duct examined.

A somewhat different condition of the lacteals was found in a case recorded by Sir T. Fawcett Streatfeild (3) where the duct, although not actually examined, was no doubt obstructed as the result of tuberculous disease. In this case the author says "scattered throughout the whole small intestine was a number of white-yellow patches varying in size from that of a pinhead to that of a small bean" and these were situated some in the mucous membranes, some in the submucous layer, and were due to dilatation and elongation of villi and small lacteals respectively.

Rupture of the dilated vessels may occur and give rise to "varicose and extravasation of clots within the mesentery and in the

(3) Arch. f. Syphil. Bd. VIII. S. 1675.
walls of the intestine (or intestine), or, where the rupture is into the abdominal cavity, to chylous ascites.

Lastly, it may be mentioned that in some cases the lacteals, after the tension had been removed from them by rupture, apparently were able to return to their original condition. Thus in Dr. Whitla's case, in which there was complete obliteration of the duct, producing dilatation and rupture of the receptaculum chyli: some months before death, the lacteals were not found dilated nor engorged.

(2) Ascites.

On the relation of thoracic duct obstruction to the production of ascites I have already dwelt sufficiently*, and here intend only to consider the condition known as chylous ascites, in which the fluid escaped is opaque and milky from admixture with chyle. In all the cases recorded of this condition, the fluid was found to be secreted very rapidly and in large amount, requiring frequent tapping. In Dr. Whitla's case the fluid, which


* Vide Supra: p. 42.
Was nearly pure chyle, was calculated to collect in the abdomen at the rate of rather more than 31 ounces per day—

Smirke states (and this fact was observed in Dr. Whitley's and in my case) that the fluid standing forms a layer of cream on its surface and that it requires decomposition for a very long time (also observed in my case). He adds that in the cases recorded the fluid varied in specific gravity from 1007 to 1016, that it contained a small quantity of albumen, and that its fatty contents varied from 0.5 to 1.8 per cent. In Dr. Whitley's case the fluid contained 0.2 per cent of sugar that I could find no trace of sugar in my case.

The following table gives the quantitative analyses of the fluid in Dr. Whitley's and in my case, and also the analysis of the normal chyle during digestion—

<table>
<thead>
<tr>
<th></th>
<th>Dr. Whitley's Case</th>
<th>My case</th>
<th>Chyle (2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Water</td>
<td>94.08</td>
<td>94.24</td>
<td>90</td>
</tr>
<tr>
<td>Solids</td>
<td>5.91</td>
<td>5.76</td>
<td>10</td>
</tr>
<tr>
<td>Albumen</td>
<td>2.87</td>
<td>4.05</td>
<td>3</td>
</tr>
<tr>
<td>Fat</td>
<td>1.03</td>
<td>0.21</td>
<td>3</td>
</tr>
</tbody>
</table>

From the above it will be seen that in my case there was a great admixture

(1) Zinsser's Cyclop. Path. of Med. Vol. VI. p. 539
(2) These are the figures given by Professor Rutherford in his lectures on Physiology.
Of serum with the chyle train in Dr. Whitley's case, in which the fluid escaped into the abdomen from a large rupture of the receptaculum chyli and consisted of almost undiluted chyle.

As to the way in which obstruction of the duct leads to chylous ascites, I find that two cases only were due to rupture of the receptaculum chyli (Canley (1) and Whitley): while in the majority of cases (Pokitansky(2), Morton(3), Oppolzer(4), Langhans(5), Marshall Hughes(6), and Pye-Smith(7)), and probably in my own case, the escape of chyle took place from rupture of a number of the minute dilated lacteals.

Professor Coats(8) warns us against confusing chylous ascites with a condition (mentioned also by Professor Columbain(9)) known as Ascites adiposus, in which the fluid is milky in appearance but owing to fatty degeneration of the cells it contains, not to admixture with chyle. He says that the distinction between the two conditions is that in chylous ascites the fat is free, while in ascites adiposus it is in cells. The two conditions appear to have been confused by some writers, notably one French writer, M. Estulle (whom Dr. Whitley(10) quotes) who goes so far as to maintain...
that "all cases of chylous ascites known up to the present time and followed by necropsy, are cases simply of chylotic tuberculosi, carcinous or neo-membranos peritonitis". These views Dr. Whitta styles "fantastic theories" when looked at in the light of his own case with its all-apparent cause; and he adds, that after such a case one is justified in assuming that there is probably a small aperture in some of the minute lacteals, which, though not visible, can still permit the direct escape of chyle.

