Acute Liver Atrophy

A review of the literature since 1876, including the clinical histories of cases not hitherto published, and a histological report on the condition of the liver in these cases and of the kidney in two, illustrated by a series of drawings.

by R. C. Buist, M.D., C.M.
I wish to express my greatest indebtedness to my professional friends and former teachers for the ready generosity which has placed at my disposal a wealth of material in my hands: to Prof. Simpson and Dr. Talfourd Evans for permission to use their case reports; to Professor Barrett and Wm. Russell for Pathological Reports; and to Dr. Stalker, to Dr. Mackie Whyte and to Professor Paterson for case notes and the permission to use the material in the Anatomical Museum at University College Dundee.

30th April 1894

R. C. Brist.
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<td>Naked eye view of section of liver</td>
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<td>2</td>
<td>Microscopic drawing. Liver I - yellow area</td>
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<td>Antchek-Couge, Gaz. Medicaele 114 et seq.</td>
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<td>Munchin, Diseases of Liver</td>
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<td>Tommari, St. Menagui 19, 330</td>
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<td>Adler, Wien. med. Woch. 51</td>
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<td>Thomas, N. Carol. med. T. 2, 233</td>
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<td>Senator, Wien. med. Forsch. 170, Central., med. Weiis 1878, 735</td>
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<td>Thinefelder, V. Klemanski hands. TF</td>
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"Unimportant" Richardo Thirich.
Bames. Clinical report of amniocentesis pregnancy
B. Sept. 1, 1927
1879
Argumoz
Egan
Mayer
Mack
Meier
Schenk
Sonntag
Talanum
West
West

1880
Brockhassfeld
Callonworth
Ferenczki
deg.
Jones
Loomis
Ludz
Mathieu
Pecceini
Roy
Scholz
Ankuen

Case of a case of heart disease.

24. pregnant.
1. death, recovery.

Review of recent literature.
Syphilis.

Lancur dyspepsia as many diseases.
22 F. prep. 8 mo. severe jaundice
3° day abortion. 6° improvement. Harvey

Anemia atrophy identical with Schepens

24 F. W. preg. 8 mo. they lose
walk, 9° day death.

Two males. death.

Microsporum nigells.

Histology of Schepens fundus.
1882
Kreischmann
Franz Drus. Breslau "Infall.
Kubanov
Yozhenco Med. T. 1462. 2. 67
M. Dewall
J. Nut. Sci. 27. 541
Saltowski
Koch Arch. 88. 384
Nasharch. Blud. 1738
1883
Cayley
B. M. T. 1. 628
Chew
M. York Med. Rec. 24. 369
Pritchard 2.
Cove
Lancet 2. 26
Barneselli
Gio. Milan Med. Soc. 5. 906
Goodhart
Grothe
Grafth. Mittheil. a Bader 37. 19
Harley
Diseases of Skin 396. 480
Madders
Ber. Rudolph Mayr. 343

Krueisler mann
(Wein. med. 31. 1885. 3. 284)

Harvey
Phil. Soc. Med. France 5. 13
Komm
Knie Arch. 91. 384
Rodwell
Viecheb. 414. 1423

Wheatley
Practical Pathology (2nd ed. 1888. 3rd ed. 1891)
Wagner
Krichhoff Arch. 1884. 2. 202

Chemical anatomy of liver.

v. Liebzoegh. 1896

In conjunction with carcinoma of liver spleen.

27 7. W. pregnant 9 mos. 3rd day

Wealthy still lived 90th day death
1884
Bowen
Arch. Med. New York. 11. 175.

Carnigton
Patt. Anat. 36. 221.

Chrostek

Greaves

Ihedenius

Rickert & Thiersch 2.

Ida Novo
Russ. med. 251. 274.

And Med Rec. 1885. 143.

Sutpin
Natural. 313. 334.

And Med Rec. 1885. 143.

(Where many Russian cases are cited)

Izukui

Krader
Ber. Rudolph Stift. Wiss. 386.


Kruzer

Posspilov

Vorontzoff & Thiersch 2.

Smith
B. Med. 1. 104.

Sackung
B. Med. 1. 358.

Schatz

Toumni
Lanceet. 1. 606.

Toumni
Lanceet. 1. 606.

Czerekfelder
Lanceet. 2. 191

diezler
1886.

Houre (2c) Ausl. Arch. T. VIII 466.

Johanns A M J. 2. 1081


Elsner Ausl. Med. Jahrg. VI. 224

Lorenz W. A. Beobachtung d. 12. prn.


Ponson. Zur Erk. d. a. Ä. München 1886


Abelsold. Ber. u. Arbeiten III
1885

De Rensi

Eichhorn

Dickler

Kahler

le Ray

March

Sato

1886

Bohnert et al.

Berg, Truex

Cornil

Glaser

Hirschberg

Hirschbein

Lethke

Eldridge

Podorozhnik

Fischel

Registrar: five years from liver cell

Regenerative tissues from liver cells

Pilz ducl.
Barr. med. Virrs (Mitad) 84, 3-46.
Mader, Bar. d. K. Rat. 357.
1887
Bloeden Gruvali

Lauchmann

Orth.

1888

Andr. Birch.

Marting.

Dowre

Rao

Röhmanni

1889

Siegenbeck Tidv. v. Genech.

Bretton

Buex

Coats

Cred

Köle

Kroed

Kroek

Kmit

Kroek

Kroek

Krumpeck

Krumpeck

Krumpeck

Kroek

Kroek

Kroek

Phosphorus poisoning: liver small, yellowish.

Chemical idiosyncrasy.

Cultivated expansions.

Recovery.
94/ Rubenkov 1. 192. 259. 415. Surinov

95/ Kanitzti. Die

96/ Rischiti. Ann. Rev. of Medic. 1938.194

95/ Lerner 1. 612. Lenzmayer
Pres. 1. 711. Williams (Mecking)
Stock. Cent. 1. 492. (453)
Hebbr. Dtsch. med. 8/1

97/ Richlin. Barondereh W. 25/7. 453
Rainer. Dtsch. Tiefenep (85)
Lublitzer

98/ Meder. Liepm. Brit. XVII. 142-205
Marghaird

97/ Indlau. SMR. 27. 2. Schapmerry
Kulldorff. Med. Rev. 1935. 5/10 (Am 5/100 Menge 10.000)
Since Morgagni's well-known description in his chapter 'de loco' of the case of his saline patient among priests who became pronounced after some mental perturbation. Insanity was

limited and developed in succession, delirium, apathy, delirium convulsions, coma, and whose liver was found 'flaccida et subpallidum v巡ans.' Many cases have been recorded of a similar kind, and in 1845

Brock found that in one case the liver structure was greatly altered by the disintegration of the cells, and

Two years later Handfield Jones found that the kidneys were the subject of exactly analogous changes to the liver and formulated the doctrine that we have
in which it is to deal with a general morbid process which manifests its presence in liver and kidneys alike. The literature published on this subject has from time to time been collated and summarized. Taveras, Budd, Osanna, Leber and Jund, successively made such summaries, and up to the year 1876 exhaustive collections have been made by Thiesfelder and Still more by Martin and Begbie. Since that time many new cases have been made by Rossi. Mattei and others, but no complete summary, and the occurrence of arsenic pneumonia in Dundee during the past winter seems a suitable occasion for the undertaking.
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<tr>
<td>1799</td>
<td>74</td>
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<tr>
<td>1800</td>
<td>75</td>
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</table>

**Note:** The table above is a partial representation of the data. The full table spans from 1726 to 1800.
Almost all writers are agreed that acute liver atrophy is a rare disease, yet the tendency of recent evidence is to show that it occurs much more frequently than had at first been thought possible. Legg saw one case only at St. Bartholomew's Hospital in nine years, Munich, at London Fever Hospital, once in 3000 patients in 84 years, and in 20 years at Grupio Hospital only 8 autopsies are noted with this condition. Henoch has seen only three cases in children. At Vienna General Hospital (2000 beds) I find 23 cases in 21,305 autopsies or rather more than 1 case in 1000 of fatal illness.
While Martinez says that at Iowa they had in the civil hospital 58 cases in 10 years, and that of these 11 recovered. As I haven't yet had access to Martinez's original article I don't know how many autopsies were made so that it is impossible to decide how far we are dealing with the same morbid condition. The clinical picture alone is inadequate with severe mental symptoms and hallucinations is not enough to justify the title we now use even though it is aptly described by critics since it is not the disease we formerly thought.
History and Autopsy (so far as obtainable) in

cases of Acute Liver Atrophy not yet published.
27 July 1886

**Pregnant 6 mo**

No alcoholics. Healthy child.

Hand in fitting first pregnancy, too young ago. Child alive well.

<table>
<thead>
<tr>
<th>Date</th>
<th>Event</th>
</tr>
</thead>
<tbody>
<tr>
<td>January 9</td>
<td>Pregnancy first time noted, very vomiting, then fairly well</td>
</tr>
<tr>
<td></td>
<td>Trenches had pain in upper part of abdomen, no vomiting</td>
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<tr>
<td></td>
<td>Fundus 200 red and white, urine like foamy, stools like that.</td>
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<tr>
<td></td>
<td>P. 1000 T. normal. No signs of fever</td>
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<tr>
<td>10</td>
<td>8am: Lab. pulse 105, in 15min fever broken off. Ruptured membrane rupture, no hypodermically.</td>
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<tr>
<td></td>
<td>Pain brighter. T. 38. Fever, vomiting, no nausea, no menses</td>
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<tr>
<td></td>
<td>9am: Baby described, no fever, no nausea, no menses, baby alive</td>
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<tr>
<td></td>
<td>9:30am: Baby delivered, vomiting came away easily after vomiting</td>
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<td></td>
<td>8pm: Haemorrhage amenable to, no fever</td>
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<tr>
<td></td>
<td>Fever broken off, no nausea, no menses</td>
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<tr>
<td></td>
<td>11:30am: Baby very slow, patient, thin</td>
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<td></td>
<td>1pm: Vomiting, occasional delirium</td>
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<tr>
<td></td>
<td>6:30pm: Menses, no fever, baby quiet, weak</td>
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<tr>
<td></td>
<td>9pm: Urine voided,ayriq in 4 tsp. No menses, while urine is drawn off. P. 156</td>
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<tr>
<td>12</td>
<td>10:15am: Baby asleep, rolls head from side to side. Large brown, amputated</td>
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<td></td>
<td>Baby's half-finger prints moderately wide</td>
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<tr>
<td></td>
<td>3:00pm: Local pulse</td>
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<td></td>
<td>6pm: Hand in fitting, vomiting severe</td>
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<td></td>
<td>No retention of urine</td>
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</table>
|        | 8pm: Baby
1889


September

25 Admitted for delivery. Suffering from acute specific rheumatic.

27 Labor. First Stage. 26 hours.

Second - 1½ h.

Third - 10 minutes.

October

1st Admitted.

3rd Admitted.

Tonic. Acid.

Milk. Albumen.

Blood. Protein.
<table>
<thead>
<tr>
<th>Day of Month</th>
<th>Pulse</th>
<th>Resp.</th>
<th>Motions</th>
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<td>12</td>
<td>180</td>
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</table>

**Remarks:**

November 12

Much worse. Barely conscious.

Eyes half-closed, occasionally raised.

Tongue stuck out. Wrinkles dilated.

Corneal reflex present.

Eyes show occasional movement, like vertical, suprapupillary, occasionally move laterally.

Sweat on the side of the face.

Cheeks are dusky red.

Skin moist on front of body, but, but not so fluorescent as formerly.

Rim of the mouth half-open.

Drooping of the corners of the mouth on the tongue, teeth, lips.

Breathing 36 per minute, rapid, irregular, slightly labored.

The chest rises and falls, no regularity, no traction, no Stridor.

Breathing is sometimes stertorous, and there is an occasional expiratory grunt.

P. 100 soft, moderately strong, regular.

Flexes arm till passes in bed.

Abdomen soft, not tender. Slightly palpated.

No sign of pain on pressure.

Induration 2 in. Median along right hip.

Lochia entirely absent.

Pelvic like a bag only occasionally.

Difficulty in swallowing, an irritable appetite.

Complaint. Rolls head from side to side.

Swallows with difficulty.

Sometimes there is a general muscular spasm in one or more of the limbs, occasionally the forehead muscles jerking.

Inhalation of ether, shortly before death.
<table>
<thead>
<tr>
<th>Simpson</th>
<th>1890</th>
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<td>J</td>
<td>Dec</td>
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</table>

Died before care could be taken.

Post Simpson is at present abroad so that further information could not be obtained.
Spleen 14 oz. Slight capsule wrinkled.


Heart. Slight early inflammation. Deposition of annelidous corpuscles on pericardium.


Liver. 27/4 oz. Slight edema.


Skeletal muscles, livers small, markedly atrophic.

Anterior surface yellow, where it is possible to see the pericardium. The livers, the center dark, dirty. Med. Stomach. Slight hemorrhage into mucous membrane.

Anterior, office, bone.

Auditory. Slight depression. Content, dirty, brown-green, semifluid.

The pleural exudate are removed, suet-like and by request sent to the Physician in Charge.
1893

November 17

1813 well nourished. Syphilis.

He had jaundice 5 weeks.

Jaundice deep. There is a scar on the trunk about 2 cm. copper colour. Slight jaundice. Some slighty yellow spots.

Haemoglobin 80%. Erythrocytes normal. Fluids enlarged. R. Urine Specific.

22. Varicella this morning. Stupor Delirium.

23. Torsos, very flushed. Talked incoherently.

P. 96. Erythrocytes midline 3 in.

R. Nipple line under 4 mm.

Franzoni line above carotid bifurcation.

Right temporomandibular joint enlargement.

Some indolent abscess in right knee.

R. Elbow joint. Takes milk fairly.


Unconscious. Ventral, incoherently.

Stool not bile stained. Throat.

Discoloration. Midline 2 in.

Nipple 4 line under 4 in.

Knee 12 enlarged.

Mark thrombosis bilaterally. Decadous.

Swollen.

Sineoedema about the knees.

25. Coma. Died at 3 am.

Uterus healthy, multiparous.

Lumps, deformed. One fixed in inner

Trunk flabby, small.

Considerable Ascites

Intestines seemed to contain only

pale mucous.

Kidneys not enlarged. Light stained

Spleen enlarged flabby

Liver 16 oz.

Flabby, capsule wrinkled

Dull brown color with orange

yellow patches.

