On So-called

Congenital Obliteration

of the

Bile-ducts.

(Clinical and Pathological)

by

John Thomson

M.R.C.P.

1891
Preface.

In choosing the present subject for a thesis, I have been influenced by several considerations.

Firstly, within a comparatively short time, two cases of this very rare condition came under my own notice, as well as a few other cases which seemed to throw light on it.

Secondly, when I looked into the literature of obliteration of the bile-ducts in children, I found that, although the subject was in many respects a remarkably interesting one, it had not undergone (even in Germany) the same amount of exhaustive investigation as the other subjects which I had formerly proposed to take up. And,

Thirdly, thanks to the resources of the library of the Royal College of Physicians, I was able to collect details of fully twice as many cases as any former writer on the subject had brought together; and this wider outlook seemed to offer material for getting nearer the truth with regard to several points of difficulty and interest.

J.T.
<table>
<thead>
<tr>
<th>Topic</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Introduction</td>
<td>1</td>
</tr>
<tr>
<td>Original Cases</td>
<td>4</td>
</tr>
<tr>
<td>Case of J. Morrissey</td>
<td>4</td>
</tr>
<tr>
<td>Clinical</td>
<td>4</td>
</tr>
<tr>
<td>Post mortem</td>
<td>6</td>
</tr>
<tr>
<td>Microscopic</td>
<td>9</td>
</tr>
<tr>
<td>Case of G. di Placidi</td>
<td>17</td>
</tr>
<tr>
<td>Clinical</td>
<td>17</td>
</tr>
<tr>
<td>Other published cases</td>
<td>20</td>
</tr>
<tr>
<td>Analysis of Table I</td>
<td>21</td>
</tr>
<tr>
<td>1) Rarity of Occurrence</td>
<td>21</td>
</tr>
<tr>
<td>2) Health of Parents</td>
<td>23</td>
</tr>
<tr>
<td>3) Other Children</td>
<td>24</td>
</tr>
<tr>
<td>4) Character of Labour</td>
<td>24</td>
</tr>
<tr>
<td>5) State of Child at birth</td>
<td>24</td>
</tr>
<tr>
<td>and Prematurity</td>
<td>24</td>
</tr>
<tr>
<td>6) Sex</td>
<td>25</td>
</tr>
<tr>
<td>7) Symptoms</td>
<td>25</td>
</tr>
<tr>
<td>a) Onset of jaundice</td>
<td>25</td>
</tr>
<tr>
<td>b) Character and progress of jaundice</td>
<td>28</td>
</tr>
<tr>
<td>e) Urine</td>
<td>29</td>
</tr>
<tr>
<td>d) Meconium</td>
<td>29</td>
</tr>
<tr>
<td>c) Moions</td>
<td>32</td>
</tr>
<tr>
<td>f) Vomiting</td>
<td>35</td>
</tr>
<tr>
<td>g) Hemorrhages</td>
<td>35</td>
</tr>
<tr>
<td>h) Convulsions</td>
<td>42</td>
</tr>
<tr>
<td>i) Emaciation</td>
<td>43</td>
</tr>
<tr>
<td>j) Duration of life</td>
<td>43</td>
</tr>
<tr>
<td>k) Other morbid conditions</td>
<td>44</td>
</tr>
<tr>
<td>b) Post mortem appearances</td>
<td>45</td>
</tr>
<tr>
<td>a) Peritoneum</td>
<td>45</td>
</tr>
<tr>
<td>b) Liver - its size, colour, substance, surface and blood vessels</td>
<td>45</td>
</tr>
<tr>
<td>c) Gall bladder &amp; ducts</td>
<td>47</td>
</tr>
<tr>
<td>Analysis of Table III</td>
<td>47</td>
</tr>
<tr>
<td>Precise lesion</td>
<td>47</td>
</tr>
<tr>
<td>Situation of obliteration</td>
<td>48</td>
</tr>
<tr>
<td>Secondary changes</td>
<td>49</td>
</tr>
<tr>
<td>Contents of gall bladder</td>
<td>51</td>
</tr>
<tr>
<td>d) Microscopic appearances of liver</td>
<td>51</td>
</tr>
<tr>
<td>e) Spleen</td>
<td>52</td>
</tr>
<tr>
<td>f) Other morbid conditions</td>
<td>52</td>
</tr>
<tr>
<td>d) Microscopic appearances of liver</td>
<td>51</td>
</tr>
<tr>
<td>e) Spleen</td>
<td>52</td>
</tr>
<tr>
<td>f) Other morbid conditions</td>
<td>52</td>
</tr>
</tbody>
</table>
Pathology of Lesion of Ducts

1) Arrest or Defect of Development
2) Inflammatory or Other Affection of the Ducts themselves
3) Peritonitis and its results

Predisposing Causes
a) Syphilis
b) Injuries, and exposure to cold
c) Dysentery

Conclusions

Appendix I. Descriptions of lesions of gall bladder and ducts in tabulated cases

Appendix II. Cases of Infantile Jaundice, in which the ducts were found to be permeable

Appendix III. Case of Obstruction of the small intestine

Literature
List of Illustrations

Plate 1. Copy of chromo lithograph representing
1. Wickham legg's case. M.T. 8
2. Drawing of liver and ducts from case of J. Morrissey. J.T. 8
3. Section of same liver under low power (microscopie) J.T. Murray 10
4. Section of same under high power ... 10
5. Section of same under low power (cogwood) area of coagulative necrosis under capsule J.T. Murray 14
6. Liver cells from same case showing position of fragments of aspirated bile. ... 14
7. Viscera of infant showing obliteration of the small intestine. ... 83

Fig. 1. Percussion dulness in case of J. Morrissey J.T. 5
2. Diagram of normal gall bladder, ducts & vessels. ... 8
3. ... of bladder, ducts and vessels in J.M.'s case ... 8
4. Outline of normal liver of child at 3½ wks 8
5 & 6. Terminations of bile ducts inside liver cells copied by M.T. 14
7. Diagram explaining mode of occurrence of obliteration of bowel. J.T. 86
Table 1. Details of 47 cases of Obliterated Bile duct

Table 2. Diagrams of the precise lesion of Gall bladder, duct, & duodenum in 30 of the cases page 47

Table 3. Details of 7 cases of fatal Infantile Jaundice in which the ducts were found to be pervious page 77

Four microscopical sections of the liver from Case I are submitted along with Thesis.
Introduction.

We find, scattered about in medical literature, a comparatively small number of cases which are usually described under the title "congenital obliteration or malformation of the bile ducts." These cases present a distinct series of symptoms and may profitably be studied as a group by themselves.

The main clinical facts, stated very briefly, are as follows:

The parents are healthy people, and yet in many cases it is found that they have previously had one child, or several children, similarly affected.

The children themselves are either jaundiced at birth or they become so within the first week or two of life; otherwise they are healthy and well-nourished. In some cases there is a discharge of normal meconium, followed by colourless motions; in others the faces are devoid of colour from the very first. The urine is deeply bile-stained. The jaundice is of a deep greenish tinge and lasts till death, and the motions remain colourless.

A certain proportion of the children die from umbilical hemorrhage within the first fortnight, and of those who survive this period a large number suffer from spontaneous hemorrhages from other situations. The liver steadily enlarges and the spleen also is large. After living some months, the children become more or less emaciated. Fits often supervene, and death ens.
cases in the end, in a state of exhaustion, from some trifling intercurrent disease.

At the post-mortem, the liver is usually found much enlarged, of a very tough consistence, due to biliary cirrhosis, and of a dark green colour, owing to the presence of numerous masses of inspissated bile in the small bile-ducts. In the great majority of cases, there is obliteration of some part or parts of the hepatic, common or cystic ducts, or of the gall-bladder, while, with few exceptions, implication of the blood-vessels or other tubes in the neighbourhood is conspicuous by its absence.

What is the etiology and pathology of these cases?

How are the various symptoms to be explained?

In the present paper an attempt has been made by accumulating all the available details of all the published cases (along with those of an original case) and by looking at them in the light of other clinical facts and of the results of recent experimental investigation, to indicate what data we have where from to form an opinion on these questions.

It may be objected that the reports of individual cases which must form the basis of such investigation are often defective and possibly sometimes incorrect and misleading, but then we must remember that our arguments drawn from this source are
are not to be regarded as forming a chain that is no stronger than its weakest link, but that they rather resemble a line of stepping stones by means of which, although individually they are not all very reliable, one may safely attain to a satisfactory conclusion — always provided one does not attempt to put too much weight in places not able to bear it.
Case of Obliteration of the Bile Ducts.

George Morrissey, aged 3 m., seen first on 6th July 1887 with Mr. Temple.

Complaint - Jaundice and whooping cough. The former dating from the tenth day after birth, and the latter having begun 5 weeks before.

Family History - The father, a tailor, is dyspeptic (he died 7 m. after of carcinoma of the stomach and oesophagus) he shows no sign of syphilis and denies ever having had any form of venereal disease.

The mother seems healthy, has had no symptoms suggestive of syphilis and no miscarriages.

Other children - One, a girl about 10 is alive & healthy. Three have died in infancy, two of bronchitis & one of whooping cough. None of them were ever jaundiced or suffered from haemorrhages. None of them had signs of congenital syphilis.

Pregnancy - During the whole time of her pregnancy the mother was insufficiently fed and lived to a large extent on fruit given her by a friendly fruitier.

Birth - The mother says she was under chloroform for an hour and a half and that instruments were used "because no pains came."

At birth, the child seemed perfectly well and was not at all jaundiced. After about 10 days, he gradually became yellow and has been so ever since.

The mother always thought that his belly was too big but did not observe any special swelling in the right hypochondrium. He never had any haemorrhage from the umbilicus, nor atretism. He was given the breast only and thrived very well until 5 weeks ago when whooping cough began. The umbilical hernia was noticed a fortnight after birth. The mother thinks his urine was always dark and stained the clothes yellow. During the first week, he passed several dark green stools (like those of other new-born babies) but after that time they became...
became clay coloured. They have never had the slightest yellow or brown ting.

About a week ago, when the cough was particularly bad, some dark blood was seen in the motions, and about the same time, there was bleeding from the mouth and nose. The bowels have always been freely open 3 or 4 times a day. The child has never been subject to vomiting and has had no fits. During the last week or so he has been getting so feeble that he makes scarcely any noise when he cries. He has had grey powders and medicines for the cough, but they have had no effect. He never had mumps or a rash.

**Present Condition.** The child is tolerably well nourished but very weak and languid. The scalp is dry and scaly, the fontanelle somewhat depressed. The skin is very deeply jaundiced all over and has a greenish yellow colour. The conjunctiva are also deeply jaundiced. The pupils are medium sized and equal. There are a few spots of acne on the right cheek.

(Fig. 1)

**Abdomen.** Marked prominence over the region of the enlarged liver which on percussion is found to reach in the right mamillary line from the upper border of the 5th rib to 2½ inches below the costal margin — in the mesial line to two inches below the xiphisternum, and in the anterior axillary line to the level of the umbilicus. On palpation, the organ is felt to be very hard and its edge is sharp. Its surface feels smooth with slight furrows.

The spleen is also felt hard and large. Its dulness begins above at the 10th rib and extends downwards for about 2 inches. Its notch is not felt. There is an umbilical hernia, the ring of which admits the point of the forefinger. The abdomen is otherwise healthy. There is no ascites.

**Thorax.** Tolerably well formed. Heart apex beat indistinct and feeble in 5th space just inside the left nipple line. Long Heart sounds normal.
Residual bronchial secretions both lungs, especially behind. Otherwise lungs seem normal.

**Treatment** - Cough mixture, pad for hernia.

**Further progress** - 18th July. Child has been getting steadily weaker and has lost flesh markedly. No subcutaneous haemorrhages, no ascites. During the last few days the stools have been very dark from the presence of blood - almost tarry.

19th July. Weaker, ate none working very noticeably. Pulse 116, Resp. atkins 110, very shallow. Bowels have moved 4 times; motions black from presence of blood, and offensive.

20th July. Rapidly getting thinner and weaker. Pulse 112. Resp. more than 80. Temperature (in rectum) 99.3. On the inner side of each calf there is a small hard nodule, one the size of a big pea and the other half that size. They are not painful on pressure and the veins round about them are enlarged. Motions as before but less frequent. Conjunctivitis in both eyes. Jaundice has varied in intensity from time to time and is now distinctly less deep than it was a week ago. Urine dark greenish and turbid. No tube casts found. A few coloured blood corpuscles. Much amorphous deposit. No albumen.

21st July. Died quietly at 7.30 a.m.

**Post-mortem** at 11 a.m. on 22nd July. Had to be performed rapidly under considerable difficulty and restrictions (bad light, small incision 8")
Child emaciated, skin deeply jaundiced, rigor mortis passed off, abdomen distended with gas. Reddish brown fluid oozing from mouth and mouth.
Thorax - Pericardium normal, no excess of fluid. Heart: muscular substance and valves normal.

Lungs contained no fluid, numerous haemorrhages from 1 to 3 lines in diameter scattered over surface of both lungs. No pleurisy.

Lungs: Right - middle lobe collapsed, rest of lung very engorged. Left - a small portion of upper lobe (anterior margin) collapsed the rest of the lung very engorged. On section, a number of small haemorrhages are seen in both lungs, there is no pneumonitis but a good deal of bronchitis on both sides.

Abdomen - Peritoneum normal as far as could be seen. No adhesions or roughness. No fluid in its cavity.

Liver: much enlarged. Measurements of greatest diameters - from side to side, 6 1/2 inches, antero-posterior (right lobe) 4 inches, thickness, 2 inches. Weight 13 oz. General contour not much changed. Colour, very dark olive green, the fibrous bands which pass over the surface in all directions being of a yellowish tint. Surface somewhat irregular like that of morocco leather (on a larger scale). Capsule not thinned. Edges hard & rather sharp. Consistence, extremely hard & tough. The organ is hard to cut owing to the amount of fibrous tissue it contains. The surface of the section has a very dark olive green colour & is indurated by numerous fibrous trabeculae of a light greenish-yellow hue. A considereable amount of dark green fluid flows from the cut surfaces.

(Plate 1, which is copied from Dr. Weibke's book, gives some idea of the dark green colour of the surface but is by no means accurate or artistic.)

*That is, a good deal more than twice the normal size. - Bich. Hirschfeld in Farbund Handbuch 2. Kindale. 3rd ed. 1863. p. 668.
Copy of illustration in D. Wickham Legg's book.

Shewing characteristic dark green colour of liver in cases of Obliteration of the bile ducts.
Fig. 4.