Same difference of opinion prevails as to the effect of the chylous fluid on Brano's membranes. Chirncke asserts that it is a perfectly indifferent fluid for the peritoneum as well as for the pleura: on the other hand Dr. F.T. Roberts states that, "as proved by Dr. Caley's case (in which rupture of the mesentericum chyli occurred and subsequent peritonitis), the escape of chyle into the peritoneum may set up fatal inflammation: It appears however that Dr. Caley's is the

only case in which the chylous effusion was ever considered as producing fatal peritonitis, and certainly the number of cases of chylous ascites in which no such fatal peritonitis ensued would seem to be opposed to the assumption of Dr. Roberts.

3. **Chylous Pleural Effusion**

has been recorded in a few cases of obstruction of the duct. Thus, Bassini found it produced by rupture of the thoracic duct in the thorax; and in Rokitansky's case it existed along with chylous ascites and was due no doubt to an escape, either by rupture or effusion, from the subpleural lymphatics, which were found distended with chyle. In Langhans' case it was present as one side only, along with chylous ascites.

4. **The condition of the Abdominal Viscera**

**especially the kidneys.**

The abdominal viscera are usually found enlarged and congested. Microscopically, they show dilatation of their lymphatics.

(1) Reference given on p. 36.
(2) Reference given on p. 30.
Which, Professor Colinckin says, "is usually not conspicuous in the liver and kidneys.
As a sequel to the chronic lymphatic congestion and oedema of the abdominal contents, this is
usually found hyperrophy of connexive tissue and fat - in my case, most marked in the
psoas, and rectus, and also observed in the liver, kidneys, and subcutaneous bodies.
Besides dilatation of the visceral lymphatics in some cases dilatation and engorgement of the
pampiniform lymphatics of the abdomen or pelvis have been observed.

But more remarkable and of greater pathological
influence, is a condition which has been seen
a few times in those cases, namely, the
evidence of the flow of chyle in a retrograde
direction; so that not only was there chylous
engorgement of the lumbar, iliac, and inferior
sacral lymphatics, but at the same time, the lymphatics of the
pelvis of the kidney were so distended with
chyle as to be visible to the naked eye as small
yellow points. Such was the condition
found in Mr. Stephen Mackenzie's case, when it
was due to obstruction of the duct by the Filaria
sanguinosa and gave rise to chyluria. It

was also observed in my case, in which, however, there had been no chyluria. — [Quincke says, that "after ligation of the thoracic duct in dogs Röhrig (Ber. d. K. Sels. p. 0. Wissenschaft, 1874) could frequently trace chylous congestion into the lymphatics of the kidneys and muscles, especially in aged animals." — These two cases have an important bearing on the explanation of the pathalogy of chyluria — a subject on which there has been no little controversy since Sir Rodolph C. put forward his view in 1861 that chyluria is due to a direct communication between the chyle-carrying vessels and the urinary tracts.

Sir Currey, in discussing this subject in 1879, said "Resuscitation of the into the lymphatics of the kidney from the testicular vessels is almost impossible; the small number of the renal trunks, the valves, and other routes were open to a retrograde current, oppose it, and in no instance of obstruction to the duct has dilatation of the renal lymphatics or chyluria been observed. The anatomical relation of the reservoir of the chyle, the thoracic duct, and the peripheral

(2) Rev. Lancet, 7th. Set. of Medicine (1874) article Chyluria (Ceylon).
Lacteal System are such that are cannot admit the possibility of any direct communication with the urinary passages. Without going into the other theories on the subject, it is sufficient here to state that Dr. Curie's view is apparently adopted by most of the works recently published; and certainly Dr. Mackenzie's case and my own prove beyond a shadow of doubt the possibility of the backward flow of chyle from the duct into the renal lymphatics.

The annexed rough sketch (which I have constructed from the description of the abdominal lymphatic system given in the last edition of Smirnoff's Anatomy) is intended to indicate, in purely diagrammatic fashion, the communication between the lacteal system and the renal lymphatics, to reach which it will be seen that the chyle must flow back from the receptaculum chyli through the lumbar lymphatic plexus.