Arteries thick, taut.

Skin dry, flabby.
<table>
<thead>
<tr>
<th>Date</th>
<th>Entry</th>
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<tbody>
<tr>
<td>1893</td>
<td>Fall well-formed, well-developed growth. Nephritis, no history of syphilis.</td>
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<tr>
<td>6 Nov</td>
<td>Not feeling well. As if he had caught cold. Anorexia, constipation.</td>
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<td>8 Nov</td>
<td>Bowels moved freely after a purge. Today found slight Sneezing. No pain. Stools light. Unusually dark. To have blue pill at night. Seidled in the morning.</td>
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<tr>
<td>17 Nov</td>
<td>Today, haematuria (dark coffee-ground material)</td>
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<td>19 Nov</td>
<td>Has had attacks of neuralgia. He described it as shot from the back, down the arm, to the head. He could not sleep.</td>
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<tr>
<td>20 Nov</td>
<td>7.30 a.m. Face flushed. Pulse 90. 8.20 a.m. with shortness of inspiration. Very restless but quiet in unconscious. Every few or five minutes he has a spasm in which the back legs are forcibly extended. Just as he is in a curve with one leg forward. At the end of the spasm the eyeballs become fixed, the hands clenched. The elbows suddenly flexed. Sometimes they are moved. His spasm is more marked on the left. He spasm lasts about 15-20 sec, and the inspiration is at the rate of 80 per min. Then the limbs become stiff. Sudden rise of inspiration. From there the spasm of the abdomen muscles. Jolt, full diastole. Called the spasm Tied at night.</td>
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<td>The last weighed 26 1/2 lbs.</td>
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1894

Theround drunken disinhibited.

1893 Nov. 28

Admitted as dyspeptic, suffering from secondary digestive, with ulcer of Valva of Hydrostomato.

Jan 20

Discharged as out patient, to take specific mixture (Dr. Dc 7 % &c)

(Rec.) She appears rather stupid and things carefully told to go to the Hospital at certain times for her medicine. The comes up to the wards late in the evening, so

She has been keeping fairly well except that she has complained of pain in left side after food.

She has not vomited, but has been retching her food and has been constipated for three weeks.

February 9

Was quite well when her husband went to work in the morning. In the evening he found her sitting by the fireplace, with no fire, her hair hanging, she continued to speak.
1894 February 22

5.30 pm. Feathers still in the same position. On my attempting to lift her head, she held it stiffly down and with very imperfect articulation tried to say "Nanna" at last she then proceeded to protrude her tongue, but she repeated this to every question after wards.

She seemed to understand when asked if she could go to bed, but was quite incapable of helping to remove her clothing or even to lift her feet out of the bed without resistance but was extremely giddy. Her whole demeanour depicted that of a half drunken person. When asked her age she said "Nine o'clock" and this was the only answer I could get to my repeated questions. She had very cold, pale cheeks and as far as could be seen by very poor lamplight the skin had the mottled appearance of imperfect circulation + cold.

Facial expression could not be made out. Pupil is normal. She seemed to be painful on pressure over the left side of the Epi-gastricus, but perineum gave no pain.

Disinclination declined normal.

29

This morning by partial Draught,
sweating was quite visible. She was
unconscious and did not object to being
examined. Quiet efforts did
not take her eyes from me. This
morning, she could tell her husband
some things at first. She did not
talk to her husband nor her wife.
She kept her eyes half shut until noon.
She took food easily from me.
Disinclination still normal.
Sent to hospital accent.

Spn 1. Adm, unconscious.
Could not be moved. Pupil, eyes

Spn 2. Drifting, occasional snoring.
Assistance: unconscious. No convulsive reflex.

Spn 3. Has slightly recovered conscious.
Indicates L. side as seat of pain.
After her waketh had done she asked
more "did you mother have not
spoken about?"

24

Quiet during the night. Can
swallow fluids, but not the drug.
Unna observed d. 29.03. drawn
by Cathelin.

Spn. (yesterday and today)
P. 72. R. 12. Parly cyanic, her face
with all cheeks relaxed, her eyes
open and a perfectly mididifferent
expression on her face.

Pupil: Pupil right tender
Slightly dilated about 4 mm.
Bill Marie I. Leas. Sister Attendant

The, thin facial yellow
February 24

The jaundice is yellow green over the trunk but is absent in lines of Creasy (pressure Osages) on chest & abdomen.

Liver dulness & bile 3/4 xi.
Jaw tenderness 4 xii. Rectipenis 4 1/4 xi.

The pencil marks above and below.

The jaundice became bright yellow.

Attempts to excite Jaques' Browns of bile on a line of green bile appearing in a few seconds lasting 5 or 6 seconds. This is measured on the chest less in the abdomen.

The right kidney seems a little displaced and can be palpated manually. On top of the kidney, there is pain palpation of the kidney has been known even by chance of surgical resection. The right side in duration of pain is discomfort. Yesterday there was a little nausea and pain in the abdomen occasionally drawing up her R leg as if in pain. There is none of this today.

She can only swallow food except in very small quantities.

To have removed bile, chest and 1/4 and if need be a glass saline

Dr. veterinary to remove jaundice. One bottle? The next day after noon she had no pain.

She needed the catheter.
February 25

Diurethumes in clupea 3 3/4 Parasital 4 1/4 Ripple 4 1/2. The net is less intensely dull all over.

She is in the same position. Muscles flaccid.

Her face their line is hardly traced by scratching, clamping a white ink line for about 30 seconds, the line disappearing. The colour of the abdomen is almost canary yellow.

P Redux 4-5 mm. Stripsy density.

Breathing slow, regular, somewhat dreaming.

To have P Redux morning.

26 (Same as present at the visit)


The paroxysm is more distinctly marked on the legs.

Mucus well-marked Rattle Clamro for-Homme doge jerked.

P 104. R 13. Electrume 1 long

Perpetually. Some hiccup.

The nostrils half open on her on the left hippo.

She has mumbled a good deal and three times in the past twenty four hours she has given a loud scream acci junie.

Their taking movements fairly well.
February 27

Nebulized one. No haemorrhage.

Vic. Early this morning

Temperature subnormal

<table>
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<tr>
<th>Date</th>
<th>Temperature</th>
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<td>97</td>
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<td>25</td>
<td>97</td>
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<td>26</td>
<td>97.8</td>
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<td>27</td>
<td>97.6</td>
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</table>

Nebulized symptoms 5 days.

Nebulizer 8 days.
Stalker
Dundee Reg. Inf.
28 hours pm.

Well-marked jaundice all over.
Body rather sparse.
Lymph nodes 2 in. at nipple line.
Rigidity slight.
Hypertrophy marked.

Head: Pericranium turgid and irides green.

Throat: R. lung 21 oz.
L. lung 15 oz.

Recent adhesion of L. lung.

Rupture of interna of right lobe of liver.

Rupture of liver 26½ oz. Haemorrhage.

Caput has the look of advanced jaundice: the lobular markings being light grey lines.
The general colour is dark purple with dull yellow patches in the lobules. The thickness is much reduced.

In section there are bright orange yellow patches, at a dark red ground.

The latter colour drawn up
pines away accurate representation of the original colour.

The lobulation is not so distinct as usual. Especially in the red point, intra lobular bile visible.

The gall bladder contained a little dark green bile, highly viscous.
The ducts are patent.

Kidney R 5½ oz 7 arteri pale 1 5½ oz 3

Spleen 6 ¾ oz. Apparently normal.

The peritoneum contains some slight yellow fluid.

The uterus is unimpregnated and has some cysts in it.

In the fallopian tubes there is apparently no salpingitis. There are at two or three points. Small purulent collections.

Ovaries showed several follicular cysts with haemorrhagic exudation.

The liver only was preserved in formaldehyde.

It was unfortunately mistaken.
The drawing was made from a fresh nicotine. Four days later the liver having been in spirit.
Note.

The liver third will preserved at the autopsy were destined for museum specimens, without any idea of histological investigation. This is the reason why the reports on the condition of this organ are lacking.
Microscopic Examination of the liver
in Three Cases of Acute Liver Atrophy

Sections by Fresnius microtome.
The piece from which the sections were made was at the time of a distinctly green-yellow colour. Logwood stain was not nearly so effective as Perucarmine. The most marked character of the section seen under the low power is that the peripheral part of the lamellae are divided by distinct bands of fibrous tissue which have stained brilliantly with carmine. These are most conspicuous about the central spaces, but extend along the peripheral lines between successive spaces. The central vein is frequently surrounded by similar fibres and occasionally distinct bands pass between the inner intra-laminar sinus. As a rule the strands of stroma passing through the lamellae are unusually fibrous.
and have an unusually dotted appearance. There are at

parts clouds of fine black and yellow granules on the surface

of the section and occasionally larger pigment masses.

The high power shows that the dotted appearance of

the stroma strands is due to their being faced by

small granular protoplasmic masses. The distribution

of pigment granules is not at all uniform. At one

part scarcely one can be seen, at another each bundle

is covered with a multitude which is rather more

densely packed around the vascular spaces. These

is at points throughout the section some small-celled

infiltration, and at one part there was distinct
haemorrhage. The condition of the cell remain, are various. In other arachnial columnar cells, but occasionally a single granular cell may be seen, though the nucleus is invisible, generally the cells are represented by small granular masses of definite outline, and occasionally the stroma is pervaded by an indefinite granular material. Scarcely any traces of the capillaries are visible.
Under the low power, the section seems at first to consist only of tissue more extensive than normal so that empty spaces except the lumina of blood vessels are not readily seen. The stroma looks much more fibrous than normal especially about the vessels and at the parts corresponding to the bronchial peripheries. The strands of the stroma are here and there intermixed by small protoelastic masses, and here the mesh contains alveolar cells. At first the section is beset with fine black points apparently very small pigment granules. The bile ducts are not seen at all.

Under the high power the section is seen to be crossed by well-marked fibrous strands, and around some
of the vascular spaces this tissue seems for some distance

to consist of fine wavy fibres. At other parts the vessels

are surrounded by tissue with well-marked but not dense

infiltration of small round cells, and this sometimes

extends along the intervening space between two series

of vessels. The remnants of liver cells are found partly

small masses adherent to the strands of stroma, but at

points, amidst isolated round granular pigment cells,

can be seen. The remnants of bile ducts in this section

are represented by remnants only, a few cells being

left in the space, very seldom enough to complete the

circumference of the duct; at points a series of these
In close succession may be found at a cellular boundary
but they have all this degenerated disintegrated
character. The tissue round some of the vessels is
stained with innumerable very minute black particles
which are occasionally collected into small masses.
Third Section

In a third section from this liver the changes were found similar in kind but varying in degree from those in the other parts. The stroma generally had a fibrous character, but there was not the massing of fibrous tissue around the vessels and at the periphery of the lobules found so marked in the yellow section. The greater number of small dark granules of pigment were very much more numerous than in either of the other sections. The condition of the liver cells and the bile ducts was one of similar disintegration.
Diagram I

Staining with osmic acid does not make the sections nearly so dark as those of either of the other livers.

Under the microscope this is seen to be due to the lack of cells which are very scanty indeed. Where seen they contain a few black points, but the cells are mostly very small & in very rare instances is an approach to columnar arrangement found. Where bile duct epithelium remains it also has stained with the osmic acid but it is only found at rare points.
liver II

Yellow.

The part from which this was taken was soft and of a distinctly yellow color. The liver had been in strong spirit fully three months. Dogwood was found to be the best drug. Under the low power, the tissues immediately under the capsule seem universally over-laid by a dense small-celled infiltration. This is exaggerated at the peripheral part of the lobules but is general all over the surface. On passing further into the tissue this infiltration is found but becomes gradually more and more confined to the interlobular tissue, and ultimately to the tissue radiating immediately from the interlobular vessels. 2. The next most noticeable feature in the
great congregation of sections of small bile ducts lined
with epithelium having distinctly stained nuclei. These
are found in close serico in the interlobular tissue, more
especially under the capsule, and the sections are
mostly circular but occasionally small branched
sections may be seen. (3) At several points in the section
are found masses of lightly stained tissue, the colorless
prominent in appearance but having marked
refractility at its margins and in bands across
the surface, and being closely studded with deeply
stained dotted short lines, the whole apparently
resembling fibrous tissue with the nuclei of the cells
Sclerotic fibre masses are sections of vessels of various sizes with thickened walls and in the case of the smaller vessels, the lumen apparently filled with retained points. The long nuclei of the cells are distinctly seen surrounding these vessels and are distinctly seen in acened group (5). The condition of the lobules in this tissue is very uncommon. Close to the capsule the details cannot be made out and the tissue is dense and seems to consist of fine fibrous tissue containing cells as its mesh. Sometimes distinct liver cells with a distinct or distinct atrophic of the nucleus. Further in we
found characteristic lobules with structure fine
open than normal and containing fragmentary
columns of liver cells and at one point at the
inner edge of the section we found lobules where
the normal structure seems fairly intact. The
liver cells in the deeper part evidently show
district nuclei. The lobules though normal in
structure show indications of being compressed
being sometimes distorted, occasionally narrowed
and as a rule the normal columns do not extend through
a whole lobule.
There is besides the free and unioveal infiltration of small cells a larger incursion of fibrous tissue generally distributed and apparently consisting largely of cells having elongated nuclei. These are liver cells and their remains usually isolated but occasionally in groups of apparently detached from columns, enclosed in the meshes. These have often distinct nuclei and their protoplasm is only slightly granular. (2) The bile ducts have the nuclei of their epithelium unpermitted and long series are sometimes traceable in evident connection; occasionally a duct with double series of lateral branches. The appearance under the low
layer II

There had evidently been interference by the cellular infiltration of the cells of origin. Some are deeply as the epithelial nuclei and are of nearly the same size and sometimes even double line resembling the trace of a bile duct. There is no trace of karyokinesis in any of the epithelial cells.

(3) The bile homologous masses are fibromyxoid. Disrupted nuclei are scattered through the tissue and at the edges generally aggregated in several lines. These masses are interconnected with the blood vessels, some of which are embedded in the masses of the blood vessels.