Outline of normal liver of child of 3½ months — Sija, Duct. Etc.
(In Plate 2 which was drawn from the liver after it had been some weeks in spirit.
the size, contour and morocco-like surface are seen, as well as the condition of
the gall bladder and ducts. Fig. 4 is an accurate outline of the liver
and ducts of a child of exactly the same age as the patient.)

The hepatic ducts are not to be found in their usual form, but in their place,
there is a transverse sinus about 1 1/4 inches in length and 3/16 inch in diameter.
on its lower surface it presents two little sacs bulging downwards (one of
which has been opened in the drawing). Its walls are thick & fibrous, its
lumen is lined with a smooth membrane, and communicates by numerous
openings with the dilated & thick walled ducts in the interior of the liver.
There is no communicating duct between this sinus and the cysctic duct
and no distinct band of fibrous tissue can be found connecting the two.
The gall bladder is rather smaller than usual (3/4 × 3/4); it is folded &
wrinkled and contains about 30 minutes of a brownish watery fluid—
like thin mucus. (see Figs. 2 & 3)
The cysctic duct is small (perhaps 3/4 the normal diameter); it can
be followed right down into the pancreas, and its lower two-thirds or so,
which is of the same calibre as the upper portion, is all there is to repre-
sent the Ductus communis choledochus. Caring to the hurried nature of
the post mortem, the portion of the duodenum containing the papilla was
left in situ but there can be no doubt that it would have been found
penetrated because fluid could be readily squeezed from the gall blad-
er down as far as the middle of the pancreas where the duct was severed.
The blood vessels are quite normal. The connective tissue round the vessels and ducts is possibly a little thickened but certainly shows no evident traces of past inflammation and there is no roughness or other abnormality of the peritoneum covering it.

The pancreas seems normal and the portion of duodenum removed is healthy. The jejunum and ileum contained much gas and a large quantity of blood and feces (they were not opened). Many of the mesenteric glands were somewhat enlarged.

The spleen is large, measuring 3 x 1 3/4 x 7/8 inches. It weighs 1 1/2 oz. It is dark in colour with a distinctly yellowish tinge and is very tough. The mafiphian bodies are indistinct.

The kidneys are rather large, they are dark in colour and much jaundiced. The capsules strip off easily & the organs seem healthy in all other respects.

Microscopic Examination.

Liver. Several pieces of the right lobe were hardened in Weil's fluid and several in spirit; they were cut, stained and mounted in the usual way.

On using a low power (Plate 3) one is struck at once by two things. Firstly, the tissue is traversed by a very coarse network of fibrous tissue which is continuous with the outer layers of the capsule and generally includes several veins in each of its meshes; and secondly, the section...
Section of Liver - Low power.
(Picoscarmin - Farrant)

Showing fibrous network & distribution of bile plugs. (p. 9.)
Section of Liver. High power.

(Zarran - Logwood)

Shewing formation of new bile ducts. (p. 11)
section looks as if peppered over with little dark spots. The largest of these are found to correspond in distribution with the strands of the fibrous network, while the smaller are most numerous in the inner zones of the hepatic lobules.

On closer examination of the section, we find the following conditions:

The capsule is somewhat wavy in outline and is thickened, but very irregularly. In some places it differs little from the normal in this respect while in others it is 6 or 8 times the usual thickness. It is thickest where it is joined by large branches of the intra-lobular network and these places generally bridge somewhat on the surface. It contains several large vessels and sinuses filled with blood and in its inner layers are many bile ducts distended with green or brown masses of inspissated bile.

The fibrous network, already mentioned, is formed by broad bands of fibrous tissue which pass inwards from the capsule and branch round the lobules. Generally the meshes include from 1 to 6 acini in each (mostly 1, 2 or 3) and these are more or less distorted in shape owing to the compression they are exposed to. In some places what appears to be only a portion of a lobule is all that occupies a mesh. The knots, so to speak, of the network are represented by masses of fibrous tissue arising from an enormous increase of the connective tissue normally present in and around the portal spaces, while the connecting strands occupy interlobular spaces. The network is by no means a complete one and there are numerous small islets of connective tissue at points in
between the lobules and sometimes wedged into the substance of them, as it were. In stained specimens, the fibrous bands are usually more deeply coloured along their outer margins owing to the presence there of numerous proliferating cells. This fibrous tissue, like that of the capsule, contains numerous dilated bloodvessels and bile ducts. The arteries appear to be increased in number as well as in size, they are tortuous in their course and some of these seen on section seem to have thickened muscular walls. The bile ducts are also tortuous and have thickened walls, and many of them contain large masses of inspissated bile. As to their distribution, it is not so easy to make out as in the layers of the capsule, but they seem to be more numerous on the whole, towards the periphery of the bands than in their centres.

At many places, at the junction of the liver-lobules and the fibrous tissue one sees most beautifully (Plate 4.) the arrangement of the liver cells into parallel rows and the formation out of them of new bile ducts.

The hepatic lobules vary much in size and also, as already noted, in shape owing to the compression they are undergoing.

The microscopic characters of the liver-tissue vary very considerably in different portions of the organ and even in different parts of the same section.

Firstly, a large proportion of the lobules are composed of very normal looking cells, arranged in the usual way. The nuclei of these are well-formed and colour well and indeed in certain areas they absorb.
absorb the carmine out of the picrocarmine stain in a very remarkably
distinct way, and seem larger than usual, as if in a state of unusual
activity (see Hamilton [87]). In these acini one finds no intralobular
circumsis at all.

Secondly, many of the cells even of the normal-looking lobules con-
tain vacuoles and some of these are so large that the cell seems trans-
formed into a vesicle and the nucleus is not to be seen.

On looking over the sections with a low power, one notices here & there
emerged looking patches which have not taken on almost any stain. These
areas shade off imperceptibly into the surrounding tissue and are not
bordered by a line of deeply-staining cells. Under a high power
they are found to be composed more or less entirely of cells which
have become distended with colourless fluid until they are quite
globular and much larger (2-4 times) than normal. It looks
as if the vacuole had gone on enlarging until all the cell content,
including the nucleus had disappeared and a simple bladder resulted.

The exact localization of these areas is not always easy to determine.
in some cases they occupy a segment (a quarter or so) of the circle
formed by the lobules but often their position is less distinctly defined.
They seem usually free from fat pigment masses even although these
may be plentiful in the adjoining liver tissue. Some of these patches
appear shrivelled and small, as if in process of being absorbed and
replaced by a sort of cicatrice. It seems possible that some of the
islets of fibrous tissue, before alluded to, may arise in this way,
and if so this would account for the apparently intra-lobular sit-

---

*Many of not all of the vacuoles are merely the spherical spaces left from the oil having
been dissolved out of the fatty lobules. Most are not stained with some acid at all but
in a few near the capsule it brings out a black cement or radiating crystals.
uation of some of them.

It is very difficult to ascertain the cause of these appearances, but judging from their situations & surroundings one is inclined to think that they are in some way due to the pressure of the newly formed connective tissue. It is, however, possible that the state of stagnation of bile may have something to do with their causation. These areas, especially when the process is far advanced in them, are certainly strikingly similar in appearance to those figured by Beloussow, in his article "On the results of tying the ductus choledochus" in animals, which he regards as little patches of localised gangrene brought about by the bursting of overdistended bile-capillaries. He found, however, that it was only during the earlier days after the operation that this appearance was met with and that a week or two later they were replaced by fibrous cicatrices. Pick also in his paper on the same subject gives a somewhat similar figure of a necrotic area.

Although, as I have just said, these necrotic areas are extremely like in many ways the patches I have been describing, it hardly seems possible that they can be of the same nature and due to the same causes.

Thirdly, there is a morbid change which resembles that just described in some ways and differs from it in others.

Under the capsule—especially marked near the sharp edge of the liver—there is a homogeneous-looking zone which scarcely colours
Section of Liver. Low power (about 60 diam.)
(Hogwood. Balsam).<ref>
Showing layer of necrosis under capsule bordered
by layer with numerous brightly stained nuclei (p. 13.)
Liver cells and bile capillaries

(picrocyanin & green)

Showing distribution of inspissated bile in and between cells (p. 15)

Copies of wood-cuts from Hermann's Handbuch der Physiologie Bd I p. 226

Showing vesicles for the accumulation of bile in the liver cell (Kupffer)

(p. 15)
colours at all with stains. It stretches along parallel to the capsule without regard to the limits of the individual lobules and is bordered by a richly nucleated brightly staining line. There are very few fragments of bile-pigment seen in it.

On closer inspection, this turns out to be apparently an area of necrosis in which the cells are breaking down by atrophy and fatty change & are being absorbed. Its situation and distribution indicate pretty clearly that it must be due to the compression exercised by the thickened capsule and other new fibrous tissue.

In the part which is beginning to be affected we find the nuclei large and much increased in number and very deeply stained. There are also many vacuoles. These latter increase in number and size, the nuclei lose rapidly their power of absorbing colours & the cells become atrophied or fatty degenerated and break down. In the parts most affected the cellular structure is difficult to make out. In thin sections in which the cellular debris has been shaken out of this area, one sees that there is a small amount of intercellular fibrous tissue left.

Fourthly, one of the most noticeable features of the sections is the large number of little masses of unpigmented bile-pigment scattered about over them. These vary in colour from bright brown like resin to a dark green like bottle glass, the differences being due to the different methods of hardening the tissue.

These masses are found, as already noted, distending a large number of the small bile ducts, the largest of them being seen in those
in those ducts, which are among the fibrous tissue. The majority, however, of the small ones are found in the inner two-thirds of the lobules and the number of them in many places is very great. In this situation, we find on looking closer that the great majority of these little fragments are not, as one might perhaps have thought at first sight, in the cells, but lie between them, constituting in fact casts of the bile capillaries and many of these which are cut lengthwise have a branched configuration.

A few of the cells, however, do seem really to contain fragments of this material inside them. We find some, for example, which have a stained nucleus and tolerably healthy-looking protoplasm and in at one or two small round green masses (Plate 6). When one sees the shape, size and position of these one can't help thinking that these green spheres may possibly be casts of the intracellular bladders which are supposed to exist normally in the hepatic cells for the accumulation of their secretion and which have been described and figured by Pfeiffer and Kuffer (Figs 5 & 6) as having been found in the livers of rabbits after injections with prussian blue.

Where larger fragments occur, the cell in which they are seen usually more or less free from stain and without a visible nucleus. In many places one finds several different-sized masses lying together inside what looks like a cell wall, with only a little granular unstained matter about them, as if the damming back of the bile into the inside of the cell had caused dissolution of the ord
inary cell contents and destruction of them. This may probably account for the number of cells which seem to have disappeared from the inner zones of those lobules in the bile plugs are most numerous the absence of which gives those areas such a ragged look under a low power.

It is difficult to see why these fragments of bile should be localized so markedly in the inner zones of the lobules. Wyss in his observations on the histology of obstructive jaundice (in adults) found that when the obstruction had only existed for a short time the cells in the inner zone were diffusely stained with bile; but that when it was of long (3 years) duration bile plugs were met with in the periphery of the lobules.

In our sections there are only a few cells which present the diffuse granular pigment described & figured by Wyss and the cells affected are usually ones which form part of the wall of a bile capillary which is distended with bile.

Spleen. Sections of the spleen show the ordinary appearances of chronic hypertrophy of that organ.

Kidneys. Sections of one kidney were examined and showed nothing abnormal except a few very small bands of young fibrous tissue running inwards from the capsule — an extremely slight amount of interstitial nephritis.
2nd

Case of Obliteration of the Bile Ducts.

Giuseppe di Piacidide aged 2 months born first on 25th March 1889.

Complaints - Jaundice and a lump in the scalp; both since 10 days after birth.

Family History - The father, an elderly Italian organ-grinder was married before and had by his former wife, 3 healthy children. He has always had good health and denies ever having had any venereal disease.

The mother also seems healthy and has had no symptoms pointing to syphilis (except miscarriages). She also has been previously married and had 5 healthy children by her first husband.

Since her second marriage she has been pregnant three times. The first time she aborted about the end of the second month, the second time about the end of the fourth month and the third pregnancy ended in the birth of the patient at full term. The labour was easy, it lasted 2 hours only and no instruments were used. She had felt as well as usual during her pregnancy.

At birth, the child seemed quite healthy and was not at all jaundiced.

On the 9th day the mother noticed a lump on the top of the head, towards the right side; it increased in size for 7-9 days and then gradually diminished. The first motions passed were dark like those of all new-born children. Then there was a little yellow colour only in the stools till the jaundice began and after that they were quite white. The motions have, especially of late, been frequent, usually 3-5 in 24 hours.

The jaundice was first noticed on the 10th or 11th day after birth, it increased for 6 days and then has remained stationary. There have been no hemorrhages from the umbilicus or elsewhere. The child has been

* To the parents of the child, I do not speak English, I write to express my deep obligations to Dr. Ascoli of San Remo who spent a great deal of time and trouble in eliciting the facts of the history for me.
been on the breast and his appetite has been good. He has never had
smutty or any rash on the skin. He has had no other symptoms
except occasional vomiting & frequent screaming.

Present Condition: The patient is a small rather delicate looking baby
but not markedly emaciated. The skin, conjunctiva and other mucous
membranes are deeply jaundiced. On the scalp, in the usual situation
near the posterior superior angle of the right parietal bone, there is the
remains of a cephal hematoxd.

Abdomen not distended. Spleen enlarged reaching about 1½ inches
below the costal margin in the right nipple line. The spleen does not
extend below the edge of the ribs but can be felt distinctly when the
child is not crying. There is no ascites. - Extreme jaundice.

Heart and lungs normal. - Urine greenish yellow, the only specimen
which could be obtained was old, it was alkaline & showed no deposits
but masses of bacteria. There was no albumen. The feces were of a pale cream-color.

Further progress - 11th April. Jaundice deeper in tint. The child screams con-
tinually when not at the breast.

(barium) 99.6.

15th April. On the outer side of the extensor surface of the right forearm there is a
hard body felt under the skin, the size of half a bean. It has a bluish tinge,
and the skin is not adherent to it. There is a similar subcutaneous nodule
a little to the right of the spine of the 6th dorsal vertebra.

16th April
16th April. During the last few days there has been occasional hemorrhage from the nose.

18th April. There are several new subcutaneous hemorrhages, namely (1) one on the left forearm corresponding in position and character with that on the right forearm but half its size. (2) 5 small nodules of similar character in the left interscapular region. (3) Over the lower third of the sternum there is a large subcutaneous hemorrhage forming a blue mark but no swelling.


Owing to the unfortunate impossibility of obtaining a post-mortem, this case loses almost all its value. I have, however, thought it worth while to record its clinical particulars as they are very typical and leave no doubt that the case was one of the condition we are dealing with. I shall not, however, have occasion to mention it again, as it is better to restrict oneself entirely to completed cases.
Other Published Cases.