That this backward flow started from the receptaculum chyli is, I think, a sufficient explanation (as a glance at the diagram will show) of the fact that

Handbook of Mad: Anthology, by Henshaw, Juan. 1894. p. 158.
Diagram of Abdominal Lymphatic System

[Constructed from the account given in Quain's Elements of Anatomy, (1895), Vol. II, Pt. II]

Note: The chyle-carrying vessels are depicted in red ink.
that the lymphatics of the cecum and of the ascending and transverse colon escaped chylous engorgement. At any rate it is noteworthy that, written in D. MacKenzie's case, was there any appearance of the backward flow of chyle into any other visera than the kidneys, which appears to favor the assumption that for some reason the renal lymphatics prove more accessible to a retrograde current than any of the other efferent branches of the lumbar glands.

Why there was no chyluria in my case I fail to see, unless it be that the backward pressure of the chyle which led to engorgement of the renal lymphatics was insufficient to cause rupture of those vessels into the renal pelvis, such as appears to have occurred in D. MacKenzie's case.

Before leaving the subject of retroperitoneal chyluria, it ought to be said that there seems to be found for believing that some of the many recorded cases of lymphorhein...
(notably that of Dr. Casswell (1)) were produced
in a similar way, namely, by a retrograde
flow of chyle from the lumbar into the inguinal
flanks and thence into the lymphatics of
the serotum, groins, or thighs, these vessels
being usually found dilated in this condition.
Simicske (2) considers that the direction
of the current of chyle in these cases is
probably dependent on the action of gravity,
aided by insufficiency of the valves.

(5. Anasarca and Sclerosis.
As to the production of anasarca in cases
of obstruction of the thoracic duct, I need
add nothing to what I have already said.
Where it occurs, there appears to be a
tendency, as with the abdominal contents,
to the rapid production of connective tissue
hyperplasia and increase of fatty tissue.

This last morbid effect to which I have to
allude is the occurrence of:

(6. Emaciation.
This may present to an extreme degree,
but, on the other hand, that it does not occur

(1) Reference from above. p. 28.
* Vide supra. p. 41-2.
in many cases was remarked by Sir Wm. Turner, and he holds that its absence may be taken as the strongest proof of the sufficiency of compensation in those cases. Its uncertainty as a test, however, is shown by Dr. Whitley's case in which, although large quantities of chyle were removed from the abdomen, no emaciation occurred. Moreover in many cases a further complication is introduced owing to the fact that the primary cause of the duct lesion is in itself a wasting process (e.g. Tubercle or Cancer).

In conclusion I shall briefly refer to the
Symptoms, Diagnosis, and Treatment of Obstruction of the duct.

From what I have already said on the subject of its morbid effects, the symptoms of this lesion will be sufficiently apparent, and a recapitulation of them would entail only needless repetition.

As regards the Diagnosis— this is called

(2) Reference from above. p. 29.
for only in cases where the lesion is uncompensated, and even in these cases the signs are usually so obscure as to leave the diagnosis a matter of uncertainty. From consideration of the myriad effects and from the clinical observation of my own case, it seems that there is no sign which can be regarded as altogether pat tro-nomical. There are two conditions, however, which are highly indicative of the existence of this lesion, namely, the presence of chyle in the pleural or peritoneal cavities; for these, except in a few instances of traumaticism, have in nearly every case been found to be dependent upon obstruction of the thoracic duct. The presence of chyle in the urine might, in some cases, perhaps, lead us to suspect an interference with the thoracic duct.

The treatment of this condition can only be palliative. The ascites will probably necessitate frequent tapping;
and, in the case of general venous congestion, the heart's action will require regulation.

The course of the disease apparently varies greatly in different cases—from a few days (vide Dr. Cayley's case) to six months or more (e.g. cases of Dr. Wilks and Mr. Whitla).

The fatal termination is no doubt hastened by the defective absorption of chyle as well as by the mechanical pressure from the large venous effusions: but, in the majority of cases, death occurs from a combination of causes, among which the original disease (e.g. Tuberculosis, Cancer, or Heart lesion) probably plays a more important part than does the obstruction of the thoracic duct.

---