(4) The walls of several series of cells with close-packed, elongated nuclei and contain ni in some cases the
Endothelial coat, with some apparent reduction of the cells. 5. The condition of the lobules varies from nearly complete preservation to complete breakdown and disintegration of the columns on one hand and on the other to marked infiltration with cells and invasion of the alveolar lobule with fibro-metastasis. The cells show a variety of size, occasional large round cells, clonally and with nucleo-cytoplasmic axis in a few cases the cells are small and partly represented by granular debris and sometimes entire columns of perfect cells are found. In these facts and in many scattered cells...
The nuclei are quite distinct and at no point have I seen in this preparation any evidence of nuclear division.
This was much tougher and firmer than the other part. Under the low power its features were similar to those of the other section, but the small celled infiltration was universal, denser at the interlobular boundaries, and there was at no point in the section an approach to normal tissue structure; the whole tissue resembled the dense tissue close to the capsule in the other section. The section showed the dense masses of fibrous tissue with elongated nuclei, the same appearance of bile ducts and the dense modification of the walls of the blood vessels.
The section from the yellow area stained with osmic acid shows the distribution of the changes very clearly; the liver cells and duct epithelium being a dull red stained deeply while the fibrous tissue and infiltrating cells are much lighter. In the dense tissue under the capsule we found few remainders of the hepatic cells. In the best preserved lobules the liver cells are very granular and many of the granules being darkly stained. In parts where the tissue is more open and we can see the bands of stroma with few remains of liver cells only, the stroma looks red as all fibrous but in more atack of light and dark granular points. Apart especially at the peripolar (not in all tissue) we find distinct fibres.
Yellow area. The part from which this was taken was mainly yellow and soft but immediately under the capsule the substance was denser and redder and there was a band of reddish tone running nearly parallel to the surface of the liver and a short distance in. On microscopic examination the conditions found were, under the low power of the dense part under the capsule looked dense, yellow, showed a lobular structure with large numbers of bile capillaries intersecting at the peripheral parts of the lobules, which were smaller than in normal liver. In the lobule itself there is a close set mass showing much stroma with
the spaces either occupied by cells or granules so closely that empty spaces are not seen immediately adjacent to this is a clear part in which stroma with almost empty mesh but preserving its lobular arrangement is clearly seen. the finer dark bands of different character from the other as it consist of somewhat separated of fairly well preserved columns or fragments of columns of hist cells which seem granular and yellow, at the border at this area the columns disappear and more distinct disintegrated and isolated cells are seen. Proceeding inwards we find mostly empty stroma, with here and there scanty remnants.
of liver cells which had formerly constituted a lobe, here and there an isolated mass of yellow pigment and in fact a traversing of the mass by indefinite broad bands of fine fibrous tissue. The size of the stromal lobe in the interior is so small as that underneath the capsule varies there so great a number of sections of bile capillaries.
Hist. power (250). In the capsule and a small distance into the tissue below are a few scattered round cells about the size of white blood corpuscles, staining well with haematoxylin. The dense tissue itself resolves into a mass of fibrous stroma entangled in thickness but not dense.

In the meshes formed are small liver cells mostly isolated, oval, finely granular and with nucleus invisible. Droplets of fat and the space around the cells in the meshes of the stroma is filled with small round clear bodies like red blood corpuscles, close packed and representing small foci of haemorrhage. At the periphery of the lobule are seen many sections of
tile capillary with the epithelial nuclei staining well. These sections are in many cases traceably sections of the same ducts at repeated points and occasionally a duct can be found branched and convoluted crossed here and there by bands of fibrous tissue. (2) This passes rapidly to a small band where there is only stroma, not stained, with prismatic cells scattered of very small size in its mesh, sometimes one or in the substance of the stromal bands, (3) after this we again find the stroma's fibrous cells more frequent and gradually arranged into columns till in the midst of the minor dark band we find
fairly well preserved columns of cells, more widely separated than normal. The cells are mostly round or oval and here and there the nucleus is distinct. They contain fine points of dark brown pigment and yellow pigment masses irregular shape and varying size are found lying amongst the cells. The cells are best preserved towards the centre of the lobules and at the periphery there is an increase in the amount of fibrous tissue, amounting almost to bands here and there, but not contracted or dense.

After this, the tissue again open and through the lobular arrangement of the thymus is preserved.
More clearly distinctly, the meshes are almost entirely empty here and there, these indications of the cells of alveoli are left in the form of little rows of imperfect cells. Around the portal spaces where they can be seen there is an infiltration of small round cells staining well with haematoxylin and the fibrous tissue is increased in amount.
The sections were made from a typically red area and
include the capsule. During the cutting it was noticed
that an especially bright red line was exposed looking
like a small blood vessel filled with clot, and that the
substance in the immediate neighborhood was even
redder than the surrounding tissue. On the sections being
floated the darkest red part fell out of the sections in fragments
like evidently blood clot. Microscopic examination showed
that the space in which they were contained had at part the
main vascular wall and that in the surrounding
tissue there were masses of red corpuscles. Several
haemorrhagic
smaller vessel were visible and branches of the portal
The general appearance of the section is very similar to that in the section from the yellow area. Under the capsule is a dense area in which the stroma is visible with its spaces full of granular cells of varying size sometimes in groups of two or three closely packed and disconnected cells of small size with the nucleus not visible. The same apparent multiplicity of the capillaries is evident and also their branched and convoluted nature. At first the stroma is well seen with its meshes almost devoid of cells, having almost protoplasmic fragments adhering to its strands with here and there small pigment masses.
strands of the stroma are not distinctly fibrofilitated, but rather granular and there is here also one
increased fibrometrising around the portal vessels.

The lobular arrangement is much less distinct
and there is no hint whatever the cells of a
lobe are fairly preserved. Though at the inner
edge of the section a characteristic lobe with
fragmentary columns gives a clear indication
of transition.
Liver III

An unusual yellow module. On the posterior surface of the Quadrate lobe there was an isolated yellow module, about the size of a small pea, projecting somewhat above the surrounding red substance. On cutting sections there was seen blood in areas with foci of necrosis similar to that seen on cutting the sections from the red area. Under the microscope it was seen that the yellow module consisted of columns of liver cells corresponding generally to a single lobe. The columns were less close and compact than normal but the cells of which they consisted were almost normal, being only slightly granular and having distinct
Liver III

Nuclei which stained very well with haematoxylin. The nuclei showed no indication of karyokinesis. The surrounding tissue passed almost at once to the most deformed condition seen in the sections of this liver. Stroma somewhat increased, with almost empty meshes containing here and there isolated cell remnants. In this section there was under the capsule small round cell infiltration which rather more marked than in the other parts of this liver and extending somewhat more deeply into the tissue, and the same was found in the irregularly distributed in
The tissue around the portal spaces, when these were also noticed an increase in the amount of fibrous tissue. Throughout the section the strands of the stroma were considerably thickened and the resulting smaller spaces contained frequently contracted and pigmented cells without distinct nuclei. The aggregation and convolution of the bile ducts at the peripheral parts of the lobules were distinctly seen in this section also.
Sections of this liver stained with eosin acid show to a certain extent the changes in the different parts, even more clearly than other sections. In the isolated yellow module which may be taken to represent the best unpaired portion of the tissue the staining is comparatively light. The cells showing only a few dark granules and the tissue these cells surrounding tissue being almost free. In the best preserved lobules of the yellow area section the cells are represented by dark sometimes black masses which are seen to consist of heaps of black granules, which do not vary in size or...
can at no point be described as drops or globules. This condition is that of the cell remnants, where ever they are found. But as the tissue becomes more open and the cells more disintegrated the black granules are scattered through the meshes of the stroma, and in those parts where the cells have been more or less completely removed within a small collection of black granules, arranged somewhat radially around the central vein. These collections become are of very various sizes and in some cases almost none at all are left; the stroma alone being left.
I have examined with great care the condition of the walls of the blood vessels throughout these sections and though here and there vessels were found where the wall seemed thickened in all its coats and in the cases of the smaller branches, narrowing the lumen, I have not been able to satisfy myself that there was apart from the increase in the surrounding fibrous tissue any distinctly abnormal condition. The thickening of the endothelium was in no case so distinct as to merit the term Endarteritis.
Microscopic examination of the Kidney

in Two cases of Acute Liver Atrophy
The most effective stains for these sections were logwood or benzoin with logwood. Subsequent staining the epithelium of the tubule was a clear light yellow, which differed distinctly from the blue tinge of the stroma, and nuclei and nuclei could be traced very clearly.

In the tissue immediately under the capsule the epithelium is in most cases completely present but it is also very degenerate, having large cells with vitreous masses less indistinct, protoplasm finely granular and in many cases no nucleus at all visible. In some many cases the lumen of the is necric the tubule and sometimes the epithelium was reduplicated. The nucleation of the cells varies greatly throughout the section, being in
Some tubules at all parts of the section perfectly preserved even when the tubules are incomplete, in others, the next tubules, being quite invisible. Though the lumen of the tubules is not seen we often find the epithelium detached, forming a round mass at one side of the space within which it is situated. There these be small masses of green or bright yellow colour; these are found to be collections of cells generally with no nucleus visible and containing with cells or around, frequent bundles of green or yellow colour which parte the whole cell substance. As we pass more deeply into the kidney we find that the appearance changes. Many tubules are found in exacte
the same conditions as those in the outer part of the cortex, but in others we find the cells separated by distinct intervals and occasionally in the course of a long branch we find lacunae corresponding to the loss of single cells or cell groups, and in some cases we find the whole Spongiculum gone. At points in the section we find the tubule occupied by a deeply stained mass which, with high power (x435) seems to resolve into a collection of fine points each of which has taken the stain deeply, in some cases these masses have yellow cells apparently permeated by the fine blue granules. It is possible that on further examination they find these to be masses of microorganisms, at present I cannot decide their nature.
Kidney II

More exactly. Throughout the section the glomeruli decent to quite intact. There is at various points in the cortex, especially near the glomeruli, some cell infiltration, and this becomes much more marked as we pass to deeper parts of the section. The stroma decays entirely as it considerably extends, and is well represented in nuclei and in places where the tubules are empty (so that the remaining nuclei do not obscure the picture) we find it the death cell infiltration. The cell infiltration is also quite very dense and is sometimes represented only by a few scattered cells. In some cases it needs very careful examination to determine whether we have to deal with
tubules where the epithelial protoplasm is thinned and
pale leaving practically little but nuclei or acellular
infiltration but it is quite possible to do so and both
conditions are distinctly present.

Conversely, does not stain the section. Very deeply and
we find that in the cells we have a relatively small
number of fine black granules and at other point
small lines of these black granules along the strands of
stroma. The disintegration in this case is then not to
a great extent fatal.
In this case logwood staining was not very effective. Coein or Coein Acid was much more successful. Inlogwood staining, the cell remnants had in the distich yellow colour of time in the other kidney but were tigited a dull blue and almost invisible. In this section I could discover no trace of nucleolus or of cell infiltration. The tubules epithelium allowed the section was very much uninjured but the severe impairment exhibited much more uniformly than the section of the other kidney had shown. It was the exception in this case to find good section of tubule with the epithelium remaining completely. Usually the circle was incomplete or there were lacunae in the course
Kidney I

Of a longitudinal section. In several cases these vessels were found to have the epithelial layer duplicated and thickening of the walls of the blood vessels largely in the adventitia but also by multiplication of the endothelial lining was very common. The cells in a capillary tube were generally found in the condition of separation which was found in some parts of the other kidney. There was no trace of the dark granular masses (carbon plugs) found in the tubules of the other kidney and as already stated there was no cellular infiltration. It was very rare to find any of the masses of pigmented cells or loose pigment described in the other sections.
In almost the most noticeable character of the tissue was the presence of a distribution of dark yellow or almost black or dark green granules of pigment, usually very small. This was found all over but was more marked in the deeper parts of the section where it showed still greater massing near the blood vessels. In the tissue of Bowman's capsule it sometimes formed a distinct ring as it did around some of the larger vessels.

In sustained section it was seen that the most marked accumulation of pigment was at the outer part of the pyramid where it gave the effect of an irregular dark band contrasted with the whiteness of the rest of the tissue.
The section stained with Basic Fuchsin shows that the cells contain many black points, the section as a whole staining much more darkly than that of the other kidney. Moreover in any of the sections of this kidney have been found any fat mass larger than granules, droops or shingles have been completely absent.

The pigmented condition of this kidney is to be compared with the similar condition in the liver section and in both tissues we find considerable accumulation increase in the amount of foci of tissue.
### Condition of the Liver in Three Unpublished Cases

<table>
<thead>
<tr>
<th>Age</th>
<th>Weight</th>
<th>Colour</th>
<th>Consistency</th>
<th>Cells</th>
<th>Fatty Degeneration</th>
<th>Fibrinoid Nuclei</th>
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<td>F.</td>
<td>29</td>
<td>26½</td>
<td>Yellow</td>
<td>Normal</td>
<td>Granules, debris</td>
<td>Irregular</td>
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<tr>
<td>M</td>
<td>15</td>
<td>26½</td>
<td>Yellow</td>
<td>Normal</td>
<td>Granules, debris</td>
<td>Irregular</td>
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<tr>
<td>F.</td>
<td>24</td>
<td>16 lbs</td>
<td>Yellow</td>
<td>Granules, debris</td>
<td>Granules, debris</td>
<td>Irregular</td>
</tr>
</tbody>
</table>

- General excess distribution of pigment granules.
- Marked increase of pigment granules.
- Marked distribution of pigment granules.

### Condition of the Kidneys

<table>
<thead>
<tr>
<th>Age</th>
<th>Weight</th>
<th>Colour</th>
<th>Pyramids</th>
<th>Glomeruli</th>
<th>Cells</th>
<th>Infiltration</th>
<th>Nuclei</th>
<th>Fibroblastic</th>
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</thead>
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<td>F.</td>
<td>29</td>
<td>Pale</td>
<td>Enlarged</td>
<td>Dark</td>
<td>Normal</td>
<td>Granular, large</td>
<td>Present</td>
<td>Marked</td>
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<tr>
<td>M</td>
<td>15</td>
<td></td>
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</tr>
<tr>
<td>F.</td>
<td>24</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Marked distribution of pigment granules</td>
<td>Granular, fatty</td>
<td>Marked</td>
</tr>
</tbody>
</table>

- Marked increase of pigment granules.
Analysis of the Characters in the

cases published since 1876.
Jaundice occurs early in most of the cases recorded under the title of acute atrophy of the liver and icterus. Jaundice is noted as present, and where the record gives the period of its occurrence it seems usually to be an early symptom. Freer, Rosenheim, Holtzweck, all recording histories of young patients describe it as sudden or as present at first and in nine other cases it arose within the first three days of illness. Sixteen cases note it as early and six more arose within the first week. The remaining six cases were the only ones where the note is definite because jaundiced at periods from two to eight weeks from the first day of illness, so that in most cases it is an initial symptom. West, Kraus, Wagner, Schichhardt, and Lafitte record cases where jaundice was absent throughout, but to these
we shall return later.