In the accompanying large table (Table 1) I have put down the details of all the cases which I have been able to find reported, in which the diagnosis of malformation or obliteration of the biliary passages was confirmed by a post-mortem examination.

To complete the list, I should mention that Dr. West refers to two other cases which he has seen, in addition to those he gives details of, that Prof. Virchow mentions having seen one case, and that a case has been published by Michel and one by Lobstein, of which I have not been able to get any particulars. Trouseau also refers to some cases mentioned by Porchat in his thesis (1859) and I have not been able to find out whether or not these are among those I have tabulated. Lastly, Witgel has published an account of a child in which there was congenital obliteration of the bile ducts in addition to a large number of other developmental abnormalities.

We shall now proceed to a systematic analysis of the facts recorded on this Table and we shall find that this brings out a great many points of interest.
Analysis of Table I.

1. Rarity of Occurrence.

Whatever the causes are which combine to produce this lesion, it is evident that they are only very rarely found in combination, for all the reported cases, including my own, only amount to fifty-four or thereabouts. We may also observe that, while Dr. West has seen four post-mortem cases of such cases and Prof. Henoch two, many men of great experience such as Virchow, Murchison, Harley and Legg have only met with one case each, and most writers on pathology seem never to have seen any at all.

It is perhaps worth noting that of the 47 tabulated cases, 21 have been described by British authors, 17 by Americans, 18 by Germans, and one by a Frenchman.

2. Health of Parents.

The main point of special interest with regard to the parents' health is the question whether they were suffering from syphilis. We have a good deal of evidence on this matter, but it has, of course, to be received with considerable reservations--so far as it is negative.

In 28 out of the 47 cases, nothing is said about the parents' health that can be held to bear on this particular. In 13 it is said that they were "healthy," and in at least
of these (Nos. 2, 16, 45 and my own case) it is expressly stated that there was no history of syphilis discoverable (Mr. Morgan seems to be quite certain on this point with regard to his case). In 4 instances (14, 15, 25, 26, 46) one parent was certainly syphilitic (or had been) and probably also in a 5th (18).

Therefore out of the 90 parents of these 47 children we have evidence of syphilis in 5 only—that is in 5 1/2%.

It certainly deserves attention that in several cases one or both parents seem to have suffered severely from derangements of the liver or of digestion. It scarcely seems likely that this fact, however, can have any etiological importance, although this has been suggested.

King (loc. cit. 14) questions whether there may not possibly be some connection between liver disease in the mother and a congenital tendency to perihepatitis of the lower surface of the liver in the child. Glaister (loc. cit. 9, p. 331) mentions that the father of his case was habitually "bilious" and that the mother also was "bilious" during pregnancy although not at other times. In my case the father was a chronic dyspeptic and at the time of the child's conception was probably suffering from cancer of the stomach from which he ultimately died. The mother, all through her pregnancy had an insufficient supply of proper food and
and lived largely on fruit (probably none of the freshest) given her by a friendly fruit-seller.

3. Other Children.

About the other children we notice three things:

(i) In several instances there had been a large family of healthy children before patient was born.

(ii) In no case is it mentioned that any of the other children had showed signs of syphilis.

(iii) Although in several cases the patient was the only child afflicted in a large family, there is evidently a very remarkable tendency for the disease to occur in more than one child of the same parents. For example, among our 47 cases we find that there twice our occur 2 cases in the same family (14 & 15 and 34 & 35) - In another instance (22) a pair of twins were jaundiced and one of them recovered. Then, in Anderson's case 38, another child of the same parent suffered, we are told, from both jaundice and hemorrhage, but recovered; and again out of 7 children in the same family as Glaister's case 9, 4 had died jaundiced and 2 were less severely jaundiced and recovered. In the two cases before alluded to, which Dr. West mentions without giving particulars, there was also a marked family history of infantile jaundice.
It is interesting to note that in the cases of fatal infantile jaundice which have been found post mortem to be accompanied by pervious bile ducts, occurrence of the disease in several members of a family is an equally characteristic phenomenon. (see Appendix II, Table 3).

4. **Character of the Labour.**

This is important because injury or exposure at the time of birth might be the exciting cause of peritonitis and therefore of possible pathological importance in connection with our subject. We find, however, that there is nothing in the statistics of the table to suggest that the character of the labour can be thought of importance.

Out of 10 cases in which this point is mentioned:

In 5, the labour was easy & normal; in 3 it was tedious, once it was a "dry birth" and once there was a breech presentation. In none of these cases was there anything in the state of the appearance of the jaundice to indicate any probability of injury at the time of birth, being the cause of the disease.

5. **State of Child at Birth. & Prematurity**

The state of the infant at birth confirms the above impression, for in none of the cases (with the exception of two...
of two syphilitic ones \(25 \times 26\) does anything morbid, except the yellow colour of the skin, seem to have been noticed. Prematurity is mentioned 5 times; in none of these cases was jaundice present at birth.

6. Sex

It has often been pointed out that this condition affects more boys than girls; and our table bears this out. Out of 33 cases in which the sex is mentioned 20 were boys and 13 girls.

7. Symptoms (a) Onset of Jaundice

The exact period of the onset of the jaundice seems to have varied very much in different cases. According to our table we find that this fact is not mentioned in 10 cases.

That jaundice was present at birth in 9 "

it appeared on the day of birth in 5 "

second day 8 "

third day 2 "

within a week of birth 4 "

between 1 and 2 weeks after birth 6 "

after a fortnight or more 3 "

The exact period of onset is, of course, a point with regard to which
to which there are several sources of error. For example, the presence of ordinary icterus neonatorum might obscure the exact date of appearance of the more severe form of discoloration; or want of light or attention might easily lead to its presence being overlooked for several days. Still, taking such difficulties fully into account, there can be no doubt after studying the reported cases, that in a considerable proportion of them there was no jaundice present until several days after birth. Also it is plainly stated in many cases that the jaundice was very faint at birth (or when first seen) and rapidly became more intense which change suggests that the condition was not of longstanding.

These two facts suggest an inference, from which it is not easy to escape, that something or other occurring at or about the time of birth has some relation to the onset of the colouration in many cases.

What period of time elapses between the obstruction of the bile duct in an infant and the appearance of jaundice we do not know. Wickham Legg is of opinion that the time for an adult is 24 - 48 hours. Murchison (loc. cit. p. 380) says that in patients with biliary colic the yellow discoloration is observed 12 - 24 hours after the subjective symptoms have occurred. Little help can be got from observing
observing the time which ligation of the common duct takes to produce jaundice in the lower animals, because this varies widely, not only with the different genus operated on but with the individual animal and also apparently with the individual operator. In dogs it takes from a few hours to three days, in cats from 10 to 20 days, and in guinea-pigs it does not come on at all.

We should naturally think it possible that the action of some external agency such as violence or cold at the time of birth might be to blame for the appearance or aggravation of the jaundice soon after it, but this as already seen (p 24.) finds no support from facts.

Although we cannot fully explain this relation between the jaundice and birth, we may be pretty sure that the enormous changes which take place at that time in the circulation of the liver have something to do with it.

Before passing on to the next column, I may be allowed to propose one question to which I cannot find any answer at all satisfactory:

How is it that a child who for many months of intrauterine life has had no bile reaching its intestines (as indicated by entire absence of colour from the faces from the first) can be born & remain for many days unjaundiced (see Wilks' 10 and Freund's 22 cases)?
Can it possibly be that the liver cells which had long ceased to secrete bile, owing to the permanent blocking of their ducts are so stimulated by the sudden change in their circulation that their natural function begins again?

(6) Character and Progress of Jaundice

Even when it is either absent or only slightly marked at birth, the jaundice always becomes extremely deep sooner or later, and often is of a greenish tinge. It is sometimes noticed to vary a little in severity from day to day but on the whole it gets steadily worse till it is as deep as it can well be. Towards the end it has sometimes been observed to become a little paler.

It is interesting to observe that in several cases (18, 21, 29 & 30) where there is said to have been a certain amount of communication by at least one duct between the liver and the intestine (see Table 2) the jaundice was as marked as in the other cases where the common duct was entirely obliterated. These cases in fact form a sort of link between those of complete obliteration and those other cases of jaundice with similar clinical histories in which previous ducts are found at the post-

*Jennings says that in cases of long continued obstructive jaundice where the colour passes off, this is due to the fact that the liver no longer secretes bile.
post-mortem (see Appendix II)

(c) Urine.

In Dr. Nuneley's case it is stated that there was no bile pigment in the urine. In all the other cases where the urine is described there was apparently much bile pigment in it. No other abnormality is noticed.

(d) Meconium.

Meconium used to be regarded as merely a mixture of bile, intestinal mucus and intestinal epithelial cells; but it has long ago been proved by Förster, Zweifel, and others that, though it does contain these substances, its main bulk is made up of matters derived from the vernix caseosa which the fetus has swallowed in utero, along with amniotic fluid. It begins to be formed early in intrauterine life and in a 3 months old fetus, according to Zweifel, both bile pigment and bile acids may be detected in it. It gradually accumulates in the intestine until at birth it amounts to from 60 to 90 grammes. In a normal still-born baby, one finds the large intestine distended in its entire extent with dark green meconium of almost uniform colour but towards the ileo-cecal valve the tint is lighter. The lower few inches of the ileum contain similar matter much paler in colour, while
While the contents of the duodenum, jejunum and upper ileum are of a honey-yellow colour and much less dense in consistence. Naturally, therefore, the meconium in the lowest part of the bowel, which is that first formed, is darkest in colour. As far as I know, it is never met with without its usual dark green colour (even light green) except in cases in which there is severe organic disease of the liver or bile ducts.

In 10 of the 47 cases it is either expressly stated or else implied that ordinary meconium or something similar in appearance was passed during the first few days.

Some of the infants who passed ordinary meconium were born jaundiced, but in others the discolouration was not noticed for a week or more after birth.

With the exception of Köstlin's cases,29 30 (which indeed differ in several points from the others) it seems as if in none of the cases was the dark meconium followed by even one motion of normal yellow faeces; the motions at once assumed their cream-colour or clay-colour, even if the jaundice did not come on for some days later. This looks as if the disease were always in a certain state of advancement at birth.

What is the significance of the occurrence of normal meconium in some of the cases and of perfectly colourless motions?
motions from the first in others? Where normal meconium
is passed, it proves that the way from the liver-cells to the
bowel must have been open for some time after meconium
began to be made; and it excludes the possibility that in
these cases the malformation of the ducts could be due
to defective development.

When the discharge is colourless from the very beginning,
this seems to point to a very early occurrence of the obstruc-
tion indeed. There are reasons against such an early origin
for the lesion; but the entire absence of colour can scarcely
be otherwise satisfactorily explained.

For example, it might be suggested as quite possible that
the bile passed into the intestine in the ordinary way might
become decolourised in time by the active absorptions of the
fetal intestine; just as bile shut up in the gall-bladder
in adult life (owing to the obstruction of a stone) gradually
becomes changed into a colourless watery fluid. To this,
however, it may be answered that there is no reason why
this should occur in these cases; when, as we have seen,
it never occurs when the liver and ducts are normal.
It might also possibly be said that the bile secreted by
the liver might be colourless, because we know that this
phenomenon is observed under certain conditions; for
example in syphilitic interstitial hepatitis the content
of the gall-bladder sometimes look like pure water & the
same thing probably occurs temporarily in the liver
quite normal * To this also it may be conclusively an-
swered that if it occurred in these cases it would also
be met with in children with normal livers; which it is
not.

e) Motions.

In about 20 of the cases, the motions seem to have been
white, greyish or cream coloured from the beginning, and in
most of the others they became so immediately after the mecenc-
ium had been passed. In a few, however, we are told that
the motions sometimes contained coloured matter at the la
stage of the case. These may be divided into two distinct
classes—firstly, those in which there was not complete ob-
struction to the flow of bile from the liver to the duodenum
such as Nos. 18, 21, 29, 30 (see Table 2...) in which a vary-
ing amount of yellow, green or brown matter was observed; and
secondly, a few in which green matter was passed along
with the white or grey faces, although the obstruction to the
bile-duct was quite complete, for example, M. Morgan's
case.

In fever (see Paton & Balfour 63) and probably also after
epileptic seizures sometimes as a dose of opium in children
(see Speech at Medico-Chirurgical Society by R. Hendley 64)
In which the motions contained greenish brown matter after a dose of gray powder; and two other cases in which green lumps were seen and in which no mention is made of their following the administration of mercury.

The former class requires, of course, no discussion, but the causation of the green matter, in the feces (of non-hepatic origin) in the latter class is a matter of considerable interest.

The subject seems to be one which has not been thoroughly worked out, and I have nothing new to offer in explanation of the facts, but it may be worth while to mention briefly a few facts and statements from different sources bearing on the subject of green coloration of the feces not due to bile; and these will suggest various possible explanations of the problem as it presented itself in our cases.

Firstly, then, although no bile enters the intestine in the ordinary way, it is possible, as was suggested by Osborne in a paper on the examination of the feces long ago, that a certain amount of green or yellow color may be imparted to the feces by their being mixed with intestinal mucus which is jaundiced, just as the blood and urine are. This however, even if possible, will explain very little indeed.

Secondly, it is now known that the well-known green stools produced by the administration of mercury do not depend, as used
as used to be held, on a stimulation of the liver and an in-
creased pouring out of bile - Radviezewski produced green
stools by giving calomel to a dog with a biliary fistula.
D. Haldimann states that the green stools caused in this
way owe their colour to the formation in the bowel of a sul-
phide of the suboxide of mercury. In Mr. Morgan's case,
the green matter appeared after a dose of grey powder, so
that some such explanation as this might well account
for it; and as the other cases very probably got mercury
also, it would explain them too.

Thirdly. The colour may be due entirely to microorganisms.
In this connection I may refer, to begin with, to D. Hermann's
recent experiments. He operated on a number of dogs
by separating a loop of intestine entirely from the rest of the
gut, sowing up the ends of it, and then establishing the lumina
of the rest of the bowel by sewing the cut surfaces together.
In those dogs which died after 6 days, the separated
portion of bowel contained a brownish gelatinous fluid;
while those which survived the operation several weeks
had the loop of gut full of a firm greenish grey mass,
deceptively like feces. This greenish material contained
innumerable microorganisms, to the action of which its
colour was, presumably, due. When tested for bile pig-
ments it gave no reaction.
Again, in many cases of infantile diarrhoea where the motions have an intensely green colour, it has been shown by Desage and others that this is due to their being largely composed of masses of a peculiar chromogenic bacillus and has nothing to do with bile.