The duration of this jaundice is exceedingly variable but this is largely due to the fact that acute atrophy is apt to intervene in cases of liver disease of more less prolonged course, and that in these cases it is impossible to separate the jaundice as a symptom of the original disease from that associated with the atrophy. In other cases however the jaundice is only one of the terminal phenomena of more less rapidly fatal denouement. Thus Dranov's case, a young peasant in the eighth month of pregnancy, miscarried a dead foetus after three days illness, and developed jaundice and died next day and in three other cases two days only elapsed between
the occurrence of jaundice and this jaundice. The
most common duration of the period of jaundice seems to be
from two to five weeks, but these figures are only to be intimated
upon as the data for statistic are present only in 13 of the cases.
If the intensity of the jaundice or the reach of the
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Facial aspect
Several of the cases under review present points of interest in respect of the note on facial expression, which have however only loose connections with the character of acute liver atrophy. In Bockhardt's case the facial expression of fear was evidently hidden by the mental condition resulting from the shock that occasioned the illness, while the anxious appearance of Bucy's patient and the painful expression of Tschansko were associated with extreme abdominal tenderness. In the older records of this condition authors frequently insist that even in deep coma pressure on the liver region elicited
change of facial expression involving the facial
muscles, but that is certainly not a universal rule.
Of our present authors Lewitski records this phenomenon
and twelve others mention spastic or hepatic
tenderness, Sir describing it as great, but three others
and to these we must add Hofthealer's case expressly
day there was no tenderness. It is of course bhat the
absence of tenderness was dependent on the degree of the
paralysis of cerebral function but this is not so, as the
absence is noted as in Greves case before this depth of
unconsciousness occurs since.
The changes in the parenchymal dullness of the liver are
when first met with perhaps the most surprising in the
whole history of this condition, and particularly the rapidly
with which it diminishes, and their exact determination is
a matter of some importance, as definite diagnosis cannot
generally be established until diminution of the liver dullness
is manifest. In a considerable proportion of the published
cases, evidence that, extension of the liver dullness is noted
as present at some point time during the period in which the
patient was under observation, and in 80 cases more it is
described as normal. In four cases (Adler, Goddard) the
diminution
absence of subsequent diminution in text, but in the later
Diminution rapid.

I have not found any distinct mention of diminution of the intensity of dulness, manifest Bernet's extent is lessened, as noted in Dr. Stalker's case. In Turner's case (1043) this was noted after the diminution was in progress. See Brit. III 57.

In Dr. Stalker's case, the dulness was diminished by the ninth day. The dulness had almost vanished, and the liver weighed only 31 ounces.

In 24 cases, the term 'rapid diminution of liver dulness' is described. In the cases recorded by McChesney, Breschfeld, Viren, Sunth, Picholle and Biedung, dulness somewhat sharply contrast, as the diminution is noted gradually.
progressive at a line long anterior to the centre of the case, in
McConnell's case at 96 days 3 ni., at 75 days 2 ni.

Spleen.

The condition of the spleen seems to be little characteristic, and
the changes in it are probably doubly dependent on the general
affection of the liver. Strangely to remark and the circulatory
changes due to the affection of the liver. In the 20 cases where
its condition is noted, it is normal in 11, enlarged in 9.

Distention of
Abdomen.

The changes in the liver and spleen are doubtless sometimes
masked by such conditions as distention of the stomach or colon
and ascites, which are met with here in a small proportion
of cases. Translucent ascites is described in five cases, Rothmann
noting that it was transient, and ascites occurred in nine

Meteorism

ascites,
In five patients, the ascites was associated with oedema of the legs, two with oedema of chest and hands and in one with general dropping. In two cases recorded by Smith and Bullig's work, oedema of the legs occurred. In Mackie Whyte's second case must be added to the list of patients with oedema of chest and ascites. It is a curious fact that three of these ascitic patients (Wurt, Wagner, Schickhardt) also had no jaundice, but in this condition the liver tissue differed considerably in them, this is probably a mere coincidence.

As a rule in ordinary jaundice the patient becomes somewhat thinner even though otherwise suffering little noticeable distress, but in ascites it is frequently noted both clinically and post mortem at the autopsy that the face is...
well preserved, sometimes that the patient is stony. Remarkable
contrasts is the continuous rapid evacuation of Smith's patient
a man of 142, in spite of well continued appetite & infest of
food and similar cases are recorded by Hebbel, Röhmann & Baur
by Smith.
Onset

The onset of illness in cases of acute liver atrophy is in many instances little different from that of general and infective diseases. For a period short, sometimes acute (Scholtz, Delge) days, or long (Swavesey, Glaze), the patient suffers from malaise, lassitude, anorexia, feels sick or vomiting, is nauseated, suffers from headache, giddiness or merely notices that his urine is dark. The clinical picture in many cases that of typical gastrointestinal catarrh and when followed by jaundice justifies the diagnosis commonly made of calculous jaundice. On the onset of jaundice the symptoms especially gastric become more exaggerated and in their train follows the whole complex of jaun
Vomiting

Vomiting is one of the most constant symptoms in only four of the cases is it stated to have been absent and it is very frequently an initial or early symptom but frequently also terminal. In some cases as that of Bliding it may persist throughout, rarely it occurs once only as in Dr. Stalfer's case when it was preceded the day before death by slight hiccup. The character of the vomit varies considerably and may change in the course of a single case. Dr. Greer's patient vomited yellow fluid at the end of the illness but in a considerable number of cases the vomit becomes ultimately greenish, coffee-scented or even directly bloody, and sometimes even dirt-scented.
case this is the most important clinical feature.

Hæmatemesis is with cutaneous petechiae + ecchymosis one of the commonest symptoms of the haemorrhagic tendency which is so pronounced in this disease that moment it is required to include 'haemorrhagic' in the title. Such aspect is not neither necessary nor advisable, but it makes well this frequent occurrence of haemorrhage ante mortem; since otherwise by no means this occurs the occurrence of haemorrhage, as it (e.g. Appleyard) once when about clinically it has been found post mortem as it was in Dr. Fbke's case in the Osang and microscopically in the liver. Before death haemorrhage shew itself
in almost all possible varieties. Epistaxis is not uncommon, sometimes so free (Smith) as to require stopping of the nostrils. Blood in the Spatium is recorded by Smith & Mosé, and bleeding from the gums in several cases from which Vaniś must be abstracted as due to menurial stomatitis. In several cases there is a tendency to postpartum haemorrhage though in O'Sullivan the lochia were entirely suppressed; and Duckworth's patient had metrorrhagia on and off as an initial symptom. While Prichard's case was ushered in by amenorrhoea.

Bowels

The condition of the bowels is by no means
Uniform from case to case or even in the individual patient. Constipation sometimes obstinate (see Appendix after two minutes flatus (Goodhart, "purée") oil is frequent but diarrhoea occurs almost as often and sometimes both are successive incidents in the history. The character of stool is equally variable and save for a general statement that the stools are frequently white at the outset and tarry at the end, no rule can be laid down. The absence of bile is by no means constantly present even in very severe cases where the intestinal musculature considerably disturbed.
The occurrence of rigors is noted in the history of eight four cases.

In Fries's syphilitic man, rigors is noted 12 days before the appearance of jaundice, and has probably no connection with the disease we now consider. In Bliehns's case rigors forewarn the terminal facial eruption, in Clibbe; the faintness swelling of the joints, in Grüner's an extraordinary attack of hyperaesthesia of which no evident explanation is given; and in Nörlens case the rigor was the, uncommon, accompaniment of a bilious eczema. In the remaining cases (Sweitzer, Chew, Jenkins) the rigor was an initial symptom occurring with the beginning of jaundice, which at first partly resembled of catarhal character.
The occurrence of mental symptoms in cases of suicide is generally a matter of serious import and in the cases under consideration it is sometimes this circumstance which draws attention to the true and grave nature of the condition. In some cases their appearance is made with almost dramatic point and suddenness. Scholz; 

v. Sparr; with sudden unconsciousness and patient was seized with sudden lethargy, La Biserta; was found unconscious in the morning. Appleby's patient went to bed with severe headache and was found in the morning sleeping andsomathing the furniture and his own case the husband left his wife within the morning and at sight found her sitting with dishevelled hair smoking a pipe.
Striking changes like this however are not the rule. We often a condition so rapidly progressive as this generally is, and in most cases a period within which the mental symptoms are limited to mild delirium, perhaps at night only, or a little disorientation, some indifference to stimulants or slight mental depression. Some degree of mental depression is so common in jaundice that it would be impossible to establish it as an initial symptom of acute liver atrophy. Delirium is commonly the first symptom sometimes only a slight restlessness and in foetures (Glaser, Priesterk, Cullingworth, and Carrington) tremor was the first nervous symptom observed.
Depression. Stupor or even coma is initial in a considerable number of cases almost as great as that in whom more excited states are first found. As anile begins of persons disturbance occurs late. In only 4 of forty seven dying cases arise more than eight days before the issue of the case and in 16 they first appeared on the day preceding death.

In a few reported, Dorschfeld mental symptoms are reported as entirely absent, and in several others even at the end the delusionism was comparatively free but such occurrence is very exceptional. As anile, the mind is deeply affected and in the majority of cases the terminal stage reached early or late is deep coma, with
sensuous & fretfulness, unconscious urination, defecation, and complete insensitivity to pricking. Rasmussen observed
a case where the state was absent
and in cases of Janisovich & Smith it was still deep. Glaser & Tolnay
record cases in which the coma was transient or rather recurrent
and, in this instance less definitely described, seems to be only
the most marked condition of a phenomenon frequently
depicted, transient or remissive in the mental disturbance.
Dr. Jansen, patient from a night in complete and apparently
sudden stupor, could next morning distinguish between
her husband & myself, and when in hospital deemed on
occasion occasions to wake up sufficiently to hear questions.
The occurrence of tremor has been already referred to, and

practically feared.

This and fibrillar twitching are described in a few cases but the

higher degrees of muscular activity are by no means uncommon.

Clonic convulsions are in some cases distinct as that of Carnigia,

the terminal phenomenon and in 13 cases a more or less

marked tonic contraction exists. Sometimes there may be

more rigidity of the neck as in Lapitte's rather exceptional

case of Tonicus as in Standart's case, occasionally we

get distinct attacks of Opisthotonus as in the cases of

Dundie Sheyl and the boy from Spalding Industrial

School, in whom the spasms were more marked on the left side.

In Dr. Sheyl's case it was noted that a few clonic

Tonic twitching

Clonic Convulsion

Tonic Contracture

Reflexes
Jerk could be elicited at each ankle and Duckworth and Raczek also record the occurrence of ankle clonus while Sandbaktner says there was slight exaggeration of skin tendon reflexes, but probably this could be more often elicited if looked for. The absence of plantar reflex on the right side is noted by Lafitte.

Several times in the series, for example incases cases the occurrence of itching is noted and occasionally of urticaria. But I have not found any note of a phenomenon exactly corresponding to that observed in Walker's case when attempted to elicit. Each evertube when the result was a very rapidly produced and transient line of gooseflesh
The phenomenon is allied to the wheals produced in cases of urticaria perforans.

In two or three cases exceptional nervous phenomena are described. Ashton found a partial left-facial palsy, and Glaser's patient had an attack of intense hyperaesthesia, especially in the palmar and soles, followed by complete but transitory anaesthesia in the limbs. McConnell's patient had repeated short attacks of deafness, associated with piddliness, which ultimately became permanent.

Cowie's patient had early several periods of oppression of breathing and dyspnœa, without pulmonary anœmia, chief, but this may have been due to the faulty heart.
by the operative inhalation of chloroform; but the paroxysmal

respiratory dyspnoea of Ralph's patient could not be due to this.

Schickhardt's patient, a woman, also suffered from dyspnoea

and cyanosis. The respiration rate was 22. Dyspnoea and
cyanosis. The respiration rate was 22. Dyspnoea and
<table>
<thead>
<tr>
<th>Name</th>
<th>Age</th>
<th>Sex</th>
<th>Race</th>
<th>Religion</th>
<th>Education</th>
<th>Occupation</th>
<th>Income</th>
<th>Marital Status</th>
<th>Health</th>
<th>Physical Condition</th>
</tr>
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<tr>
<td>John</td>
<td>45</td>
<td>M</td>
<td>White</td>
<td>Catholic</td>
<td>College</td>
<td>Teacher</td>
<td>$50k</td>
<td>Single</td>
<td>Good</td>
<td>No</td>
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<tr>
<td>Jane</td>
<td>30</td>
<td>F</td>
<td>Black</td>
<td>Protestant</td>
<td>High School</td>
<td>Nurse</td>
<td>$30k</td>
<td>Married</td>
<td>Fair</td>
<td>No</td>
</tr>
<tr>
<td>Mike</td>
<td>25</td>
<td>M</td>
<td>Asian</td>
<td>Buddhist</td>
<td>High School</td>
<td>Engineer</td>
<td>$45k</td>
<td>Single</td>
<td>Poor</td>
<td>Yes</td>
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<tr>
<td>Lisa</td>
<td>35</td>
<td>F</td>
<td>White</td>
<td>Jewish</td>
<td>College</td>
<td>Lawyer</td>
<td>$60k</td>
<td>Married</td>
<td>Excellent</td>
<td>No</td>
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**Notes:**
- Age is approximate.
- Race categories are broad and may include more specific subcategories not listed.
- Religion is self-reported and may not be fully accurate.
- Education levels range from primary school to graduate degrees.
- Occupation includes various types of work.
- Income is annual and subject to change.
- Marital status includes single, married, and other.
- Health status is categorized as good, fair, poor, or other.
- Physical condition is noted as being good, fair, poor, or requiring assistance.

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**Tabular Statement of the Condition of the Union**

- Total number of persons: [Number]
- Average age: [Age]
- Median income: [$Income]
- Prevalence of chronic conditions: [Percentage]
<table>
<thead>
<tr>
<th>Source</th>
<th>Amount</th>
<th>SG</th>
<th>Urine</th>
<th>Ammonia</th>
<th>Lactic</th>
<th>Bilirubin</th>
<th>Bilirubin</th>
<th>Alkaline</th>
<th>Carbonate</th>
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<tbody>
<tr>
<td>Roseckaia</td>
<td>1076</td>
<td>1.8%</td>
<td>0.03%</td>
<td>no</td>
<td>no</td>
<td>no</td>
<td>0</td>
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<tr>
<td>Jees</td>
<td>1029</td>
<td>1.18%</td>
<td>-</td>
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<td>no</td>
<td>no</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Konin</td>
<td>1072</td>
<td>2.17%</td>
<td>little</td>
<td>little</td>
<td>little</td>
<td>no</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Akeley</td>
<td>1079</td>
<td>0.57%</td>
<td>-</td>
<td>no</td>
<td>no</td>
<td>no</td>
<td>-</td>
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<td>-</td>
</tr>
<tr>
<td>Flumina</td>
<td>1079</td>
<td>1.97%</td>
<td>-</td>
<td>no</td>
<td>no</td>
<td>no</td>
<td>-</td>
<td>-</td>
<td>-</td>
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<tr>
<td>Stueer</td>
<td>1078</td>
<td>1.6%</td>
<td>-</td>
<td>no</td>
<td>no</td>
<td>no</td>
<td>-</td>
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<td>Blubing</td>
<td>1079</td>
<td>4.36%</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>no</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Stauffi</td>
<td>1033</td>
<td>1.6%</td>
<td>-</td>
<td>no</td>
<td>no</td>
<td>no</td>
<td>-</td>
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<td>-</td>
</tr>
<tr>
<td>Ludwig,</td>
<td>1024</td>
<td>4.36%</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>no</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Oppolzid</td>
<td>1075</td>
<td>4.36%</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>no</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Wagner, Curt.</td>
<td>1027-35</td>
<td>1.13%</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Bonnet</td>
<td>53</td>
<td>1.13%</td>
<td>-</td>
<td>-</td>
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</table>
The examination of the urine is often most imperfectly recorded. In some cases very incomplete, which is perhaps scarcely to be wondered at as the facilities for its complete chemical investigation are absent in private practice and in most hospitals which are not attached to a medical school. The quantity of urine it is often impossible to determine as it is so often passed unconsciously. In eight cases where it is noted it was scanty, in four (Blueden, Wagne) Reese & McConnell), normal, in two (Greve, Reese), in Davanov's (68 oz) & in Smith's, Hunter's polyuria (116 oz).

The specific gravity is an interesting characteristic
nor is the colour, for though bile pigment is present in most cases, it is absent from some even where there is jaundice (Buso) or variably present and absent (Appleyard, Rolfe Carrington). Bile acids are very seldom noted and equally and may be present or absent. The presence of albuminuria has been used by some writers to establish a theory of uraemia in Explanation of the severe nervous symptoms, but the facts scarcely warrant such a foundation. For albumin absent in 22 cases, in 6 could merely be traced and while present at some point in 17 cases in 5 was only found in large proportion (Mossé Gräser).
The amount of urea and its near-chemical allies could seem a matter of some importance, as it might be expected to give some idea to the extent to which the physiological duties of the liver were performed, an expectation which is in some measure realized. The estimation has in most cases been made by the Hypobromite method, and in the majority diminution, sometimes considerable, has been found. Lewitski, Casley, Rosenheim, Rangee found less than 1%, in dry cases it was under 2%; Rulfs found it normal, Bleding abundant. While in worst case it was 3.6% and in Guttman's 4.36%.
Knights have been expected that the proportion of
urea would vary inversely to that of the leucin
and tyrozin, which are regarded as its bodies
not completely aminated, whose ultimate
destitution is the formation of urine, but in the
cases of Hiley and Rosenheim where urca was most
scanty, leucin and tyrozin were absent, while in
that of Tatarin they were both present in
considerable quantity. The explanation of this
discrepancy must be looked for in some
other direction, possibly by the investigation of
the bodies set free by décomposition of the liver.
The presence of Leucin. Tyrosum in the urine demands have been almost the most frequently investigated point in the urine of acute liver atrophy, which is poverty due to diseases having pointed out their existence and considered them pathognomonic. That it was in error is now well known and would appear again from the cases under review, where the absence of both was found in 22 cases, as it was also in Dr. Kellogg's cases after most careful investigation. In 8 cases both Leucin. Tyrosum were found, in 3 instances Leucin. alone was found and in 2 Tyrosum alone. Much the matter has lost the importance it had when.
These substances were regarded as mere alternates of phlegm, the matter still deserves investigation. Even if merely for the physiological interest involved, clinically Dr. Anderson of Darlington would have ascertained the importance of the presence of leucocytosis still lower, but as he has not published the method by which he found them in all classes of disease from Rottler to Perniciosa, one is not surprised in accepting his evidence which has not been confirmed by any of the most competent observers who have investigated the point. (Zoppe-Dyser, Bleulermann.)
The chemical misstatement of Röhrmann (Berl. chem. Woch. 1888, 861) and the experiments in Typhous feeding made by Bludenmaier & Baeu (Zeit. f. phys. Chemie VI. XI) make it probable that in future the examination of the urine should take a slightly different direction, towards the search for Phenol and the Aromatic Acids, which arise from the destructive destruction of Typhous and are found in cases of destructive processes within the intestine. Further the relation of the Xanthine bodies is of importance as it may give instructive information of intrinsic tissue change. Selman showed
that Xanthin is formed from xanthin in the blood and it's Xanthin to Xanthin
Phosphate Acids and we should expect that
where the destruction of nuclei is more than
ordinary or if it were trace of these bodies
might be shown by the increased of these bodies
in the urine. Stilling and Babinsky have shown
that Xanthin occurs normally in urine, the
percentage (Babinsky) in these cases being .0028
.0038 .0030. Now Roehnave found in his
cases of Acute liver Atrophy .0086% of Xanthin
a nearly 4-fold increase which is at least suggestive
The result of the oxidation of hypoxanthin is
Uric Acid, and when the kidneys are intact
this process is completely performed, but Xanthin:
this have been found increased in the urine of
Nephritic children and in leukæmia when
the nuclear processes in the leucocyte bulk
is largely we find a large increase of Uric
Acid. In future their the urine should be
much more carefully examined as to its
content of Uric Acid, Phosphoric Acid
Oxy-acids & Xanthin Bodies Shend.

Sharpey & Stephnson [Guru Inst. Rep. 43, 275, 1890]
Examining the urine in a case of phosphorus poisoning found a large increase in the content of ammonia, and in Frankel's case in another the ammonia content was increased fivefold. This point has been so far examined by Stadelmann that increase from two to fivefold was no uncommon affection of the liver. Briefly speaking, 12 out of the 13 liver cases examined.
<table>
<thead>
<tr>
<th>Name</th>
<th>Age</th>
<th>Weight</th>
<th>Colour</th>
<th>Consistency</th>
<th>Degeneration</th>
<th>Cells</th>
<th>Cell Infiltration</th>
<th>Fibrosis</th>
<th>Ducts</th>
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<tbody>
<tr>
<td>Green</td>
<td>18</td>
<td>12½ oz.</td>
<td>Yellow</td>
<td>soft</td>
<td>invisible</td>
<td>pigmented fatty</td>
<td>normal (cholang)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Smith</td>
<td>50</td>
<td>18½</td>
<td>Yellow</td>
<td>flabby</td>
<td>none</td>
<td>none</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dressfield</td>
<td>25</td>
<td>68</td>
<td>Brown</td>
<td>deep yellow</td>
<td>little marked</td>
<td>normal, granular</td>
<td>w. fat drops</td>
<td>some portal</td>
<td>diseased</td>
</tr>
<tr>
<td>R (Ro)</td>
<td>20</td>
<td>72</td>
<td>Red</td>
<td></td>
<td></td>
<td>normal, fatty</td>
<td></td>
<td>diseased</td>
<td></td>
</tr>
<tr>
<td>Senator</td>
<td>8½</td>
<td></td>
<td>Red</td>
<td>Yellowish</td>
<td></td>
<td>normal</td>
<td>fatty and</td>
<td></td>
<td>diseased</td>
</tr>
<tr>
<td>Dressfield</td>
<td>22</td>
<td>23</td>
<td>Reddish black</td>
<td>2 yellow patches</td>
<td>diseased</td>
<td>perfect normal, scanty</td>
<td>Central diaphragm</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Oth (Buss)</td>
<td>19</td>
<td>large</td>
<td>Red</td>
<td>Sun</td>
<td>almost normal</td>
<td>granular</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Brodowski (Sw)</td>
<td>15</td>
<td>small</td>
<td>Red</td>
<td>(none), large</td>
<td>Swollen pro. pigmented</td>
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<tr>
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<td></td>
<td></td>
<td>(1) large</td>
<td>Small &amp; numerous</td>
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<td>Cells</td>
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<td>Remaining</td>
<td>Ducts</td>
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<tr>
<td>Keboel</td>
<td>24</td>
<td>31</td>
<td>Yellow</td>
<td>Shackly</td>
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<td>Abnormal</td>
<td>Normal</td>
<td>Wide tract.</td>
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<td>Fatty - mucinoid - detritus</td>
<td>Strands</td>
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<td>Rorschfeld</td>
<td>28</td>
<td>24</td>
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<td>Soft</td>
<td>Small</td>
<td>Swollen fat granules - detritus</td>
<td>Some</td>
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<td>4</td>
<td>2½</td>
<td>Yellow</td>
<td>Soft</td>
<td>Large</td>
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<td>Portal space</td>
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<td>Forneulles adult</td>
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<td></td>
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<td>Portal wall</td>
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<td>33½</td>
<td>Yellow</td>
<td>Yellow</td>
<td>Small granules - intrabiliary</td>
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<td>Ductitis</td>
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<td>16½</td>
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<td>Ductitis</td>
<td>None</td>
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<td></td>
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<td></td>
<td></td>
<td></td>
<td>Red</td>
<td>Fine</td>
<td>None</td>
<td>Ductitis in fibrous capsule</td>
<td>Artenua</td>
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<td>Cells</td>
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<td>Formation</td>
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<tr>
<td>Fris</td>
<td>25</td>
<td>M</td>
<td>Yellow red</td>
<td>firm</td>
<td>advanced</td>
<td>fat</td>
<td>decreased</td>
<td>few preserved, portal canals</td>
<td></td>
</tr>
<tr>
<td>Rosemarijn</td>
<td>10</td>
<td>M</td>
<td>Yellow red</td>
<td>firm</td>
<td>marked</td>
<td>fat</td>
<td>decreased</td>
<td>few preserved, portal canals</td>
<td></td>
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<td>Breuckhardt</td>
<td>32</td>
<td>M</td>
<td>Yellow red</td>
<td>firm</td>
<td>marked</td>
<td>fat</td>
<td>decreased</td>
<td>few preserved, portal canals</td>
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</tr>
<tr>
<td>Dreschke</td>
<td>38</td>
<td>M</td>
<td>Yellow red</td>
<td>firm</td>
<td>marked</td>
<td>fat</td>
<td>decreased</td>
<td>few preserved, portal canals</td>
<td></td>
</tr>
<tr>
<td>Macallum</td>
<td>24</td>
<td>M</td>
<td>Yellow red</td>
<td>firm</td>
<td>marked</td>
<td>fat</td>
<td>decreased</td>
<td>few preserved, portal canals</td>
<td></td>
</tr>
<tr>
<td>Ehrigard</td>
<td>28</td>
<td>M</td>
<td>Yellow red</td>
<td>firm</td>
<td>marked</td>
<td>fat</td>
<td>decreased</td>
<td>few preserved, portal canals</td>
<td></td>
</tr>
<tr>
<td>Cayley</td>
<td>30</td>
<td>M</td>
<td>Yellow red</td>
<td>firm</td>
<td>marked</td>
<td>fat</td>
<td>decreased</td>
<td>few preserved, portal canals</td>
<td></td>
</tr>
<tr>
<td>Curnes</td>
<td>23</td>
<td>M</td>
<td>Yellow red</td>
<td>firm</td>
<td>marked</td>
<td>fat</td>
<td>decreased</td>
<td>few preserved, portal canals</td>
<td></td>
</tr>
<tr>
<td>Scholz</td>
<td>18</td>
<td>M</td>
<td>Yellow red</td>
<td>firm</td>
<td>marked</td>
<td>fat</td>
<td>decreased</td>
<td>few preserved, portal canals</td>
<td></td>
</tr>
<tr>
<td>Author</td>
<td>Age</td>
<td>Liver</td>
<td>Colour</td>
<td>Consistency</td>
<td>Degeneration</td>
<td>Cells</td>
<td>Infiltration</td>
<td>Fibrosis</td>
<td>Other</td>
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<td>----------</td>
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<tr>
<td>Tedennis</td>
<td>28</td>
<td>31½</td>
<td>Yellow nodules, soft</td>
<td>firm</td>
<td>soft dist.</td>
<td>Hyperplasia, atrophy</td>
<td>portal</td>
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<td>25</td>
<td>31½</td>
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<td>firm</td>
<td>soft dist.</td>
<td>fatty, complete destruction</td>
<td>portal</td>
<td>portal</td>
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<tr>
<td>Bornet &amp; Boy Tobbien</td>
<td>69</td>
<td>63½</td>
<td>Dark yellow</td>
<td>firm</td>
<td>almost vanished</td>
<td>portal</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Haun.</td>
<td>27</td>
<td>small</td>
<td>brown, yellow</td>
<td>firm</td>
<td>firm</td>
<td>Early</td>
<td>Many</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sandberg</td>
<td>34</td>
<td>31</td>
<td>brown yellow</td>
<td>firm</td>
<td>firm</td>
<td>Fatty</td>
<td>few</td>
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<td></td>
</tr>
<tr>
<td>McConnell</td>
<td>51</td>
<td>38</td>
<td>yellow-brown</td>
<td>firm</td>
<td>firm</td>
<td>Fatty</td>
<td>few</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Smith</td>
<td>42</td>
<td>½ size</td>
<td>yellow, pale</td>
<td>firm</td>
<td>firm</td>
<td>Fatty</td>
<td>few</td>
<td></td>
<td></td>
</tr>
<tr>
<td>---</td>
<td>33</td>
<td>49</td>
<td>dark yellow</td>
<td>firm</td>
<td>firm</td>
<td>Fatty</td>
<td>few</td>
<td></td>
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<tr>
<td>Musié</td>
<td>44</td>
<td>67</td>
<td>yellow, firm</td>
<td>firm</td>
<td>firm</td>
<td>Fatty</td>
<td>few</td>
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Sodium, with clubbing, near portal, canals branched.
<table>
<thead>
<tr>
<th>Author</th>
<th>Age</th>
<th>Liver</th>
<th>Colour</th>
<th>Consistency</th>
<th>Lobulation</th>
<th>Cells</th>
<th>Infiltration</th>
<th>Fibrosis</th>
<th>Duets</th>
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<tr>
<td>Gruitz</td>
<td>Adult</td>
<td>medium</td>
<td>icteric</td>
<td>Sticky</td>
<td></td>
<td></td>
<td>Kellenweic, fett, deformative.</td>
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<tr>
<td>Shattock</td>
<td>6/2</td>
<td>7 oz.</td>
<td>Pale</td>
<td>Sticky</td>
<td>Granulated, granular,</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Leach</td>
<td>17</td>
<td>30</td>
<td>pale</td>
<td>Sticky</td>
<td>Granular, melanin,</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Jhanova</td>
<td>24</td>
<td>81</td>
<td>yellow</td>
<td>Sticky</td>
<td>Granular, melanin,</td>
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<tr>
<td>Sutupni</td>
<td>19</td>
<td>19</td>
<td>orange</td>
<td>sticky</td>
<td>Granular, melanin,</td>
<td></td>
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<tr>
<td>Heukelman</td>
<td>3/2</td>
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<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Döpfel</td>
<td>31</td>
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<td>yellow</td>
<td>Resorbable</td>
<td>'acute atrophy'</td>
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<tr>
<td>Strandheinsen</td>
<td>19</td>
<td>19 3/4</td>
<td></td>
<td>Pulpy</td>
<td>'acute atrophy, biliary contraction'</td>
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<tr>
<td>Appleyard</td>
<td>35</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Goodhart.</td>
<td>23</td>
<td>13</td>
<td>Red</td>
<td>normal</td>
<td>Scarpinjo, fat, hepatic cells, only oil globules.</td>
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<tr>
<td>Tries</td>
<td>26</td>
<td>42</td>
<td>Yellow</td>
<td>soft</td>
<td>Scarpinjo, fat, pigment, well preserved liver cells.</td>
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</tbody>
</table>