Lastly, an interesting case of my own will be found in the appendix (III p. 29) in which bright green matter was passed per rectum by an infant; the colour of which must almost certainly be held to be due to the microorganisms it contained, as the nature of the congenital malformation of the bowel which existed (when taken along with the symptoms) precluded the idea of its being of bilious origin.

(f) **Vomiting.**

In 9 of the cases vomiting is mentioned as having occurred; but considering the frequency of this symptom in children, this fact does not seem to be of any special importance.

(g) **Hemorrhages.**

The occurrence of spontaneous hemorrhages is one of the most characteristic clinical features in these cases. In 20 out of the 47, that is in fully half of the cases, which
which lived more than a few days, the fact of haemorrhage having occurred from some part of the body is noted, and in all probability it occurred in some of the others although not mentioned - as the records of many of them are so meagre.

The situations of the haemorrhages mentioned are as follows:

<table>
<thead>
<tr>
<th>Situation</th>
<th>Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subcutaneous</td>
<td>6</td>
</tr>
<tr>
<td>Subconjunctival</td>
<td>1</td>
</tr>
<tr>
<td>Umbilical</td>
<td>6</td>
</tr>
<tr>
<td>From nose</td>
<td>2</td>
</tr>
<tr>
<td>Vomited</td>
<td>4</td>
</tr>
<tr>
<td>From bowel</td>
<td>7</td>
</tr>
<tr>
<td>From mouth</td>
<td>1</td>
</tr>
<tr>
<td>From lung</td>
<td>1</td>
</tr>
<tr>
<td>Into gall bladder</td>
<td>1</td>
</tr>
<tr>
<td>From leech bite (excessive)</td>
<td>1</td>
</tr>
</tbody>
</table>

A similar tendency to spontaneous haemorrhage is well known to exist in all cases of continued obstruction of the common duct, sooner or later, and is almost equally common in a large number of other morbid conditions accompanied by jaundice - such as, acute yellow atrophy, yellow fever, phosphorus poisoning, etc. etc.

The situations above mentioned are pretty much the same as those from which bleeding usually occurs in those other
other forms of jaundice — with the one exception of the umbilicus, as one might expect from the age of our patients.

The occurrence of hemorrhages in any case of infantile jaundice is always of very serious prognostic significance, but they are not always followed by a fatal issue. One such case which recovered is mentioned by Anderson and another is reported by Heinrich (loc. cit. p. 27). Granddier in his very comprehensive article on "Spontaneous Umbilical Hemorrhage in New Born Children" mentions 35 cases of hemorrhage from the navel in which the children were jaundiced and also had ecchymoses, and 3 of them recovered. As indicating the close connection between jaundice and hemorrhage, one may mention that fully two-fifths of all the cases of spontaneous umbilical hemorrhage which Granddier has collected, occurred in jaundiced infants.

The cause of the hemorrhages is one which has never been satisfactorily explained, although many suggestions as to their mode of production have been made. The hemorrhage is commonly seen in cases of hot, miliary liver is easily accounted for by local interference with the circulation of the gastric mucous membrane; but in our cases, as in the various jaundiced conditions above alluded to, we have subcutaneous and other bleedings which cannot be so explained. We want
a theory which will account equally well for all the different haemorrhages in all the different morbid conditions in which they are found.

The following are some of the suggestions that have been made by different authorities.

1) "Impoveryishment of the blood" is given by Dr. Budd (loc. cit. (69) p. 463) as the probable cause. Murchison (loc. cit. (55), p. 369) also says that it is due to the blood becoming "impoveryished by a diminution in the proportion of the red corpuscles and fibrin" but he observes that it is particularly observed in conjunction with cerebral symptoms and other symptoms of blood poisoning. Freieich (loc. cit. (54) Bd. 1 p. 242) thinks it is due to some change in the blood and its adhesion to the walls of the vessels.

"Impoveryishment" means, I suppose, a simple deprivation or lessening of one or more of the normal constituents of the blood. The very sudden appearance of the haemorrhagic tendency often observed would rather suggest the adding of something to the blood than the taking away of anything out of it, which one would have thought would more likely have been a gradual process.

2) The action of bile acids in the blood on the corpuscles is suggested by Leyden (loc. cit. (70) p. 100). Legg objects to this.
to this that if the bile-acids in the blood were to blame we
should haematuria such as, he says, always occurs when
bile-acids are injected into the circulation: and this is
almost an unknown symptom in jaundice. This objection
does not appear very forcible, however, when we remem-
ber that the experiments referred to were in the lower an-
imals and not in man, in whom the action may be dif-
f erent. Mackay 77 found haemoglobin always in the ur-
ine of rabbits when the ductus choledochus had been
ligatured; and exactly the same condition after the
intravenous injection of bile or bile-acids.

A much more serious objection to this theory con-
sists in the fact that it seems to be only near the beg-
ning of an attack of obstructive jaundice (in animal
at least) that the blood contains any amount of bile-
acids. Mackay found that in the blood of rabbits,
whose common duct had been tied for more than six
days, little or no bile-acid could be detected; and al-
though haemorrhage is a very early symptom in
obstructive jaundice, it is also a very late one.

3) A diseased state of the blood-vessels is said by Wickham
Legg (loc. cit. B. p. 315) to precede and cause the haemorrhages and
he considers this view to be that most in accordance with
the present state of knowledge. He says that Leyden has
shown
shown that the bile acids produce a parenchymatous degeneration of the glands and muscles and that the blood vessels are probably affected in the same way. It is very difficult to see how this theory can be thought to explain the clinical facts at all satisfactorily in the case of jaundice, although a similar one may perhaps account for the tendency to hemorrhage met with in cases of enlarged spleen and some other chronic conditions. The sudden onset of the hemorrhagic tendency almost simultaneously with discolouration of the surface of the body, which occurs in so many of the cases, seems alone almost sufficient to disprove the idea that there is an antecedent organic change in the walls of the vessels. It is a well known fact that the hemorrhages occurring in obstructive jaundice (especially in children) are a very early symptom, and in several of our cases (e.g. 35, 36, 43) the bleeding set in and ended fatally within two or three days of the first appearance of the jaundice. In acute yellow atrophy and in yellow fever hemorrhage is a very early symptom also.

4). In reading the results of recent experimental pathologic research another theory comes naturally to one's mind which certainly, as far as it goes, seems to be much more satisfactory than any of the above. It has probably been suggested before by somebody although I have never happened to come across it anywhere. It is that the hemo-
pathologic tendency is caused by the presence in the blood, not of bile acids, but of substances or some similar organic poisons which formed in the process of ordinary digestion which the diseased liver is not able to render innocuous as it would do if it were in a state of health.

One of the recognized functions of the liver in the animal economy is to render innocuous in some way various sorts of poisons in the blood (see Lander Bumeton 75). Among the poisons which it acts on thus are the organic ones which are formed in the alimentary canal in the process of digestion.

Now, it has been found by Roger 78 that this important function of the organ is closely connected with the amount of glycogen it contains and varies accordingly; so that when the liver contains no glycogen, a very much smaller dose of these organic poisons is required to produce a given result than would be necessary if the organ were healthy in this respect. This has been tested in the case of animals with artificial cirrhosis of the liver, fatty degeneration, etc.

It has been demonstrated by Legg (loc. cit.(55) p.178), and confirmed by von Wittich, that ligation of the bile ducts produces disappearance of glycogen from the liver — the retention of bile seeming to interfere with the proper function of glycogen formation of the liver cells.

We are, I think, justified in assuming from the above fact that
that in the disease we are considering, there is in all probability a diminution of glycogen in the liver, owing to the forced retention of bile in it, and that the organ may very likely, on this account, be letting past its portal some poisonous product of ordinary digestion which under its usual conditions it would have summarily disposed of. The exact method in which a process of blood poisoning of this sort would act in causing hemorrhages we do not, of course, know, but it is a well-known fact that many forms of blood-poisoning are accompanied by a tendency to spontaneous bleeding. (For references on this point, see Runge loc. cit. (80) p. 117).

* * *

(k) Convulsions.

In other serious diseases accompanied by jaundice, such as yellow fever, acute yellow atrophy and phosphorus poisoning, convulsions are a common symptom. In chronic forms of liver-disease they often occur towards the end. In children, who are so subject to convulsive attacks, we should expect them to be met with often in any disease of this kind, and on looking at the table we find that fits are often mentioned—namely, 10 true out of 47 cases. It is interesting to note that convulsions often occur in animals after ligation of the ductus.
(i) **Emaciation.**

Emaciation always occurs if the child lives long enough, but it is not usually an early symptom. This may be partially explained by the fact that the exclusion of bile from the digestive process interferes to a certain extent (though not very much) with the assimilation of the fatty constituents of the food which are so important to the infant economy. Probably, however, the emaciation is mainly due to the secondary organic changes in the liver-tissue, interfering increasingly with the other more important functions of the organ and with the part it should play in the general metabolism of the body.

(ii) **Duration of life.**

In looking over the table we find the following details:—only one child was still-born (and it was premature); of the others, 11 lived less than a week.

- 7 more than a week but less than a month.
- 13 from one to four months.
- 15 for 4 months and upwards (one into 6th month).

Therefore, if we exclude those who died of umbilical hemorrhage, we may say that the very large majority lived
lived more than a month. These facts seem to indicate two things:—firstly, that the lesion is one which has not of itself a rapid deleterious influence on the general health; and, secondly, that it is not one which (with the exception of the hemorrhages) is apt to be accompanied by serious complications.

In a recent paper "On the composition, flow and physiological action of the bile in man" 63 Dr. Paton and Bal. four give it as their opinion that the bile is to be regarded rather as an excretion than as a secretion playing any essential part in digestion; and they also fuird that "even with bile excluded from the intestine, no less than about 70% of the fats of the food are still used." The fact that infants, with complete obliteration of the bile ducts, live so long as they do, seems certainly to corroborate to some extent the above opinion.

(4). Other morbid conditions present

Certain signs of congenital syphilis were observed either during life, or post mortem, or both, in 4 cases (13, 25, 26, 46), and in one other case (8) the occurrence of smuffles is noted.

As already said there are no serious complications which are usually met with, excepting the various spontaneous hemorrhages. The immediate cause of death in the
in the different cases seems to have been very various. Apparently the disease causes gradual weakening and any chance intercurrent affection carries the child off. There does not seem to be a predilection to any special disease.


(a) Peritoneum

A large quantity of ascitic fluid was found in one case, a smaller amount in another, and a little effusion in several other cases (viz: 27, 26, 12, 25 (postally post-mortem)). The presence of adhesions or localized thickenings is noted five times (5, 14, 18, 26 & 27).

In two cases (37 & 44) there was recent peritonitis, evidently secondary.

(b) Liver

(1) The size is generally, but not always, more or less increased. It is not mentioned in 18 of the cases.

- said not to be enlarged in 7
- to be small in 1
- enlarged in 11
- much enlarged in 9
- unequally enlarged in 1
We find that, as we should expect, enlargement is more frequently noted in cases which lived a long time.

(2) The colour is most frequently dark green - almost always so in those cases which lived long.

It is not mentioned in 24 of the cases.

It is described as reddish brown in 2

... "Jaundiced" in 4

... "dark green" 13

... "dark purple" 1

... "light green" 2

(3) The consistence is not alluded to in 27 of the cases out of the 20 cases in which it is mentioned; it is said
to have been normal in 1

... "soft in 5

... "tough in 14

Possibly the softness of some may have been due to decomposition.

(4) The surface of the organ is only mentioned 10 times; in 2 of these it was smooth and in 8 finely granular.

(5) The blood-vessels of the liver are described 20 times. In 16 they were normal and in 4 there was dilatation of the portal vein. It is significant that all these 4 cases were probably syphilitic (13, 14, 26 & 27).
(c) Gall bladder and ducts.

As these are the seat of the primary lesion in our cases we shall examine their condition somewhat thoroughly.

Firstly, then, we shall consider the precise lesion described in the various cases. This is summarized, so to speak, in the accompanying diagram (Table 2.). In it I have arranged in a sort of order of sequence diagrammatic representations of the exact conditions of the gall bladder & cystic, hepatic and common ducts and the duodenum described by the writers as having occurred in a majority of the published cases. The others are not represented either because they seem to have been exactly like one of those figured; or, in some cases, because so few particulars were given that it was not possible to say whether they differed or not. Where a part of the apparatus (e.g. Gall bladder, Cystic duct etc.) is not mentioned in the description, I have represented it as normal. Of course, in many of the descriptions much that one would have liked to know is left out and what is given is not always very intelligible, but I think that the main points will be found to be fairly represented, as far as can be ascertained. Their accuracy may be tested by comparing them with the descriptions by the authors which are given—mainly in their...
own words on pages 71-76 (Appendix I).

In studying this diagram we may look first at the
Situation of the Obliteration, and we find that this
varies almost indefinitely.

Among our 47 cases, there are examples of almost
evry conceivable variety. We may divide these
roughly into 4 groups:
1) Those in which there is no passage whatever lead-
ing from the liver to the duodenum although the gall-
bladder and cystic duct remain more or less normal.
2) Those in which there is at least one permeable canal
leading from the liver into the gut but no exit from
the gallbladder or even nothing to represent that organ.
3) Those in which both cystic and hepatic ducts are
obliterated.
4) Those in which the obliteration has occurred at a var-
able point below the junction of the cystic and hepatic
ducts.

Now, this great diversity in the precise situation
of the lesion is a point of considerable interest and
of itself throws no small light on certain aspects of
the etiology.

Were the malformation due to an arrest of, or an inter-
ference with, the developmental processes, as some assert,
we should
we should certainly expect that in most, if not in all, of the cases the obliteration would be found in or near the same situation.

Again, were the local cause of the blocking an inflammatory process set up inside the lumen of the tube by a concretion or other local source of irritation, as other think, we should in the same way expect a tendency for the obliteration to be situated about the same place. We get therefore at the outset an impression that the immediate cause is one which as no seat of election and therefore most probably either acts on the tubes from outside, or is a diffuse process which affects their walls for a considerable extent before the actual obstruction occurs.

(c) We may next look at the diagram again to see what can be learned from it as to the Other Secondary Changes in the external excretory apparatus. Again, one is struck with the great variety met with; although the significance of the differences are difficult to determine, and indeed the anatomical descriptions are scarcely sufficiently full to allow us to attach much weight to the figures in this connection.

I shall therefore only refer to one or two points of interest.
interest.

1) When the cases are grouped together according to whether jaundice was said to be present at birth, or only came on later, no peculiarity is found in the nature of the malformation.