**Footnotes:**

- *Fett, deformative* indicate fatty degeneration.
- *Granular, melanin* suggest the presence of melanin granules.
- *Resorbable* indicates the tissue can be absorbed or broken down.
- *Scarpinjo* refers to a type of pathology.
<table>
<thead>
<tr>
<th>Author</th>
<th>Age</th>
<th>Sex</th>
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<th>Cells</th>
<th>Infiltration</th>
<th>Fibrosis</th>
<th>Ducts</th>
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<td>53</td>
<td>yellow</td>
<td>firm</td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Schickhardt</td>
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<td>20</td>
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<td></td>
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<td>Lafitte</td>
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<td>33(\frac{1}{2})</td>
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<td></td>
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<tr>
<td>Mozé</td>
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<td>57</td>
<td>yellow</td>
<td>green</td>
<td>normal</td>
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Anatomy of the Liver.

Reading the isolated descriptions of the liver tissue from several cases in a series one might fancy that the processes recorded under the title were very different, yet when the results are analyzed it is found possible to arrange the whole in a more or less coherent graduated sequence and to recognize that we are dealing with a more or less exactly definite but still individual method of process, the condition of the tissue being roughly indicated by the color on the section viewed by the naked eye. In the very great majority of cases the colors of the liver are red or reddish brown and a rich orange yellow.
and as a rule the yellow parts are deeper than the red. The condition of the red tissue is not always the same. In a few cases, (in most it is the red of the advanced disintegration known as red atrophy. The yellow colour of the tissue during the earlier stage of disintegration, is due to the accumulation of bile pigment which seems however almost more intense than could be as a result of general jaundice only. The colouring matter of the yellow tissue is loosely connected...
and found off the surface in every washings, and in microscopic section is found in loose granules or small accumulations. No very clear explanation of the nature of the most degenerate tissue has been given, but in at least three distinct instances in my sections it was associated with microscopic haemorrhages.

In a few cases the colour is described as green, yellow green or olive green, but this seems to me probably a postmortem change which I found in Dr. Whyte's case distinctly marked, and in the case of the boy, icteric three months after the death of the patient.
In the classification of the liver on histological ground, two elements are to be taken into consideration: (1) the condition of the hepatic cells (2) the condition of cell infiltration and fibrous tissue formation. These conditions are frequently modified by the characteristic changes occurring in alveolar blood already the seat of previous morbid process, as in Dr. Schefeld's case where the acute atrophy intervened on a condition of fatty infiltration.

In cases of acute liver atrophy the cells when first affected become cloudy, their protoplasm becomes granular and the nucleus
less distinct or it may be found dividing with the cells smaller and the protoplasm granular. The granular condition of the protoplasm is in this condition usually degenerative, the granules being fat, pigment, and albuminoid matters. Afterward the cells may shrink still retaining their nuclei or the protoplasm may become indistinct and the cell gradually resolve into a mere heap of granular debris. These changes seem as a rule to occur primarily in the cells at the periphery of the laminae especially those next the portal spaces from which indeed
the sources of their nutrition or in this case malnutrition arise. The debris resulting from this disintegration of the cells is then gradually removed the liver tissue being left as clear and the other tissue elements having thus far been scraped off. The changes taking place are then at different stages even in the same lobe and when we find cell remnants they are generally arranged radially from the central vein.

The reports of the published cases seem to show farther an unwise desire to deny a diagnosis than
to arrive at a correct understanding of the normal process, and while the condition of the hepatic cells is noted that of the remaining tissues has been less frequently examined. This is not without advantage for while the less degrees of cellular infiltration have probably been missed in many cases where there is no note of their occurrence, the mention of them when made is mostly in the report of highly skilled and professional pathologists and is therefore more trustworthy. More extreme degrees of infiltration and fibrous tissue formation are of course so marked as in my first and second cases that they attract attention even
Despite the liver cells can be examined, but earlier's slighter degree requires careful search and may rarely be entirely absent as in the deep yellow tissue of Kupffer's case. Usually a little infiltration of small round cells (leucocytes) may be discovered at some at least of the portal spaces, sometimes this is considerable and may stray along the bile canaliculi tissue as well as between the columns of liver cells, occasionally it becomes excessive and pervades the whole tissue radiating from the circumference of the portal vessels. At times when the liver cells are absent from already integrated lobule and we can see the
Stroma. Clearly, we find that it is friable, granular and not extended, but it is most common to find the strands wider with small degenerating cells adherent to the strands, sometimes a little cellular infiltration along its bands and frequently especially in the bilateral areas we find it markedly increased in extent, distinctly fibrillated and fully supplied with flat nuclei. In some cases we find masses of firm tissue of this character occupying the whole of the lower power field and visible to the naked eye as distinct gray bands or cut-cut surface projections as whitish points.
The essential characters of this morbid process are the granular, sometimes fatty, degeneration of the liver cells spreading from the portal spaces, their tendency to become reduced in size or even disintegrated, and their ultimate absorption; and associated with this we have the tendency to cellular infiltration lesser more marked and the spread of the interstitial tissue; from this as an ultimate stage we have contraction, a certain amount of cirrhosis and the pigmentary degeneration of slow atrophic processes. We have in fact parenchymatous and interstitial hepatitis ending in advanced extensive cellular necrosis.
After the house has been integrated there occurs a contraction which in at least two of my cases are most marked under the capsule, a fact somewhat counter to the explanation by Shedd and Macallum found in the inverse condition in their case. This contraction of tissue is a matter of considerable importance especially with reference to the appearance of multiplication of the bile ducts which has been frequently noted since the first descriptions of it by Waldeyer and which has been made the basis for a theory of attempted regeneration of the liver by justification from the epithelium of the bile ducts.
As a rule to which there are occasional exceptions, in the whole case, the epithelium of the bile ducts is but slightly involved by the degenerative processes, and being thus preserved, the actual length of the tube will not be diminished. If then the volume of the liver is considerably reduced (Tobacco's case 450 c.c. instead of 850 c.c.) the distance between two points on the duct must be reduced (in the proportion of the cube roots). The result is that the ducts will become convoluted and thus in any plane of the liver the same duct will appear repeatedly and in sections will be repeatedly cut and may occasionally be
loops have been cut in both branches for the appearance of a multiplication of ducts sections and so an apparent multiplication of ducts. This however is not the whole extent of apparent multiplication. It has been pointed out by Goodhart and others that by the contraction of the tissue which the reduced size and distortion of the lobules indicate, quite separate ducts are comprisestarless.

Of These two occurrences of which the figure illustrates the first by a drawing which shows a single bile duct convoluted with some of its bends concave to the eye crossed by distinct bands.
of its non-tissue and at its convex loop cut. Showing the circle of epithelium more less distinctly according to the obliquity of section, and in my opinion the multiplication of ducts in my sections, and it is very flat may be completely explained in this way. Of the club-like outgrowths described by Kobo my sections merely confirm the adverse opinion of Keppifer and Hava. Such processes are abundant but they show an epithelium as fully developed and a membrane propria as complete as the neighbouring central duct and are in fact merely truncated branches.
Nowhere in all my sections have I found the slightest indication of nuclear division or other proliferative process in the epithelium of the bile capillaries. This, however (Champ. Bio. Dorpat 1886) later reporting on three cases states that he has found 

division of the epithelium and that the phase of transition in their regeneration from the bile ducts can be traced. And as I have not seen the original paper itself must hold some reserve is shown of the perfectly efficient mathematical explanation which has been given above.

Regeneration of the liver

The question of regeneration of a liver when it has
Vicious subject to the morbid process of acute atrophy is not however limited to the theory of reproduction from the bile ducts, and it may be as well at this point to summarize the evidence on this matter. The liver in normal conditions has an almost astonishing potentiality of regeneration. Langenbuch (Bstl. Klin. Woch. 1888) removed about a pound of human liver with no other apparent disturbance of function than temporary ascites. Parfick (Vieh-Arch. 118, 193) experimenting on rabbit excised parts of the liver varying from one to three quarters of the whole organ, found that the loss of one quarter was completely or almost overcompensated.
in 8 days, and Reisnikow (Arch. path. d. gr. 1851) confirmed the result in guinea-pigs. von Heigel-Certalbl. f. allg. Path. 1851) found that in dogs and rabbits a large part of the liver took place in 36 days, by hyperplasia and hyperplasia of the liver cells, that the secreted urine diminished in proportion to the portion exceeded, and that increase of the albumen occurred.

Slow poisoning phosphorus (Klingig, Arch. path. 110, 572 finds all division in ducts.) District, Halle. 1885) finds that phosphorus poisoning leads to hypostasis and division of the liver cells. On the other hand, Rotoff (Centrallbl. f. med. Wien. 1887, 574) finds that poisonous lupins produce in sheep
Jaundice and in the liver, changes typical of acute yellow atrophy or of nephrotic liver. Recovery when it occurs ends at partial atrophy and is very rarely complete. Again distinct karyokinetic changes in the liver cells have been recorded in several cases. Hirshberg and Dinkler have already mentioned and lately a detailed report of this occurrence has been published by W. Muddu Macallum (P.M.T. 1894). In my observations I have examined very carefully the nuclei of the liver cells as well as of the bile duct epithelium and the only instance in which I have found karyokinetic change was in the yellow
Masses of small cells.

Masses of liver cells are found almost always arranged in a more or less circular manner. This arrangement has been used as an illustration of regenerative effort, an interpretation which it is unable to support. Neumann (Anatom. Zeits. Berli. 1888) on the influence of poisons on the size of the liver cells, working in stilbene
laboratory on white mice found the greatest variety in the size of liver cells (18.7 - 115.6 micro millimetres) in different stages of nutrition of the cells. In an earlier research publication (Pflüger's Archiv 30. 385) found that in dogs starvation greatly reduced the size of the liver cells.
In a few cases only has the condition of the bloodvessels in the liver been noted, but, especially in view of the marked haemorrhagic tendency, the results are of considerable interest. It is impossible to form any definite notions as to the amount of blood in the liver except perhaps that it may vary from congestion to anaemia. Noma found the condition to vary greatly from part to part of the liver, even within the yellow portion where when the cells were numerous the bloodsupply was rich and at points where the cells were few the bloodsupply was absent, there being but few vessels in corporcles. Noma compares the ridicule in his case with numerous corporcles in reticuli of Brodowsky found under a microscope. The field almost full of small capillaries full of blood. After Carrington...
In this case, describes the old tissue as vascular granulation tissue, and Kahlke found the capillaries markedly impreciated and provided with well nucleated lining. Schickhardt found the vessels dilated, and Murer found blood in the intralobular mesh.

The arterial walls have been found thickened by the same two observers and by Bossenfeld. Thickened and infiltrated with connective tissue, the intralobular capillaries enlarged, while Bordowski found the senescent wall substituted by cells. In my own cases, I have at present rest examined embalmed sections and histological sections could be placed on negative results, and the positive results are that in my two cases considerable minute hemorrhages were found

with substance of the liver that in the smaller vessels
the lumina were seldom clear, frequently blocked by the lining endothelium which seemed sometimes so thickened that the outer layers of the arterial walls were frequently thickened so that in Prussian Blue staining fatty particles were found sparingly distributed in the walls. The changes described seem to justify a conclusion that a certain amount of activity is not uncommon, and I may here note that Pashelev (2. June, 1861) found particles in the cutaneous vessels a priori to which we shall have again to turn.
microfungemia, and the condition is often found in the skin, particularly in the hair and nails. The presence of microfungemia in these areas has been linked to an increased risk of infections, especially in individuals with weakened immune systems.

In the case of patients with microfungemia, treatment is often necessary to prevent further complications. Antifungal medications are typically used to treat the condition, and lifestyle changes, such as improving hygiene and reducing stress, may also be beneficial.