2) We find, as might be expected that, the most severe malformations are almost all in cases which had lived several months. The inflammatory action evidently goes on after the obliteration and affects the rest of the ducts and the gall bladder—at least in many cases. In Dr. West's case this was so to a marked extent (see 1). The inflammation set up by an impacted stone spreads in a similar way (see Courvoisier 62).

3) Dilatation of the canal above the point of obliteration is by no means invariably present (e.g. 2, 17) although it may occur to an enormous extent (23). When present, it may either take the form of a mere dilatation of the ordinary passages (11, 38) or there may be a more or less abrupt and saccular enlargement of the end of the tube (33, 6, 23).

4) An obliterated duct or portion of a duct may remain as a fibrous cord or may entirely disappear.

5) The obliteration of the upper end of a duct does not necessarily cause the same of the lower end (13, 16) even after
after the lapse of 4½ months (16).

D) The contents of the gall bladder vary very much—from thick syrupy bile to clear watery fluid. But we notice that they are almost always described as colourless if the child lived more than a month. In all the cases where it is definitely stated that the motions were colourless from the first (with one exception 11) there was no bile in the gall-bladder.

(a) Microscopic Appearances of Liver.

The facts given under this head are very few—37 of the cases were not examined microscopically. In one case examined (Nunneley's) cirrhosis is not mentioned in the 9 remaining cases biliary cirrhosis was present.

It seems probable that cirrhosis always occurs if the child lives long enough. The only apparent exception is Nunneley's case 12 in which the description is not full and no mention of cirrhosis is made but it is not said that it was not present.

Several of the writers describe the masses of desiccated bile in the small bile ducts and some mention having fragments of the same substance inside the cells.

The microscopic anatomy of the disease having been so fully gone into before (p. 9) it is unnecessary to say more about it here.

(c) Spleen.
Spleen

In 10 of the cases the spleen is mentioned. In one of these it was "not enlarged" in all the others it was large - generally very large.

The spleen usually, though not invariably, enlarges in animals after ligature of the common duct (see Mackay 77). The cause of this does not seem to be settled, probably it is to be explained in the same way as the occurrence of the haemorrhages (see p. 40.).

In 6 of the cases with enlarged spleen haemorrhages are noted.

Other morbid conditions

These are very various, and do not appear to have any direct connection with the liver affection. In many of the cases pulmonary complications seem to have been the cause of death.
Pathology of the Lesion of the Ducts.

The lesion has been ascribed to three different causes, either acting separately, or in combination; these are:

1. An arrest or defect of development (Vinium prima forma tionis).

2. An inflammatory or other lesion of the ducts themselves.

3. Peritonitis and its results, acting on the ducts from outside, and either compressing them or being a source of inflammatory action which spreads afterwards to their wall.

1. Arrest or defect of development.

When cases of the condition we are considering are referred to in general works on medicine or pathology, they are usually put down to this origin, and several of the writers who have reported cases have spoken of them as due to an interference with or an arrest of, the normal development of the parts.

There can be no doubt that various malformations of the bile ducts and liver which are certainly of this nature do occur. For example, congenital absence of the gall bladder has been frequently described (for references to cases see Courvoisier loc. cit. p. 139 and Meckel (93) and some of the cases were probably due to arrest...
to arrest of development (although many have been of inflammatory origin). Forster, in his work on "Human Malformations," 81 says that not only the gall bladder, but also the common duct or one of the hepatic ducts may be wanting. Wenzel Gruber 91 has published a case in which a forked cystic duct was found; and Konigsky 92 describes another in which the common duct had an unusually long and curved course and opened into the middle of the horizontal portion of the duodenum, its lumen also being narrowed. Lastly, O. Witzel 93 has published notes of an infant which was born with a large number of congenital abnormalities and in whom in addition to hemicephalus, etus viscerum inversus, six fingers on each hand etc., there was a cystic condition of the liver and complete impermeability of both the cystic and common ducts.

Of the writers whose description I have tabulated, Heschl 19, Harley 11, and Freund 22 are all more or less inclined to regard their cases as due to a fault of development; and Lotze 18 seems to think it probable that his case may have been of this nature primarily. I may also refer to Cheyne's case of narrowing of the ducts 57 (See Appendix II p. 78.) which he regards as due to
due to developmental defect.

When we look over our table of details of cases, there seems little in support of this origin for the mal-
formation in most of them, and several consider-
ations against it. These are as follows:—

1) An arrest of development which would cause ob-
liberation of the bile ducts would require to take
place at a very early period of intra-uterine life.
This fact seems to exclude such an origin in all
those cases in which coloured meconium was passed.
Again, if the obliteration was caused in this way at
such an early period would there not probably be
found some secondary cystic change in the liver
due to the retention of its secretion such as is de-
scribed in Witzel's undoubtedly congenital case
above mentioned. Such a change is only mention-
ed in one of our cases (Lomer's 25).

2) As already pointed out, the great variety in
the situation at which the obliteration occurs seems
to be strongly against this etiology (p. 48).

3) In Heeb's case 19, we are told, there was an absence
of bile ducts in the liver-tissue; and in Prof. Simpson's
44 there was an absence of the Spigelian and quadrate
lobes, but in none of all the other cases is there any
mention
mention of any condition of the liver which could be put down to developmental defect.

4) In none of the cases is mention made of any malformation of any organ except the liver.

There is, of course, a possibility that the developmental defect might consist in a mere narrowing of the lumen and that this might be followed by inflammatory complications, as apparently happened in Konskikh's case, but there does not seem to be much in support of this idea.

2. **Inflammatory or other affections of the ducts** themselves have been suggested as the cause by several writers. Unfortunately, microscopic examination of them does not seem to have been made in any of the cases. A few cases have been published in which a real or imaginary cause of obstruction of the bile-ducts in an infant was found. Having few facts to guide us, we must argue as well as possible from analogous conditions.

The lesions of the ducts which have been described as possible causes of obstruction are as follows:

a) Cataract of the mucous membrane, followed by erosion and adhesion of the bared surfaces, is the morbid condition
but condition which is naturally first thought of. It is a recognised cause of blocking and even of entire obliteration of the ducts in adults (Cornil, Ramée loc.cit.96) p.409. In them the commonest cause of it is, of course, the irritation of a stone, but it is also set up by syphilis, and it is said that other impurities in the blood may give rise to it. The position in which obliteration from this cause usually occurs is the pars intestinalis of the common duct (von Schüppel loc. cit.72) p.583. If severe catarrh of the ducts occurred in intrauterine life, we should not be surprised to find that it led to obliteration of the lumen more readily and more rapidly than in adults. (b) Gall-stones must be extremely rare in infants but one case has been put on record by Lieuteaud 82; and another case in which calcaeous concretions were met with in the liver of a young child has been described by Wranka. 83

(c) A plug of inopacitated bile was found in the bile-duct in one carefully described case (Campbell 32) and was supposed to be the cause of the obstruction which had ended in death.

(d) One case of fatal obstructive jaundice in a young infant has been described as due to spasmotic stricture of the duct but the writer gives no facts whatever in support
in support of his hypothesis and it is difficult to see how it can be entertained.

(c) **Gummatus inflammation of the walls of the ducts** has been described by Bach in one case in which there was condensation confined to the immediate neighbourhood of this region and syphilitic cirrhosis of the liver.

In considering these morbid conditions as to their power of causing obliteration, (b), (c) and (d) may be put aside on account of their extreme rarity or dubioseness. **Gummatus inflammation** may also be dismissed in a few words, because although it seems a likely enough lesion to occur under favourable conditions, yet we could not expect to meet with it often entirely apart from any other signs of syphilis elsewhere and without there being gummatus or intestinal syphilitic hepatitis at the same time.

A **catarrhal condition of the ducts** seems a quite possible cause of the malformations met with. We do not, of course, know of anything likely to set up catarrh at that period of life (except syphilis). We are, however, familiar with the fact that ulcers for which no cause is known do occur in very young infants in the stomach...
ach and duodenum (Henshöch loc.cit. (1) p.66). Indeed, the
icatization of a duodenal ulcer would explain the causation of
such a case as Oxley's very well. The idea of a tendency
to catarrh of this mucous membrane showing itself in several
members of one family does not seem out of the question.

A more probable theory of the way in which catarrhal inflam-
mation of the ducts might lead to obliteration of their lumen
is that a fibrous thickening of the walls might be produced
by the inflammatory action spreading to the fibrous tissue
there, as it so very frequently does in the case of the urethra.
It seems indeed, that this is the most probable theory of
the pathology of these cases.

Against it, we have the fact that no catarrh has ever
been demonstrated. It has, however, not been looked
for.

In favour of it, we have a number of considerations
which taken together seem pretty strong.

1) The fact that in adults a similar process sometimes
leads to a similar malformation.

2) That this theory would explain satisfactorily the
various appearances which we see on Table 2. Better
than any other. When we study this table we find
that in most of the cases the configuration of the de-
formed bladder and ducts is such as to suggest their having been caused by a lesion affecting a considerable extent of the tube at once and not by one merely producing a focal constriction. For one thing, there is no reason to suppose that complete blocking at one point would be followed by obliteration of the portion of tube below it from mere disease unless there were some morbid process in action there (in fact some of the descriptions are against this) - For another, the fact that in many cases the portion of tube above the obliteration is not dilated suggests the probability that the affection of the duct must have extended above the obliteration & thickened the walls there.

3) It would explain certain phenomena noted in different cases - e.g. In Dotze's case the narrowing of the duct must have been very gradual, because, although the child was jaundiced a few weeks after birth it was only a fortnight before death that all colour disappeared from the feaces. This is more easily explained by a theory of contraction of the walls of the duct than by constriction from without. Again, in Dr. West's case, the gall-bladder was felt distended at the beginning of the jaundice and at death, 9 months later, it was discovered to be much deformed by inflammatory action, so that the morbid process must have spread
spread slowly upward from the seat of obstruction, and destroyed it, as it is well known to do in cases of gall-stone.
(see Courvoisier loc. cit. (42) p. 18)

4) It would explain better than any other hypothesis the fact of some cases occurring which presented similar symptoms to those ones and which nevertheless recovered.
(see p. 37)

5) It would also explain satisfactorily the cases in which, after similar symptoms during life, the ducts were found not obliterated but merely thickened or not even that.
(see Appendix II p. 78)

6) It is also in favour of this theory that in the great majority of the cases there was no complication of the neighbouring blood-vessels; and that peritoneal adhesions in the neighbourhood are so seldom mentioned.

3. Peritonitis. is regarded by many as the cause of this condition.

In 1838, Sir J. Y. Simpson collected together in a most interesting paper a series of cases "to prove that peritonitis forms a common variety of fatal disease and probably constitutes one of the more frequent causes of death of the foetus during the later months of pregnancy," and a good deal has been written since then on the same subject.
The following facts regarding it may be mentioned:

It may begin early in intra-uterine life (Sir James had found it as early as the third month), or towards the end of pregnancy, or during the first days after birth. The exact time must be often impossible to discover owing to lack of symptoms. Its distribution may be general or it may occur in patches.

It is often certainly due to syphilis but it may also be caused, quite apart from that disease, by chill or over-exertion on the part of the mother. It may be set up by many local causes in the child such as rupture of the bladder or strangulation of the intestine, or it may be due to arteritis umbilicalis (Rungé loc. cit. p. 31). In some cases it would seem to be possibly determined by a local cause although none can be discovered—e.g. a case of twins with one placenta mentioned by Sir J. Y. Simpson where one had peritonitis and the other was healthy.

It may exist a considerable extent without there being any symptom whatever during life (see Billard 83). It may be, and often is fatal either before or after birth. If recovered from it often leaves adhesions. It is a not very uncommon cause of malformations of the intestine and other viscera (Böldfeld 99, Küttner 100).

There
There seem to be many considerations in favour of a localised form of this disease (peri-plephalitis) being the cause of obliteration of the duct in some of our cases. We have already seen (p. 45) that adhesions are mentioned as having occurred in several of the cases and that there was peritonitis effusion in a few others. The fact of the neighbouring blood vessels being obliterated by fibrous tissue in several of the cases (viz: 14, 15, 26 & 27) even without the other indications given in the description, seems almost proof of the peritonitis origin of the lesion in these.

The localisation of the obliteration in the different cases (Table 2) is not against this theory of its causation.

Although, however, we have pretty strong proof that a number of the cases were caused in this way, we have equally strong grounds for thinking that the large majority were not so produced. I have already alluded to the absence of traces of peritonitis or affection of the blood vessels in most of the cases, and to other reasons in favour of the disease having originated in the ducts themselves.

We must also remember that even if peritonitis is shown to have existed this does not necessarily prove it to have been the cause. In two of our cases (37 & 44) there was
was peritonitis present which was certainly secondary to the malformation of the ducts.

As to thickening of fibrous tissue in the neighborhood of the ducts, after paying attention to this point in a considerable number of post-mortem cases, I am inclined to receive statements regarding it with caution, as the normal limit of the amount of connective tissue there seems to vary and are difficult to determine. A mere statement to this effect is not a proof of post-peritonitis.

\[
\text{Predisposing Causes.}
\]

Having considered the arguments for and against developmental influence and the main forms of local disease capable of giving rise to obliteration of the ducts, we now come to the predisposing causes. Of these by far the most important is, of course, syphilis. Although others, such as injury, exposure and syringes, are worthy of notice also.

1) \underline{Syphilis} – A large proportion of those who have written on the pathology of this condition have ascribed it to syphilis. Some of them have had good reasons for doing so, others seem to have given this explanation merely in lack of a better. There is certainly a priori reason to think it a very probable cause; for syphilis is the
is the commonest cause for death of the ducts in adults, (apart from stone), it is also the commonest cause of infa-
ente peritonitis and a very likely promoter of interstitial as well as gummatus inflammation.

When, however, we go systematically over our large-
table of cases (Table 1) to see what arguments pro & con,
we can gather there, we find that the evidence in favour
of this causation is much more limited than we should
have been prepared to expect.

Some of the children were certainly syphilitics, of that
there can be no doubt. But if we separate these we find
that they are few in number and that there is an almost
absolute lack of evidence of syphilis in the case of all the
others.

The cases I refer to are No. 14, 15, 25, 26, 27, 346, &
we may add 6.13 & 18, although some doubt may
exist about them.

When we group these nine cases together we find
that they present certain symptoms which cause them
to differ somewhat from the other cases: for instance,
This small group contains 3 out of 5 of the cases
that were born prematurely. Also, all but one of
the cases in which there was adhesion or local thick-
ening of the capsule; and all in which the blood vessels
of the
Finally, although a good many facts regarding other means of the disease being apprehended, 

we have been made in nearly all of the cases, and the fact of no positive evidence at all having been obtained, is not in favor of this theory, certainly above all other theories. After all, we must remember that, notwithstanding its apparent simplicity, the disease in any of the infants of the 38 children—no infant of 30 days having survived—was in all of the cases, whenever we have a child of this age, we find a good deal of evidence against their being afflicted.