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of the liver is independent of dried bacterial action in the liver cells. Microorganisms not always the same have however been found in the liver even when removed (Wirsching) 30 minutes after death, and Bonnet and Bay Leissier found in the liver a diplococcus which they had found in the blood 30 hours before death. Balger has found organisms in the liver cells, but the results of Edouard are perhaps the most important further positive contribution. Itist made to this subject. In three cases, acute liver atrophy (25 ml), necrosis of liver tissue and interstitial hepatitis he has cultivated from liver substance, from heart blood and from systemic blood at abscesses, the same in all.
cases and which was not found in any of six control cases. Inoculation was made to rabbits with vibriospio, and so for large rats and dogs which survive after an illness, after intravenous inoculation, and the bacillus is weakened by inoculation into the animals. After intravenous injection to vibriospio die in from 12 to 36 hours, and the liver is found much swollen containing innumerable numbers of the bacillus. In parts the capillaries are dilated, the cells smaller or destroyed part being decidedly necrotic with bacilli in the centre. On intra-peritoneal injection the liver is enlarged and dotted with grey yellow patches due to necrotic masses from which the bacillus...
is easily grown. Rabbits at first show oedema for a day or two then decompose quietly will yet they die in about a month and the liver is found atrophied even though it maintains its volume. In rapid cases necrosis occurs but atrophy is not found in cases of long duration the necrotic tissue is replaced by connective tissue growth. Thus Gianotti thinks may be explained the connection between acute infective jaundice, acute atrophy and acute fatal asbestosis.

[This rather inconvenient concept is somewhat recent literature especially American a tendency to speak of Acute Infective Jaundice as Weil's Disease]
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<tr>
<th>Tissue</th>
<th>Kidney</th>
<th>Heart</th>
<th>Muscles</th>
<th>Stomach</th>
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<tr>
<td>Smith</td>
<td>Bilis stained. Impair in color</td>
<td>Slightly fatty degeneration</td>
<td>Epithelium fatty</td>
<td>Epithelium fatty, degeneration. Diaphragm, cuticular fatty degeneration. (Pancreas Fibrous.)</td>
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- Epithelium degenerated, convoluted tubules, less in straightness.
- Striation of sheaf.
- Granular, striate metachromic tubule.
- Full of fat.
- Flattened epithelium, cloudy swelling.
- Slight epithelium, cloudy fatty, old diaphragm, cell infiltration, lamina propria, muscularis, and muscle.
<table>
<thead>
<tr>
<th>Histology of</th>
<th>Kidneys</th>
<th>Heart</th>
<th>Muscles</th>
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<tbody>
<tr>
<td>Tissue</td>
<td>parenchymal degeneration</td>
<td>fatty, granular degeneration</td>
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<td>cell infiltration, no organisms.</td>
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<td>Cilia</td>
<td>epithelium cloudy glomeruli, hyaline change</td>
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<td>Mesothelium</td>
<td>cloudy swelling, amorphous areas</td>
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<td>Gastritis mycose.</td>
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<td>Bacteria in bowel, at base of mucosa. Few in fundus.</td>
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In 1847 (Cheltenham) Sandfield Jones found the kidney epithelium coarse-granular and asserted the identity of the process in both kidneys and urine that time as considerably numerous observations upon condition of the kidneys have been published. In the more recent cases (when not examined merely to confirm the diagnoses) a report on the condition of the kidneys is usually given and in almost every instance some kidney mischief more or less extensive is revealed. This is plausible what we should expect from the occurrence of albuminuria or urine containing casts. Sansbury says that in his case there was nothing noteworthy in the condition.
of the kidney, but as each kidney was a little enlarged weighing 703, I do not know if it is safe to conclude that they were absolutely normal.

As a rule the cortex of the kidney is to the naked eye a little broader than normal, and the pyramids darker and frequently congested. On microscopic examination the epithelium is found in a state of brunt cell swelling or in the advanced condition of granularity. Sometimes pigmented and not infrequently disintegrated as to be absent at part of the section. The disintegration is as a rule more marked in the convoluted than in the
straight troubles, but both are involved sometimes to an equally great extent. In addition to this there is frequently noted on several occasions a certain amount of cellular infiltration (Moné) or an increased interstitial fibrous tissue (Duckworth). This is however by no means constant and has been found absent by Harris & Woman, so that in the kidney the lesion may be summed up as a parenchymatous nephritis, with occasionally some interstitial cellular change, which describes accurately the condition in my cases.
Heart

The heart muscle as a rule shows some alteration in the direction of fatty degeneration. This may be very slight as in Carvalho's case where the striæ were quite distinct on extreme microscopy recorded by Ammitage.

Other muscles have been less frequently examined than the heart and the changes in them occur less extensively (Schönberg) though no advanced fatty heart of Ammitage's case was associated extreme fatty degeneration of the atria thrombus.

Stomach

The frequency of haematemesis and gastric symptoms was sufficient to attract attention to the condition of the stomach. As a rule even
in Trischfeld's case where haematemesis was the
major symptom no abrasion can be discovered by
the naked eye, though sometimes there are ecchymoses
in the gastric surface. But microscopically these
seems to be very frequently fatty degeneration of the
 gland epithelium or even disappearance (Rosenkranz)
and commonly also infiltration of the interstitial
tissue. In some cases (Heckelom, Sijenbeek)
microorganisms have been found. From these these
authors have concluded that gastritis bycoticca
may give rise to acute liver atrophy by
stonanie poisoning.
Table showing the Age & Sex in the cases used for analysis and in the cases of females whether pregnant (-m) or lactating (-l).

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Frerichs-31 cases-22 women-11 pregnant

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<td>Buist</td>
<td>97</td>
<td>51 females, 10-3 lactating</td>
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Utrecht General Hospital
26th June
16
Etiology

The recent cases apart perhaps from the bacteriological investigations furnish little definite contribution to the etiology. They however as will be seen when one looks at the tables and cases drawn from cases where the twin cells are found destroyed examined microscopically fill up the lacuna between 5-10 years in which no case had previously been recorded and they tend to alter the incidence of the marked process from the female and male sexes about the degree to reduce the proportional influence of pregnancy and lactation. There is again a notable number of cases in which psychiatric disturbance has
determine the production of this disease, and the
listed 10 cases in alcoholics and 14 in syphilitics
emphasized the influence of these liver-disturbing
agencies, in preparing the way for more serious
changes. Mene's case after inhalation of Arlatsium
which is to be compared with a similar one in Bamberger's
Krankheiten des hypophyseischen Systems, Büchel's case
the list of cases with proctoscopy first emphasized
the influence of proctoscopy in this connection. It is
a matter of special interest to note that this
lesion of the liver is not confined to the
human species but has been found in horses
(Sensu lato) and is produced in Sheep, Goats, Cows, and a few other animals when they are fed with Lupini (Pelto). In West Mecklenburg and with regard to the immediate agents in producing the destruction of the liver cells we have not yet arrived at any definite determination.

Bacteria have been found in the liver and (Garavic) should be adequate causes of the condition, but they have been so often absent that the direct action of bacteria cannot be essential. Microorganisms have been found in the wasp's stomach (Heckelom, Steigenbeck) and the theory that their presence have or have been
The determining influences harmonizes well with other cases of definite etiology. Whatever be the cause, the which determines the destruction of liver cells, its influence is not confined to this organ, but affects every tissue in the body, producing necrotic changes and usually degenerative changes to a greater extent. The influences which impair the previous health of the liver cells aid greatly the development of the atrophic process.

The relation of the destruction of the liver to the symptoms observed at the bedside is perhaps the most interesting of the many questions.
At issue in this matter and yet it has definite conclusions have been attained. It is clear that we must endeavour to separate the symptoms into three groups: 1. None due to the agent which affects the liver cells. 2. None due to the cessation or disturbance of the normal liver function. 3. None due to the presence of toxic decomposition products from diseased live in the body. Anorexia of the whole clinical picture does not yield a single symptom which so far we have at present may not result from a poisonous or infective
agent that in which does not come into the first class. It is then necessary to attack the question from another side and to ask what are the decomposition products of the liver and what are their physiological effects.

The chemical investigations are as yet unfortunately too few to give us any sure basis. Salkowski has found a large increase in the amounts of Peptone and Alkaline in the small, liver, kidney, and stomach. Stein respectively have shown that these bodies do not predominate again. We also know the influence of peptone in reducing
The constancy of the blood as far as haemorrhage and in other respects. Vital products are of necessity in used to explain the clinical picture. It is known as well be considered immediately probable that the haemorrhagic tendency is dependent on another factor. Rötmann's analysis practically infers this result. Salkowski's as he found in the liver an alburnusoid, peptide, lactic acid, leucin, traces of lynari, aromatic organic acids and xanthine bodies.
Cause of haemorrhages. Poepielow has made a careful study of the changes of the skin occurring in cases of this disease, and has found that muscles, vessels, and subcutaneous are all affected. The connective tissues are thickened, their fibres are cloudy, granulated, infiltrated with fat droplets. The subcutaneous were enlarged and fatty and the vessels showed indurations. Dompars in 1868 found fatty emboli in the lung arteries containing perfect liver cells in cases of Delinia. Women, Eclampsia, and Clefts in the liver of women dead of delirium tremens in pregnancy or shortly after delivery has found punctiform haemorrhages, with
fat-droplets in the crowded cells of the neighbourhood.
Round them were thrombi in the neighbouring portal
branches and within the portal thrombi, live
cells so that we have the choice at both ends.
Weiss had already made a similar discovery in
haemorrhagic small-pox when he found embryos of
bodily micrococci in the vesicles. An analogous
appliance for autogenous haemorrhaga has been found
by Richard and Kugener (Leit Fett: hist. x. 244) in the
form of Sudarative thrombi, in all kinds of
haemorrhagic diseases. I append a drawing of
Richard's from a case of Purpura haemorrhagica.
Which may be compared with the Jackson (91) in my small series showing the lumen of a liver artery blocked by multiplication of the endothelium along with the record of a similar nature by other observers.

This will furnish a cogent explanation of the microscopic hemorrhages found in inspection.
dit group of cases claiming to be recovered from acute liver atrophy.

Harley, 26. liver. M. 58. F. 15 pregnant.

St. Petersburg, med. week. 36. 1878. F. pregnant 8 mos. abortion.

Grammatikate. F. 22 pregnant.


Scot. Skinning.

Senator J. syphilis. Incontinent.

Lodi. 42 M.

Mueller 11 M.

Weising 26 F. pregnant. 3 mos.

Michael 60 M.

Morse 57 M. 24 M. 36 M.

Martinez 11 at ten years. Savannah civil hospital.
We have already considered the histological evidence in favour of regenerative changes taking place in alvei which has been the subject of the initial changes at least indicate incipient atrophy and we have seen that such a process may be inherently possible but to a certain extent frequently present in the living cells. The clinical evidence of the occurrence of recovery must of course and different and less secure basis. While there is little reason to go into this matter with the bogey of an enthusiasm of B.B. Stanley or Martinez there is good ground for supporting
the diagnosis in some of the cases. With all the clinical symptoms of "icterus gravis" we have a diminution of liver dullness so distinct that there is no doubt as to the diminution of the liver volume, from a diminution from which the liver is practically restored. The structural basis of this diminution is of course less surely established. It is difficult desirable to have certain experiments made as to the extent to which the diminution of the liver tissue affects its volume. In cases of biliary color with severe liver is enlarged by distention of the bile ducts with the vitriol fluid within the liver
volume at the normal. The obstruction reduces fluid palpable that in the closed orifice might the edge with the liver has reached its nerves, a change quite comparable with the marvellous rapidity of liver diminution in the last days of an acute atrophy. Upon such has made some prominent that might have given information on this subject. It has been after the time examining the influence of various nutritive conditions on the liver cells. He found that irritation of the liver nerves induced contraction of the corresponding lobe of the liver, and that section of the nerves dilated them by dislocation of the branches
The portal vein and hepatic artery, produced oedema and round cell infiltration and led to the growth of the interstitial connective tissue. Possibly among the other diverse phenomena we may attribute to some extent the rapid diminution in the size of the liver. It is possible that more definite chemical examination of the urine in the cases might by means of larger proportion of xanthine bodies and analogous constituents may enable us to determine the existence of actual nuclear destruction in these cases, but up to the present this investigation has not been made. It is of course work for the physiological chemist.
First of Cases arising after Psychical Influences.

Moria. Great mental fatigue

Tommasi. Strong psychical disturbance

Appleyard. Slight melancholia for some weeks (Chill - Sanguine Suffer)

Röhmann. Continuous psychical excitement.

Neuritic heredity. Had convulsions for some years (Hysterical). 


Bunkhardt. Sickered after visit to Railway Collision. (Same day)

One sister insane.

Duckworth. Sufferer youngest child almost run over. (Next day)

2 Cases unter histological suspicion.

Edwards. Much mental strain for five years.

Hawes. Eclampsia in first pregnancy. In great fear of this pregnancy.

Suckling. Mother seven years in Asylum.

Mr. Hudson. Slight infantile Paralysis.
death. Two other boys jaundiced tomorrow leave for training ship.

 Daly. Mr. Anderson. Child jaundiced recovered.

Arnold's Company. Ten soldiers occupying the same barrack.
Cases associated with poisonous ingestion

Supposed to have exposure to heroin from herbalist shop. Another died after similar illness.

Ralf w. W. after employer's 'brain attack'.

Daly. Attributed to foul esterin water. Several others ill, one jaundiced.

B coursework. Rapid illness after haem.

Troupe. Preliminary jaundice due to demonstrable error in diet.

Vern 7F. Stomatitis by prolonged treatment for jaundice.

Tico 26M Siphilitic

- 25 F -

Shelver 29 F.

with free mercurial treatment

with poisonous inhalation

Trinkel. Attributed illness to inhaling chemical fumes at his work.

Chew. Inhaled three ounces of Chloroform during Renal Colic.
Smith: Had taken beer freely & been frequently drunk.
Kebbe: Till lately very intemperate.
Adler: 'Potato.'
Jones: Drank for about a fortnight at Christmas holidays.
Morgan: Intemperate.
Cluthe: Formerly heavy drinker, abstinence for months.
Cayley: Dissipated & drunker.
Carrington: Good head. Had been drinking for six weeks.
Smith: Hard drinker.
Jenkins: Given to alcohol. Had been drinking when taken ill.

Expressly denied in some cases.
List of cases associated with syphilis

Fris 1 F. death. 1 M. death.
Sainsbury 1 F. death. March 1 F. death.
Senato 1 F. death. 1 F. after syphilis.

Bauer 1. Auer 1.

Epidemic Excluded in 82 cases.
Cases associated with other diseases.

Arginase, Yellow Fever. Five cases in hospital last winter before (no fuel)

Watson, Heart Disease.

Tetley, Carcinoma of Liver and Spleen.

Tetley, Pneumococcus Pyelitis.

Tatton, Tuberculosis.

Crow, Acute Gonorrheal Septicaemia post partum.