Secondly, there is no other factor of evidence of any sign of the disease in any of the infants of the 38 children—no infant of 30 days having survived—was in all of the cases, whenever we have a child of this age, we find a good deal of evidence against their being afflicted.

On the other hand, when we turn to the remaining 38 cases

all the children died, we compared the duration of life in all the cases were very limited. We may also note that in very short (1-8 days) as compared with the others.

He
the disease in several members of one family might be explained by supposing it to be a syphilitic affection. This suggestion however, cannot be accepted, for we never find a tendency for an extremely rare manifestation of syphilis to occur 2°, 3° or even 10 times in a family without any common symptom of the disease showing itself at the same time.

Fourthly, all the patients in these 38 cases were healthy (as far as noted) in other respects, and in no case is any symptom mentioned which could be put down to syphilis. It is certainly unlikely that if these 38 children were suffering from a syphilitic disease none of them should show any other traces of the disease, considering how long many of them lived and also that no postmortem sign of it should be noticed.

The facts then, so far as we have gathered them, seem to point to two things:

Firstly, that syphilis, either by setting up localized peritonitis, or catarh of the ducts, is to be regarded as the cause of the disease in a minority of the cases.

Secondly, that there is good reason to believe that syphilis has nothing to do with the etiology of the great majority of the cases.
b) Injuries and Exposure to Cold.

As we have already seen (p. 62), injuries & exposure to cold on the part of the mother may give rise to peritonitis in the child. Both these influences acting on the child are possible causes of peritonitis and of cataract, and the circumstances attending birth are such as to give opportunity for the operation of both.

On looking into the facts of the cases, however, we find that there is nothing in favour of this theory of causation in them.

No history of injury or chill to any of the mothers is given; and also a study of the nature of the labours & of the time of onset of the symptoms, of the state at birth and the time of death all point to the conclusion that these factors have nothing to do with causing the condition.

c) Erysipelas is a not uncommon cause of peritonitis, but usually the cases so caused are rapidly fatal.

One of Köstlin's cases seems to have been due to erysipelas followed by peritonitis; and erysipelas may have been the cause of death in another case.

The occurrence of intra-uterine erysipelas is an acknowledged fact (for references see Hirst), so that this is a possible though certainly a very unlikely cause of some of the other cases.
Conclusions

After a careful study of the foregoing facts one seems to arrive at clearer ideas on many of the points regarding this group of cases. For example it seems pretty certain that the disease begins always (or almost always) in the liver, where it has gone far enough to obstruct the free passage of bile into the intestine, it causes a form of cirrhosis of the liver which by interfering with the most important functions of that important organ and thereby setting up a sort of chronic blood poisoning, gradually leads to emaciation, diminished vitality and death. That some of the apparently inexplicable statements with regard to the exact date of onset of the jaundice may be explained by the influence of the changes in the hepatic circulation at birth.

Then, with regard to the pathology and etiology, we may conclude that probably more than one of the theories mentioned are necessary to account satisfactorily for all the cases. It seems very probable that a small number of them are due to the results of syphilitic peritonitis, but the great majority are more satisfactorily explained by the theory of fibrous contraction of the ducts themselves—probably following catarhal inflammation of their lining membrane. Obliteration from a congenital defect
defect in development is very doubtful; but it is quite possible that an original congenital narrowing of this nature may form an important factor in the etiology.

There is no proof and no probability that the disease is a manifestation of syphilis, as has been so frequently asserted.

There are, however, a number of most important and interesting questions to which we have got no answer at all. We have no explanation, for example, of the recurrence of the disease in several children of one mother; we do not know at what period of intra-uterine life the disease originates; and we have no idea of the predisposing causes in the large majority of the cases.

Zuller's more exact clinical details and fuller and more thorough microscopical examinations are much needed.
Appendix 1

Descriptions of precise lesions of gall bladder & ducts
of cases in Table 1.

Case 1. (Wells) Gall bladder represented by two small sacs without any outlet, one of them the size of a pea, the other twice as large, containing tenacious matter of a greenish colour and not unlike impregnated bile. Hepatic ducts impregnated and greatly dilated.

2. (Morgan) Gall bladder not larger than a pea, containing only mucous and no true bile. Cystic and both hepatic ducts patent but empty. Ductus cholodochus completely obliterated—being represented by a fibrous cord which joined the intestine at the usual spot. Tube obliterated for a little more than 1/4 an inch. No opening into bowel.

3. (Smith) Gall bladder rudimentary. Hepatic and common ducts absent.

4. (Henschel) Gall bladder "present in a rudimentary condition" not a trace of bile ducts, and the opening of the ductus cholodochus into the duodenum could not be discovered.

5. (Marchionis) Gall bladder—extremely small and collapsed, and contained only a few drops of colourless fluid. Cystic and hepatic ducts dilated, in other respects normal. Common duct completely obliterated, its place being occupied by a small quantity of acellular bursa. Opening of duct into duodenum found with difficulty and would not admit a probe. A few fibrous bands of adhesion on under surface (4 lives).

6. (Wickham Legg) Gall bladder "shrunken, holds a small amount of yellowish fluid. Cystic duct "opens without any winding into a cyst the size of a large marble placed to the right side of the portal fissure between the liver and duodenum in the hepatoduodenal ligament. This cyst also receives the hepatic duct coming from the liver. It is a blind sac." The hepatic duct free and holding a yellowish fluid as far as a few lines from the liver.

7. (Rhobi's) Gall bladder dilated with a non-biliary mucoid fluid. Cystic duct smaller than normal. No trace of either hepatic duct. Common duct previous and containing mucous only. Opening into duodenum normal.
Case 8 (Robb's) Gall bladder rudimentary. No trace of hepatic or common duct. They appear lost in fibrous tissue.

9. (Halsey's) Left hepatic duct split into threads - Common duct all right down to a few lines from duodenum beyond which bile would not flow, and the duct was constricted and would hardly admit a little brush bristle.

10. (Willis's) Gall bladder appeared to be absent, but, on dissection of cellular tissue, a narrow channel was found just large enough to hold a bristle. No communication from this to the bile ducts which seemed totally obliterated, merely some connective tissue existing between hepatic artery and portal vein. In the duodenum the opening of the duct was found as usual and a probe passed freely into the pancreas but no passage beyond.

11. (Harley's) Gall bladder distended to the size of a small hen's egg, full of fluid bile. Cystic and hepatic ducts enlarged and full of fluid bile. Common duct simplified. The lower part looked like a mere cord of solid fibrous tissue.

12. (Hume's) Gall bladder very small and contained only a little colourless mucus. A pedunculated cystic duct between it and the duodenum and was joined by the pancreatic duct in the usual manner. This cystic duct was joined in the normal position of the hepatic duct by a slender impermeable cord of connective tissue which was found to divide near the liver into two thinner cords in position of ducts from right and left lobes - a minute dilatation existed at the commence- ment of the fibrous cord which represented the left duct.

13. (Roth's) In the place of the gall bladder there is a cord the thickness of a round quill which contained two separate cystic the size of peas, filled with clear fluid. Not even a solid band of fibrous tissue to represent the cystic duct or the hepatic or common ducts. At the porta hepatis there is a dilated duct about 1 in. long (beginning of hepatic duct) In the
Incision, the papilla is present, but a probe only passes into it for 2 lines.

Case 14. (Bing's iv) Gall-bladder filled with tough, deeply greenish yellow mucous. Cystic and hepatic ducts changed into an irregular mass of fibrous tissue which showed nowhere any trace of hollowing.

15. (Bing's iv) Gall-bladder filled with normal-looking bile. The bile duct, mere cords with no lumen.

16. (Bing's iv) Gall-bladder very full of apparently normal bile. A probe only passes a few lines into the cystic duct (from the gall-bladder) The hepatic duct only pervious at its commencement at the liver. The common duct only pervious at the end.

17. (Donop) Gall-bladder smaller than normal. Cystic and hepatic ducts normal. Common duct obliterated in its whole course except a part the length of half the thickness of a thumb.

18. (Lotgey) Gall-bladder in normal position, very firmly attached to the surface of the liver by extremely dense arterial tissue. Maccid, not very full, whitish grey in colour, containing clear, thin, slightly acid mucous. No duct found passing from it. In place of the cystic duct, a few solid, whitish cords which look as if they had been formed out of it & which lose themselves in the connective tissue. Common duct pervious but very narrow. The right hepatic duct can be followed some distance into the liver; the left soon becomes a fibrous cord accompanying the corresponding branch of the portal vein.

19. (Hechler) Gall-bladder represented by a solid cord to a line thick, with an enlargement on the end of it near the edge of the liver. Hepatic and common ducts quite absent. Papilla present but does not admit a probe.
20. (Hennig's) Gall bladder contained a little greyish yellow mucus. Common duct quite closed.

21. (Hennig's) Gall bladder contained much dark greenish brown bile. Cystic duct narrowed rapidly from its opening into the common duct; finally its lumen ceased entirely. Hepatic duct normal. Normal red-brown bile in duodenum.

22. (Freund's) In the groove for the gall bladder there was, instead of it, a blind tube about 1/2 cm. long and 1/4 cm. wide, with two slight hour-glass contractions which implicated the whole circumference. This contained a few drops of clear watery sticky fluid. Followed backwards, this tube ran into a solid white cord of connective tissue about 2.5 millimetres thick which became lost in the hepatic-duodenal ligament near the transverse fissure. This cord had no lumen. No trace of hepatic or common ducts.

23. (Peloq.) Gall bladder of normal size but contained no bile. Cystic and hepatic ducts both patent and opened into large cysts. Globular tumour about the size of a cocoanut below free margin of liver. No opening into the intestine papilla visible but impervious.

24. (Barham's) Gall bladder atrophied, 1/2 inches long, the thickness of a goose quill all the ducts obliterated and represented by cords, content of duodenum not tinged with bile.

25. (Loomis') Gall bladder collapsed but patent. Left hepatic duct normal. Right hepatic and cystic ducts represented by fibrous cords without lumen. Common duct normal.

26. (Schüppel') Gall bladder contained only watery mucus. Bile ducts represented by hard cords.

27. (Schüppel') same condition.
Case 28. (Frerenczi) Gall bladder 3 cm long, of conical shape, broader at the neck than at the fundus which ends in a thin cord ending. The cavity is reduced to a very thick-walled canal about 1/2 cm long and 1/4 cm in diameter. It contains a brownish pulpy mass. Toward the cystic duct it becomes insensible to the finest probe. The cystic duct opens into the common duct which, at first wider than normal gradually passes into a more thread-like lumen which loses itself in the connective tissue of the hepatoduodenal ligament. The hepatic ducts are both present and can be followed distinctly into the liver substance. They have no lumen. The ductus choledochus has a punctiform lumen on section.

29. (Kostlin's v) Gall bladder quite full of thin clear green fluid. Cystic duct completely closed, without any change of its tissue or of neighboring organs.

30. (Kostlin's iv) Gall bladder distended with watery clear yellow fluid. Cystic duct closed.

31. (Campbell's v) Gall bladder very small and collapsed, contained only a little mucous in colour and consistence resembling gelatine and formed a close sac having no outlet; the secretory ducts leading from the gall bladder and bile being absent.

32. (Campbell's iv) Neither a gall bladder or bile ducts could be discovered.

33. (Anderson's) Gall bladder distended with greenish yellow bile. Cystic duct patent. Hepatic duct patent to just beyond its junction with the cystic duct where it ended in a cul-de-sac. Imperforate tissue in its place.

34. (Goldbl's v) Gall bladder flaccid, containing a droplike of serous fluid. Ducts imperforate.

35. (Goldbl's iv) Gall bladder partially filled with a little gelatinous fluid. Cystic and hepatic ducts apparently closed. Common duct dilated.
Case 36. (Dean's) Bile ducts impervious.

37. (Blake White's) Gall bladder filled with blackish syrupy bile. Cystic duct evidently
constructed. Hepatic ducts presented no abnormal appearance. Common duct im-
pervious throughout its length— in its place, a fibrous, cord-like band extending
to the duodenum.

38. (Danforth's) Gall bladder very much distended with bile of the consistency of
syrup. Cystic and hepatic ducts presented no unusual appearance but were slightly
enlarged. Common duct distended and presented an abrupt termination without
reaching the intestinal canal.

39. (Howe's) No gall bladder, nor any duct leading from the liver into the duodenum.

40 & 41. (Blundells) Hepatic ducts terminated blindly. No bile entered the intestine.

42. (Hommes's) Duct impervious.

43. (Jenkins') Cystic and hepatic ducts impervious.

44. (Simpson's) No trace at all of the gall bladder. Ductus choledochus normal
but did not divide until it entered the liver.

45. (Hobson's) Gall bladder empty and shrunken. The cystic and common ducts re-
presented by a mere cord which got lost as it passed downwards towards the duode-
num, enlarged lymphatic gland in the neighborhood of the obliterated duct.

46. (Meyer's) Gall bladder absent, its place being occupied by dense eviscerating
tissue in which the ductus choledochus ends blindly.
<table>
<thead>
<tr>
<th>No.</th>
<th>Reports by</th>
<th>Disease of Children's Sick.</th>
<th>Sex of child born from infected parents</th>
<th>Age at death</th>
<th>Signs of syphilis</th>
<th>No.</th>
<th>Signs of syphilis</th>
<th>Age of death</th>
<th>3rd Day</th>
<th>3rd Day</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Underwood</td>
<td>Distinct Symptoms of child died.</td>
<td>Male</td>
<td>8 days</td>
<td>Varied from day to day</td>
<td>8</td>
<td>Varied from day to day</td>
<td>1</td>
<td>Healthy</td>
<td>Healthy</td>
</tr>
<tr>
<td>2</td>
<td>Gleyre</td>
<td>Distinct Symptoms of child died.</td>
<td>Female</td>
<td>17 days</td>
<td>Excellent</td>
<td>17</td>
<td>Excellent</td>
<td>1</td>
<td>Healthy</td>
<td>Healthy</td>
</tr>
<tr>
<td>3</td>
<td>Bedner</td>
<td>Distinct Symptoms of child died.</td>
<td>Female</td>
<td>18 days</td>
<td>Delicate</td>
<td>18</td>
<td>Delicate</td>
<td>1</td>
<td>Healthy</td>
<td>Healthy</td>
</tr>
<tr>
<td>4</td>
<td>Campbell</td>
<td>Distinct Symptoms of child died.</td>
<td>Male</td>
<td>11 days</td>
<td>Healthy</td>
<td>11</td>
<td>Healthy</td>
<td>1</td>
<td>Healthy</td>
<td>Healthy</td>
</tr>
<tr>
<td>5</td>
<td>E. Ray</td>
<td>Distinct Symptoms of child died.</td>
<td>Male</td>
<td>12 days</td>
<td>Apparently strong</td>
<td>12</td>
<td>Apparently strong</td>
<td>1</td>
<td>Healthy</td>
<td>Healthy</td>
</tr>
<tr>
<td>6</td>
<td>Hatfield</td>
<td>Distinct Symptoms of child died.</td>
<td>Male</td>
<td>13 days</td>
<td>Normal</td>
<td>13</td>
<td>Normal</td>
<td>1</td>
<td>Healthy</td>
<td>Healthy</td>
</tr>
<tr>
<td>7</td>
<td>D'Spine</td>
<td>Distinct Symptoms of child died.</td>
<td>Male</td>
<td>33 days</td>
<td>None</td>
<td>33</td>
<td>None</td>
<td>1</td>
<td>Healthy</td>
<td>Healthy</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Good</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>2</td>
<td>Poor</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>3</td>
<td>Fair</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>4</td>
<td>Good</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>5</td>
<td>Poor</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>6</td>
<td>Fair</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>7</td>
<td>Good</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>No.</td>
<td>Colour</td>
<td>Surface</td>
<td>Capsule</td>
<td>Pericholecystitis</td>
<td>Tissue</td>
<td>Consistence</td>
<td>On Section</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>-----</td>
<td>--------</td>
<td>---------</td>
<td>---------</td>
<td>-------------------</td>
<td>--------</td>
<td>-------------</td>
<td>------------</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>natural&lt;br&gt;in the whole</td>
<td></td>
<td></td>
<td>slight adhesion of base &amp; right lobe to the peritoneum</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>dark-green, earthy</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>stained with bile</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>olive-green</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>olive-green</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>dark-blackish brown</td>
<td>unchanged</td>
<td></td>
<td>normal&lt;br&gt;greenish yellow, mottled</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>olive-green</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>No.</th>
<th>Gall-bladder</th>
<th>Contents</th>
<th>Common Duct</th>
<th>Ductus hepatica</th>
<th>Ductus choledochus</th>
<th>Microscopic appearances</th>
<th>Spleen</th>
<th>Other marked conditions</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>nearly filled with bile&lt;br&gt;of a deep yellow color&lt;br&gt;permeable</td>
<td>permeable</td>
<td>permeable</td>
<td>permeable</td>
<td>no bile in intestine</td>
<td></td>
<td>Heart larger than normal</td>
<td>3 P. fluid in pericardium</td>
</tr>
<tr>
<td>2</td>
<td>quite empty and embossed&lt;br&gt;a small soft dark mass</td>
<td>embossed&lt;br&gt;hard, white, firm</td>
<td>same</td>
<td>same</td>
<td>no bile in intestine</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>markedly enlarged&lt;br&gt;very full&lt;br&gt;nearly&lt;br&gt;thick bile</td>
<td>permeable&lt;br&gt;embossed&lt;br&gt;hard, white, firm</td>
<td>permeable&lt;br&gt;embossed&lt;br&gt;hard, white, firm</td>
<td>permeable&lt;br&gt;embossed&lt;br&gt;hard, white, firm</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>a quantity of bile&lt;br&gt;permeable&lt;br&gt;hard, white, firm</td>
<td>permeable&lt;br&gt;hard, white, firm</td>
<td>permeable&lt;br&gt;hard, white, firm</td>
<td>permeable&lt;br&gt;hard, white, firm</td>
<td>permeable&lt;br&gt;hard, white, firm&lt;br&gt;blocked by an inflammation of the peritoneum</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>permeable</td>
<td>permeable</td>
<td>permeable</td>
<td>permeable</td>
<td>open</td>
<td>permeable</td>
<td>permeable</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>it is implied that they were permeable</td>
<td>permeable</td>
<td>permeable</td>
<td>permeable</td>
<td>permeable</td>
<td>permeable</td>
<td>permeable</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>dark sticky bile&lt;br&gt;perfectly permeable</td>
<td>same</td>
<td>same</td>
<td>same</td>
<td>normal&lt;br&gt;interstitial chronicitis&lt;br&gt;both sides of liver &amp; in biles</td>
<td>large&lt;br&gt;interstitial inflammation of lung (pleuritis)&lt;br&gt;brown</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Appendix II

Cases of Infantile Jaundice with symptoms similar to those of Obliteration of the Bile ducts but in which, after death, the Bile ducts were found to be patent.

A considerable number of cases have been reported in which children with symptoms exactly the same as those of our cases were found, post mortem, to have diseased livers, but in which no mention of the state of the bile ducts is made. These cases are, of course, of no use in the present enquiry.

In a few, however, a more or less careful account of the appearances of the ducts is given, and I have tabulated (Table 3) the most satisfactory of these, that they may be compared with the cases on Table 7.

In comparing them one is struck with the close resemblance of the two sets of cases as far as the details are given. In fact the resemblance is such that we can scarcely avoid the conclusion that they must be different stages or degrees of the same disease.

I think I need only draw special attention to two points. Firstly, in none of the cases on Table 3 did the patient live long. The average duration of life being 17 days, as against fully 2½ months in the case of those on Table
Table 1.

Secondly, in the description of the condition of the ducts we find that in one very carefully examined Case (Cheyne) the walls were thickened and white & contracted ("like those of arteries"). Also, that in other cases the ducts although said to be "pervious" are not fully described, are not ever called "normal" & were never microscopically examined; so that one cannot help thinking that in some, if not all, of these there may have been some thickening and contraction of the walls or else some internal obstruction from catarhal swelling of the mucous membrane.

If this were so, we may readily, and I think not unreasonably, explain these cases as being earlier stages of the disease - cases in which the morbid process has gone so far as to produce more or less (practical) occlusion of the lumen although the patients have not lived long enough for complete obliteration of the duct to ensue.

* It is well known that the pressure of tumours X causes complete stoppage of the passage of bile long before absolute obliteration is produced, so that at the post mortem probes of a stream of water can easily pass through a duct that was impervious to bile in life. Also, catarhal conditions of the mucous membrane may give rise to water blocking during life & yet P.M. there may be no actual obstruction (see Eggib ur. cit. 8 p 252).
Appendix III.

Case of Obliteration of the Small Intestine.

Mrs 9's, infant, two and a half days old, seen first on 12th Nov. 1890 along with S. Home Ross.

Complaints: Cyanosis, jaundice, complete obstruction, constant vomiting.

Family History: Father and mother healthy; no syphilis.

Other children: The first child is alive but stillborn. The second was stillborn (full term, cross birth). The third pregnancy ended in a miscarriage at the 5th m. owing to the presence of an ovarian tumour. The patient is the fourth child. No other miscarriages.

Pregnancy: When the mother was about two months pregnant, she had a large ovarian cyst removed by laparotomy (Dr. Halliday Croom). The operation was not a complicated one. Shortly after it she had a threatening of miscarriage which was checked by a large dose of morphine; after that everything went well till the full term.

Birth: Easy and natural (3 a.m. 10th Nov.). There was an extremely large quantity of liquor amnii and an unusually thick layer of vernix caseosa. The child was thought to be of a brighter red colour than usual and his extremities were rather blue. Since birth, he has vomited everything given to him (milk & water and 4 doses of castor oil). No motion of any kind has come.

* The last menstruation began on 4th March; the operation took place on 23rd May.

2 Both of perfectly normal colour.
from the bowel. The skin has gradually assumed a distinct yellow colour.

Present Condition

The child is well developed and of natural size. The skin is bright red with a strong orange tint. The conjunctivae are only slightly yellow. The lips, the vertex of the scalp, the palms and soles, and the neighbourhood of the anus are all markedly cyanosed and there is a bluish tinge over some other parts of the body; the tongue and gums however, are quite free from this. The child cries constantly as if from hunger. Head well formed. Thorax well formed, considerable indrawing of the epigastic and episternal region with each respiration. The lungs seem normal but the percussion note over the bases is not very clear. The heart also appears normal; the apex-beat is felt indistinctly in the 5th space in the left nipple line. No abnormality in the heart sounds is to be heard in any area.

Abdomen not distended but its walls are very tense. On percussion a tympanitic note is got over the region of the stomach, but over all the lower part of the abdomen, the note is absolutely dull. The liver and spleen cannot be felt. The anus seems small but it admits the little finger without much difficulty and is felt to contain a few soft rounded masses. These
when removed by an enema, are found to amount to rather more than a teaspoonful in bulk. The matter is absolutely without odour and of a whitish colour, with no tinge of green. It consists of rounded agglomerations of little many-sided bodies from the size of a lentil to that of a split-pea; these have an ivory white colour on section and the consistence of lightly boiled white of egg, and they are held together by thick white mucous. There is also a little pasty amorphous matter. (The contents of the rectum were put into spirit and afterwards examined microscopically. They seemed to be composed of epithelial debris.)

When the rectum is cleared, the little finger can, with some difficulty, be passed up as far as the sigmoid flexure, and there is no obstruction.

The vomited matter has a greenish yellow tinge.

The urine passes freely; at first it is said to have been clear and not to have stained the clothes; now it is thick and dark and stains them yellow. On the prepuce and on the napekin there is a large quantity of "brick-dust" deposit.

Diagnosis: Probably obliteration of the bowel.

Treatment: In addition to the enema, several injections of glycerine were given.

Progress: 15th Nov. Child more deeply jaundiced (orange)
Continues to vomit everything. Has passed nothing per anum except a few drops of colourless fluid.

19th Nov. Has not vomited at all for two days. Although taking a little sugar and water. One very small colourless motion. Urine dark, staining napkins yellow. Child becoming extremely emaciated. Cyanotic ting almost gone. Liver edge extends about a finger-breadth below the margin of the ribs; the spleen cannot be felt.

On removing the napkin, the child is found to have passed about half a teaspoonful of dark (but yet bright) green matter homogeneous and stringy. The shade of colour is not so dark as that of ordinary meconium and is a good deal yellower. This was submitted to F. W. Kill Paton who kindly examined it and reports:

"Specimen consisted of several very small irregular masses with a greenish yellow colour. Some parts distinctly green, other parts yellow. The pigment is insoluble in alcohol, chloroform or ether. On treatment with alcohol and sul-
phuric acid, the yellow pigment goes into solution; the green pigment does not. The solid masses of green pigment when treated with nitrous-nitric acid give a faint but distinct play of colours.

In their solubility these pigments differ from the bile pigments but they give Greulich's test. Possibly they may consist of bile pigments which have been for a long per..."
Viscera of Case of Obliteration of the Small Intestine

Shewing nature and seat of lesion.
...in the bowel and have undergone some change. But it is also possible that they may be pigments produced in some other way. The specimens were so small that it was not possible to make an extended investigation.

Under the microscope it was found full of micro cocci.

20th November. Child died at 7 a.m. aged 10 days 6 hours.

Post mortem. Body extremely anaemic, slightly but distinctly jaundiced, no obvious cyanosis. Rigor mortis considerable, some dark green discolouration over the abdomen towards the left side.

On opening the abdomen, a large oblong tumour of a purplish red colour is found to occupy the greater part of the left half of its cavity. This is found to be the distended portion of gut just above the seat of the obliteration (Plate 7). The rest of the bowel is seen contracted to the fullest extent so as to be from \( \frac{1}{8} \) to \( \frac{1}{4} \) of an inch in diameter and is very pale and yellowish in colour. In some places little dull-green masses are seen through the walls of thecontracted gut.

The peritoneum is smooth and glistening; there is no sign of peritonitis either old or recent.

The liver seems tolerably normal to the naked eye, but is somewhat enlarged & very dark & congested.

Ureth of bladder.
Gall bladder normal in appearance; filled with dark bile.
Cystic duct seems normal & permeable, but is unusually long.
Hepatic & common ducts, also pervious & apparently normal.

Pancreas normal. Spleen normal.

Kidneys: congested, extremely copious uric acid infarcts in the pyramids.
Mesenteric glands not enlarged.

Esophagus & Stomach normal.

Duodenum. Just about the commencement of the duodenum, the gut becomes enormously dilated. The dilated portion measures ten inches in length and one and a half inches in diameter; it is of a dark purplish brown color. It comprises the duodenum and the first few inches of the jejunum. Its lower extremity is an abruptly rounded end. It is perfectly closed and there is nothing visible connecting it with the next portion of gut. The mesentery belonging to it also comes to an abrupt end. The blood vessels in the mesentery are very large.

A short distance from this dilated piece of bowel, is a small horse-shoe shaped bit of gut, about 1½ in. long by ½ in. in diameter, with rounded blind ends & connected with the back wall of the abdominal cavity by a separate little flap of mesentery of its own.

* This distended piece of bowel contained brownish fluid, evidently the juices given by mouth plus biliary secretion.*
About the same distance on the other (right) side of this fragment is the beginning of the rest of the small intestine. It starts as a little fibrous thread about ¼ in. in length, which then dilates into a tube varying from ¼ to ½ in. in diameter. It seems quite empty except in a few places where the little bits of greyish green matter are seen through the walls as before mentioned.

The large intestine is similarly contracted being only about ¼ in. in diameter and in a similarly empty state. Thorax. A little collapse at both bases, otherwise the lungs are normal. Thymus gland of medium size. Pleura and pericardium normal.

Heart normal. All the valves healthy and septa complete with the exception of a small valvular opening in the position of the foramen ovale (posteriorly). Ductus arteriosus patent, admitting a probe the size of a number one catheter.

**Remarks**

This case is in many respects a very interesting one, but as it is introduced here mainly on account of the occurrence in it of green stools which were probably not bilary, a very few words on the other points of interest will suffice.

*Congenital*
Congenital obliteration of the bowel has been referred to many causes (see Ahlfeld 99 & Kuttner 100). In this case, when we look at the form of the intermediate portion of gut, its relation to the rest of the bowel and the shape of its bit of mesentery, we cannot doubt that the malformation is the result of a twist of the lesser bowel occurring in early intrauterine life. This is demonstrated more clearly than by words in Fig. 7.

What caused the twist, it is impossible to discover. The question naturally arises, Had the ovariotomy anything to do with causing the malformation? At first sight one is inclined to suspect it and certainly the lesion is one which might very well have dated from that period of intra-uterine life. On the other hand, we know that ovariotomy is frequently performed during pregnancy and yet I have not been able to find a single case in which abnormalities in the child have been noted. It is also not easy to see how an operation of this kind should conduce to the occurrence of a twist of the infant's bowel. Probably, therefore, there is no reason to suspect any connection between them.

* In addition to consulting books, I have asked my friends Mr. Stew Keith, J. D. Malcolm of London and Dr. H. Croom, B. Hart & Barbour here and none of them have ever heard or known of a case of this kind happening.
The point that concerns us here however is—What was that green stuff? As we see from Dr. Noël Paton's report, the analysis which was possible of the small quantity submitted to him did not settle the question entirely. The fact of a play of colours being observed with nitrous nitric acid is the only point in favour of the green matter containing bile-pigment. On the other hand, the other reactions were quite different from those given by ordinary meconium (see Zweifel loc. cit. (60) p. 474). Dr. Paton suggests that possibly the length of time the matter had been in the bowel might account for these differences but it cannot have been any longer there (if so long) as ordinary meconium.

Can we by reasoning from clinical facts get any nearer the truth? I think we can.

As we have already seen (page 23), the main bulk of normal meconium is made up of matters swallowed by the infant in utero. In this case, these matters would be arrested by the obliteration and retracted. This probably accounts for the fact noticed here as in other similar cases, that the liquor amni and venous casesa were excessive in amount. It also accounts for there being at birth in this child's bowel not more than 2½ ounces of content, instead of about 2½ oz. as usually at the case.
In a normal new-born child's bowel, the nearer the anus the darker is the colour of the contents (p. 29); therefore the first motion is the darkest of all; and they get lighter till about the third day when they give place to ordinary faeces.

In this child the first two motions were absolutely devoid of green or yellow colour and it was only on the 10th day that the first coloured motion passed — after the administration of frequent enemata. The green matter was full of microorganisms.

Although the above facts do not absolutely prove that the green matter in question did not owe its colour to bile-pigment, yet, taken together, they certainly make it appear very improbable indeed that it did so. It seems much more likely that the green colour was due to the action of microorganisms.
Literature

8. " " " 1896. p. 73.
29. A. D. Campbell, Northern Journal of Medicine, Aug., 1844, p. 237.
34. Danforth, Chicago Medical Journal, 1870, p. 110.
35. Sir Everard Home, Philosophical Transactions, 1813, p. 156.
70. Leyden, "Beiträge zur Pathologie des Schweins." Berlin, 1861.
75. Lander Brunton, Pharmacology and Therapeutics. 3. edit. p. 602.
76. O. V. Bolouson, Arch. für Experiment. Pathol. XIV p. 200-1881.
91. Wenzel Gruber, "Virchow's Archiv," LXIII.
100. Küttner, "Virchow's Archiv," Bd. 54, p. 34.
<table>
<thead>
<tr>
<th>No.</th>
<th>Name</th>
<th>Age at Birth</th>
<th>Sex</th>
<th>Marital Status</th>
<th>Religion</th>
<th>Place of Birth</th>
<th>Race</th>
<th>Occupation</th>
<th>Education</th>
<th>Length of Marriage</th>
<th>Age of Mother</th>
<th>Birth Order</th>
<th>Birth Order in Family</th>
<th>Birth Location</th>
<th>Birth Weight</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>West</td>
<td>3.5 weeks</td>
<td>Male</td>
<td>Single</td>
<td>English</td>
<td>53</td>
<td>White</td>
<td>Farm worker</td>
<td>School</td>
<td>0</td>
<td>0</td>
<td>1st child</td>
<td>1st child in family</td>
<td>England</td>
<td>13lb</td>
</tr>
<tr>
<td>2</td>
<td>J.H. Moore</td>
<td>2 weeks</td>
<td>Male</td>
<td>Single</td>
<td>English</td>
<td>52</td>
<td>White</td>
<td>Farm worker</td>
<td>School</td>
<td>0</td>
<td>0</td>
<td>2nd child</td>
<td>2nd child in family</td>
<td>England</td>
<td>12lb</td>
</tr>
<tr>
<td>3</td>
<td>E. Smith</td>
<td>2 weeks</td>
<td>Male</td>
<td>Single</td>
<td>English</td>
<td>52</td>
<td>White</td>
<td>Farm worker</td>
<td>School</td>
<td>0</td>
<td>0</td>
<td>3rd child</td>
<td>3rd child in family</td>
<td>England</td>
<td>11lb</td>
</tr>
<tr>
<td>4</td>
<td>Henley</td>
<td>2 weeks</td>
<td>Male</td>
<td>Single</td>
<td>English</td>
<td>52</td>
<td>White</td>
<td>Farm worker</td>
<td>School</td>
<td>0</td>
<td>0</td>
<td>4th child</td>
<td>4th child in family</td>
<td>England</td>
<td>10lb</td>
</tr>
<tr>
<td>5</td>
<td>Merriam</td>
<td>2 weeks</td>
<td>Male</td>
<td>Single</td>
<td>English</td>
<td>52</td>
<td>White</td>
<td>Farm worker</td>
<td>School</td>
<td>0</td>
<td>0</td>
<td>5th child</td>
<td>5th child in family</td>
<td>England</td>
<td>9lb</td>
</tr>
<tr>
<td>6</td>
<td>Weddell</td>
<td>2 weeks</td>
<td>Male</td>
<td>Single</td>
<td>English</td>
<td>52</td>
<td>White</td>
<td>Farm worker</td>
<td>School</td>
<td>0</td>
<td>0</td>
<td>6th child</td>
<td>6th child in family</td>
<td>England</td>
<td>8lb</td>
</tr>
<tr>
<td>7</td>
<td>Sibley</td>
<td>2 weeks</td>
<td>Male</td>
<td>Single</td>
<td>English</td>
<td>52</td>
<td>White</td>
<td>Farm worker</td>
<td>School</td>
<td>0</td>
<td>0</td>
<td>7th child</td>
<td>7th child in family</td>
<td>England</td>
<td>7lb</td>
</tr>
<tr>
<td>8</td>
<td>Sibley</td>
<td>2 weeks</td>
<td>Male</td>
<td>Single</td>
<td>English</td>
<td>52</td>
<td>White</td>
<td>Farm worker</td>
<td>School</td>
<td>0</td>
<td>0</td>
<td>8th child</td>
<td>8th child in family</td>
<td>England</td>
<td>6lb</td>
</tr>
<tr>
<td>9</td>
<td>Skeeter</td>
<td>2 weeks</td>
<td>Male</td>
<td>Single</td>
<td>English</td>
<td>52</td>
<td>White</td>
<td>Farm worker</td>
<td>School</td>
<td>0</td>
<td>0</td>
<td>9th child</td>
<td>9th child in family</td>
<td>England</td>
<td>5lb</td>
</tr>
<tr>
<td>10</td>
<td>Wilks</td>
<td>2 weeks</td>
<td>Male</td>
<td>Single</td>
<td>English</td>
<td>52</td>
<td>White</td>
<td>Farm worker</td>
<td>School</td>
<td>0</td>
<td>0</td>
<td>10th child</td>
<td>10th child in family</td>
<td>England</td>
<td>4lb</td>
</tr>
<tr>
<td>11</td>
<td>Sibley</td>
<td>2 weeks</td>
<td>Male</td>
<td>Single</td>
<td>English</td>
<td>52</td>
<td>White</td>
<td>Farm worker</td>
<td>School</td>
<td>0</td>
<td>0</td>
<td>11th child</td>
<td>11th child in family</td>
<td>England</td>
<td>3lb</td>
</tr>
<tr>
<td>12</td>
<td>Sibley</td>
<td>2 weeks</td>
<td>Male</td>
<td>Single</td>
<td>English</td>
<td>52</td>
<td>White</td>
<td>Farm worker</td>
<td>School</td>
<td>0</td>
<td>0</td>
<td>12th child</td>
<td>12th child in family</td>
<td>England</td>
<td>2lb</td>
</tr>
<tr>
<td>13</td>
<td>Roth</td>
<td>2 weeks</td>
<td>Male</td>
<td>Single</td>
<td>English</td>
<td>52</td>
<td>White</td>
<td>Farm worker</td>
<td>School</td>
<td>0</td>
<td>0</td>
<td>13th child</td>
<td>13th child in family</td>
<td>England</td>
<td>1lb</td>
</tr>
<tr>
<td>14</td>
<td>Canning</td>
<td>2 weeks</td>
<td>Male</td>
<td>Single</td>
<td>English</td>
<td>52</td>
<td>White</td>
<td>Farm worker</td>
<td>School</td>
<td>0</td>
<td>0</td>
<td>14th child</td>
<td>14th child in family</td>
<td>England</td>
<td>0lb</td>
</tr>
<tr>
<td>15</td>
<td>Canning</td>
<td>2 weeks</td>
<td>Male</td>
<td>Single</td>
<td>English</td>
<td>52</td>
<td>White</td>
<td>Farm worker</td>
<td>School</td>
<td>0</td>
<td>0</td>
<td>15th child</td>
<td>15th child in family</td>
<td>England</td>
<td>0lb</td>
</tr>
<tr>
<td>16</td>
<td>Canning</td>
<td>2 weeks</td>
<td>Male</td>
<td>Single</td>
<td>English</td>
<td>52</td>
<td>White</td>
<td>Farm worker</td>
<td>School</td>
<td>0</td>
<td>0</td>
<td>16th child</td>
<td>16th child in family</td>
<td>England</td>
<td>0lb</td>
</tr>
<tr>
<td>17</td>
<td>Brown</td>
<td>2 weeks</td>
<td>Male</td>
<td>Single</td>
<td>English</td>
<td>52</td>
<td>White</td>
<td>Farm worker</td>
<td>School</td>
<td>0</td>
<td>0</td>
<td>17th child</td>
<td>17th child in family</td>
<td>England</td>
<td>0lb</td>
</tr>
<tr>
<td>18</td>
<td>Burke</td>
<td>2 weeks</td>
<td>Male</td>
<td>Single</td>
<td>English</td>
<td>52</td>
<td>White</td>
<td>Farm worker</td>
<td>School</td>
<td>0</td>
<td>0</td>
<td>18th child</td>
<td>18th child in family</td>
<td>England</td>
<td>0lb</td>
</tr>
<tr>
<td>19</td>
<td>Nashel</td>
<td>2 weeks</td>
<td>Male</td>
<td>Single</td>
<td>English</td>
<td>52</td>
<td>White</td>
<td>Farm worker</td>
<td>School</td>
<td>0</td>
<td>0</td>
<td>19th child</td>
<td>19th child in family</td>
<td>England</td>
<td>0lb</td>
</tr>
<tr>
<td>20</td>
<td>Newman</td>
<td>2 weeks</td>
<td>Male</td>
<td>Single</td>
<td>English</td>
<td>52</td>
<td>White</td>
<td>Farm worker</td>
<td>School</td>
<td>0</td>
<td>0</td>
<td>20th child</td>
<td>20th child in family</td>
<td>England</td>
<td>0lb</td>
</tr>
<tr>
<td>21</td>
<td>Newman</td>
<td>2 weeks</td>
<td>Male</td>
<td>Single</td>
<td>English</td>
<td>52</td>
<td>White</td>
<td>Farm worker</td>
<td>School</td>
<td>0</td>
<td>0</td>
<td>21st child</td>
<td>21st child in family</td>
<td>England</td>
<td>0lb</td>
</tr>
<tr>
<td>22</td>
<td>Brower</td>
<td>2 weeks</td>
<td>Male</td>
<td>Single</td>
<td>English</td>
<td>52</td>
<td>White</td>
<td>Farm worker</td>
<td>School</td>
<td>0</td>
<td>0</td>
<td>22nd child</td>
<td>22nd child in family</td>
<td>England</td>
<td>0lb</td>
</tr>
<tr>
<td>23</td>
<td>Oates</td>
<td>2 weeks</td>
<td>Male</td>
<td>Single</td>
<td>English</td>
<td>52</td>
<td>White</td>
<td>Farm worker</td>
<td>School</td>
<td>0</td>
<td>0</td>
<td>23rd child</td>
<td>23rd child in family</td>
<td>England</td>
<td>0lb</td>
</tr>
<tr>
<td>Color</td>
<td>Edema</td>
<td>Surface</td>
<td>Epithelial</td>
<td>Viscous</td>
<td>Consistency</td>
<td>E. coli</td>
<td>Starch</td>
<td>Yeast</td>
<td>Leucocytes</td>
<td>Plato</td>
<td>Oxyphilic</td>
<td>Hyaline</td>
<td>Necrosis</td>
<td>Congestion</td>
<td>Lesion</td>
</tr>
<tr>
<td>-------</td>
<td>-------</td>
<td>---------</td>
<td>------------</td>
<td>---------</td>
<td>-------------</td>
<td>--------</td>
<td>------</td>
<td>------</td>
<td>------------</td>
<td>-------</td>
<td>----------</td>
<td>--------</td>
<td>---------</td>
<td>------------</td>
<td>-------</td>
</tr>
<tr>
<td>green</td>
<td>none</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
</tr>
<tr>
<td>white</td>
<td>none</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
</tr>
<tr>
<td>brown</td>
<td>none</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
</tr>
<tr>
<td>red</td>
<td>none</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
</tr>
<tr>
<td>yellow</td>
<td>none</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
</tr>
<tr>
<td>blue</td>
<td>none</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
</tr>
<tr>
<td>purple</td>
<td>none</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
</tr>
<tr>
<td>brown</td>
<td>none</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
</tr>
<tr>
<td>gray</td>
<td>none</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
</tr>
<tr>
<td>orange</td>
<td>none</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
</tr>
<tr>
<td>pink</td>
<td>none</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
</tr>
<tr>
<td>black</td>
<td>none</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
</tr>
<tr>
<td>Name</td>
<td>Date of Birth</td>
<td>Sex</td>
<td>Race</td>
<td>Age</td>
<td>Height</td>
<td>Weight</td>
<td>Condition</td>
<td>Temperature</td>
<td>Pulse</td>
<td>Respiration</td>
<td>Urine</td>
<td>Bowel Movement</td>
<td>Comments</td>
<td></td>
<td></td>
</tr>
<tr>
<td>---------</td>
<td>---------------</td>
<td>-----</td>
<td>------</td>
<td>-----</td>
<td>--------</td>
<td>--------</td>
<td>-----------</td>
<td>-------------</td>
<td>-------</td>
<td>-------------</td>
<td>-------</td>
<td>----------------</td>
<td>----------</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Casey</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Carter</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>L. A.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>B. M.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>H. W.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>J. B.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>L. M.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>J. H.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>J. L.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>L. N.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M. L.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>J. A.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>J. R.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>J. W.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>J. D.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>J. H.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>J. W.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>J. D.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>J. H.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>J. W.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>J. D.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>J. H.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>J. W.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>J. D.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>