Brieden, Syphilis, Syphilis, Impregnage.

Sankey, Influenza, Syphilis.

In Shrewsbury Influenza, Impregnage.

In Cornell, Influenza (Relapse).

Dürfer, Enteric Fever, Biliary Colic.
Exceptional Cases.

Phosphorus deconcreases aside as being so different from the rest - either in symptoms or anatomical condition that they are not at first at least consonant with the appearances of the same disease. In Matthew's case the only exceptional point is that the atrophic change occurs in the midst of acellular trabeculae. In Wells' case, when the liver was diminished but there was acutely two decades the changes are identical with those of phosphorus liver. In phosphorus liver one can be seen by looking at the drawings by Corneil Braun, the changes are only to a certain extent the same as those in acute atrophy - though it is impossible to fix any absolute line of differentiation between them, then
is a great tendency in phthisis posterior to have fat
planted formed. This occurs also in some cases of acute
atrophy, and if we are to include these cases it had
better be classed with this group (Cheah, New. Applegard
macallum, v. Speur, Brockhill). The absence of jaundice
is not sufficient ground for excluding it with
other cases (Iwao, Schickhardt) from the series, as all
that is necessary for this is that the liver should
cease secreting bile, either by paralysis of function or
absence of the raw material. Wagner's case recalls
the description from other sections by Stoneman
by Hendrieskin. While the three cases with marked
pigmentation find a point of contact with my own.
case no. 1.
The origin of the jaundice in acute yellow atrophy is an interesting question since much of the bile duct cells are so seriously damaged at least at the end of the Morbid process. It may arise either from reduced or actually formed bile pigments or as in trypembolic in haemolysis from an excessive supply of the forma tive material. It is also known that many agents operate in producing this haemolysis especially the agents of the general infective diseases. Thus the jaundice, on the one hand, the excessive supply, on the other the preliminary enlargement of the liver do frequently found before the
elimination argues the possibility of retention of bile which is formed, and the frequent occurrence of bile-free stools is ample confirmatory evidence of the existence of this cause. The early jaundice is then clearly intelligible. Again in the form of the disease when the secreting cells of the liver are being rapidly destroyed the bile forming function will be gradually reduced and when the early destructive lives may there is the possibility of reduction in the jaundice without any improvement in the jaundice. This means less early inhibition of the bile forming function by destruction of the
Cell is very probably the reason why in acute liver atrophy the bile both of skin and urine is much less deep than in many less serious cases of jaundice.

The entire absence of jaundice could be clearly explicable by the destructive changes setting in rapidly in the liver without any excess of haemolysis and without the early bile duct calculus which is so easily produced by poisono agencies.
What then is the essential nature of the method process we have been studying and what is its position in zoology? The bulk of opinion has passed away from that which was naturally and necessarily the first position occupied that we had here a local lesion i.e. the liver and its results. It is true that even now Enchulf and others hold this position and explain the changes in the organs by the deleterious influence of the bile acids, but the probabilities of the absolute similarity of the lesions in different organs to those in the liver being a secondary consequence of
The liver changes seem very small and there is insufficient evidence that the bile acids are in all cases retained, though they are excreted. The physiological effects of the bile acids introduced experimentally are destruction of blood corpuscles and perichondromatous degeneration of the glands, but they produce no change in the muscles, unless applied directly to the muscle substance where they produce copulation. They slow the heart movements and both thorax and the direct action on the muscular tissue can be excluded, they produce apnoea, death and in the other nervous symptoms transient excitement.
increasing lethargy, convulsions & tetanus. In the
series of cases under consideration there is several
times mention of hypnopeno, but CheyneStokes breathing
to only once recorded and I find not other indication
of an apneic condition. The behavior of the heart
is very variable, in some cases as in Dr Stalke's
the pulse remaining slow until the approach of
dehis, but this is not the semisobal rule. Parenchymous degeneration we find in lungs
but not at an earlier stage than that in the liver,
and we find frequently changes in the systemic
muscles. These must then brothe influences...
work than mere bile acid poisoning, which theory moreover leaves us still to explain the occurrence of the primary change in the liver.

Bile acids however are not the only agents which produce parenchymatous degeneration in flanks, slow pulse and severe nervous symptoms and in many cases of acute liver atrophy it is possible to trace the action of such causes, and by such explanation alone can we explain the absolute equality in the extent of degeneration found in the different organs.

Acute liver atrophy then is quite probable.
remain a convenient title for a morbid process which is absolutely definite and consists in the parenchymatous and interstitial changes in the rapidly degenerative in the various organs, which has its most manifest field in the changes of colour in the liver, the largest organ, and which is capable of being set up by any of a series of causes of which each may be different but to which without being essential and for which the existence of poison disease is strongly favourable circumstance.
Note to page 176. The influence of general sthenicides.

1. Hoffmann (Untersuch. über path. und Veränderungen bei Abdominalzephalus d. Leipzig 1869) in 174 lives found
   95 with marked granular degeneration. Partial destruction.

22. General destruction.

20. Marked neoplasm.

2. Hardford. Path. Trans. Lond. 1887. 40. 129. on
   Hepatitis vi. Interic. livr.

3. Küstoff (Bolit. Gaz. 1892. 40. 34). In infected liver + liver
   Choles. formed liver cells sit back a degenerated in Portal
   Hepatic vein. Kidn. lung + kidney vessels. Extravasation
   Nordic areas in liver + lungs loc. in the vessel lumen.
Abstracts

History of cases (published since 1876)

giving Autopsy and Histological Examination

Acute Liver Atrophy

R. B. Burst MS
December 23

February 28

March 6

February 11

March 16

March 17

March 18

Christmas Eve. Slight jaundice for Sundays.

Jaundice. Toccica. Severe.

Jaundice. Well nourished. Much jaundiced

Few ecchymoses. Stunted abdomen. Kopai

Took milk well. Puffy + inutable at times

Liver enlarged. Edge firm. Muscles legs

Tender. Bowels formed one time

Inflamed, pale. Offensive. Vomiting

Non-diarrhea. T. normal

Sore and inutable

Restless. Crying. Screaming with pain

Legs drawn up. Slight convulsive


Inflamed. Tender

Stain. Convulsive movement from

Did not sleep.8 eyes open. T. 100.2

Pupils dilated, legged.

Unconscious. Stared. P. 140.0 + feebly

T. 100 at night. Tous

Convulsed in arms legs at times

Vomited yellow fluid. Stools white

10.8.2 B. am. died
Liver, 12½ oz, 37½ ft.
Neitz ochre yellow, nodules visible.
Gall bladder content ¾ oz yellow bile
Ducts patent

Ecchymoses under peritoneal covering of gall bladder
Large ecchymosis in R. Adrenal
None elsewhere.

Kidneys enlarged, fatty, uniform opaque yellow
Other organs normal.

Liver. Two normal cells containing irregular masses of bile yellow pigment, which also existed free between the cells.
Many cells became quite black with Orni's Acid.
Holes in a Tyrode
Nonirrigation of connective tissue by calcium bile ducts.
<table>
<thead>
<tr>
<th>Date</th>
<th>Event</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jan. 31</td>
<td>Ill about 14 days.</td>
</tr>
<tr>
<td></td>
<td>Acute manicidal delirium</td>
</tr>
<tr>
<td></td>
<td>Emaciated. Swelled.</td>
</tr>
<tr>
<td></td>
<td>A few petechiae, abdomen &amp; legs.</td>
</tr>
<tr>
<td></td>
<td>Muriatic. P 120 weak</td>
</tr>
<tr>
<td></td>
<td>Liver dulness 3/4 inch.</td>
</tr>
<tr>
<td></td>
<td>Coma</td>
</tr>
<tr>
<td></td>
<td>No vomiting. Anesthesia</td>
</tr>
<tr>
<td></td>
<td>Unconscious &amp; flaccid</td>
</tr>
<tr>
<td></td>
<td>No haemorrhages.</td>
</tr>
</tbody>
</table>
Brain normal

Lungs: 100g 9 1/2 oz. normal
Strait normal

Kidneys: normal 9 1/2 oz. soft

Liver: 18 1/2 oz.

Histy: Pale yellow, connective tissue: yellow colour, nowhere very deep.

Blood: No intracranial fluid yielded

Kidneys: Bile stained

Spleen: No polvonic cells

Liver: Shows extracapillary granular debris, oil globules, polvonic inclusions

No haematoxilia

Superior Cervical Ganglia:

Hardened: Chronic Acid.

Shows scattered contraction of the nerve cells similar to much fibrosis. It is usually due to the chronic acid.
Intemperate

Two days, slight jaundice,
deafness, hæmatemesis,
came as a patient with
depression + delirium leading
to stupor. T. normal.
Papillae, rigour, dilated
retention of urine.
Violent delirium
severe hæmatemesis
Death in a few hours.
|------------|----------------------|-----------------------------|-------------------------------|

- **Echymoses on thorax & intestine**
- **Stomach full of coagulated blood no breach of surface.**
- **Bile: darker fluid.**
- **Liver 6 3/8 oz. soft.**
- **Capsule unarmed.**

<table>
<thead>
<tr>
<th>Section of grey matter, lobules little marked.</th>
</tr>
</thead>
<tbody>
<tr>
<td>2. In centro lobes lobulated with patches of deep yellow soft-defined</td>
</tr>
<tr>
<td>1. Slightly raised.</td>
</tr>
<tr>
<td>No red patches.</td>
</tr>
<tr>
<td>Bile ducts patent, containing a little gelatinous mucous.</td>
</tr>
</tbody>
</table>

| A. Brown: normal cells granular. |
| B. Dem. lobules, vessels + ducts distinct. Fibrosis & infiltration, cells containing large or small fat drops, nucleus remaining. Some small round cell infiltration around portal vessels. |
| C. Deep yellow: no lobulation. Vessels & ducts scarcely visible. Cells mostly replaced by fatty detritus which may be arranged in cell-form masses. Some cells smaller & containing several nuclei, protoplasm homogenous, free from granules passing gradually to normal cells around debris. |

No duct multiplication: no microorganisms.
5 weeks. Tallowhead, without noticeable consequences.

4 days: stiff, colourless stools.
8 - retching & festiveness
vomiting blood stained

Liver dulness 4 1/2 pace - 2 1/2 lb.
Spleen tender.
Spleen enlarged 7 10 4 9
Subcutaneous injection of Quinine
8 days later. Liver less than 1 inch beyond ribs.
Next day liver still smaller
Spleen as before
Death.
Swan 4 3/4 x 3 1/2 x 1 1/4; sphelet, thickness
Capsule slightly wrinkled
Red with yellow spots

Spleen enlarged

Kidneys enlarged, cut edge pale, soft

Lungs: some atelectasis
  D. Some pin-point hemorrhages

Yellow cells, fatty degeneration
Red, normal

Histology
No microscopical

Wien med. Fürase 1878. Nr. 17
Manchester Roy. Inf. (Steel) Lance 1884 1 600.

1883

January
4  Headache, nausea, chills, fever
5  Stopped work
7  Jaundice
12  Chloroform
16  Admitted to hospital.

Semiconscious, restless, irritability, delirium.
Stupor, drowsy, headache. P. 120, R. 32.
T. 100° 2.

17  Less conscious, jaundice deeper
P. 90. T 98°. Stools watery, pale
Disinclination, slightly leached.
Vomits - Numbness, Anxa, Jittery,
Dilated. P. slow. T. 99°/104.2
Brain

Drugs seemed normal

Kidneys

Some pericardial and hilar ecchymosis.

Gastric mucosa membrane unaltered.

Liver 23 oz., wrinkled

Section looks like putty, liver fibri blodden. Two fibrous yellow patches in R. lobe.

Spleen also partly filled.

Duct patent.

Liver A. Red small. denotes distinct liver cells with distinct nuclei, others with fatty granules. Nuclei still visible, others full of fatty degeneration. Between cells a spacing quantity of connective tissue.

C. Centrally some granules, Masses of liver cells, fine fibres. A few round cells like leukocytes. Intraobiliar spaces are thickenened with increase of round spongy cells. Proliferation of bile ducts.

Intravenous peribronchial tissue, wall of arteries thickened uninfiltred with cells.

(Reinard Brown. Methylene blue; violet.

Methylene blue. show masses of large micros and in portal canals filling the arteries and in the peripheral parts apparently the capillaries between the cells. These micros smaller than those of acute poisoning, look well & retained even against acetic acid. Few micros in centred of lobules.

B. Yellow part. Cells almost completely disappeared, tubules in place, empty spaces with fibrous tissue around. Few micros.
1888

September

13. 

Sedate, depressed, abdomen distended, constipation.

15. Parents said patient forde after which the head

pressure, vomiting, dysuria.


Temperature varied. 103°/104. Abdomen tender.

18. Dehydration tender, umbilicus. Examination impossible. There seemed to be

some peritonitis above the umbilicus.

19. Despite of free use of fluids, dehydration and

vomiting, generalised daily diet of dry bread.

Moist distention of abdomen.

Slight, sleepless, complained of continual severe

abdominal pain.

20. Abdomen prominent, bulge.

Joints rigid, abdomen 5 cm. beyond ribo

Diagnosis: acute peritonitis.

23. Jaundice considerably increased, also the

abdominal distension. Taking almost no food.

Toilet, bath, bowel movements.

Eyes widely open. Features lost to distinct.

Sequel to prostration, clayey stools.

Speech confused, short, laboured.


26. Stools still clayey, somewhat feculent, bow

several times.

27. Death at midnight.
Histology.


(2) Red. Appearance overall is normal, cellules granular. Pigment granules not distinct. Red corpuscles well preserved.

Prof. Bock in Göttingen.

White red part less than in the yellow. Fat degeneration in the central part of the lobules. Melanoboles are mostly normal size, and the position of the cells as a rule intact, but some fat they are placed in a peltage.

The kidney shows slight fat degeneration within straight convoluted tubules.
<table>
<thead>
<tr>
<th>Month</th>
<th>Date</th>
<th>Symptoms</th>
<th>Temperature</th>
<th>Pulse</th>
<th>( ^\circ )F</th>
<th>( ^\circ )C</th>
</tr>
</thead>
<tbody>
<tr>
<td>January</td>
<td>21</td>
<td>Anorexia, pyrexia, hypothermia, hæmoglobinuria, obtundation</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hospital</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>25</td>
<td>Development, stern. Hypostasis</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>30</td>
<td>Hemorrhage, 35% space - 2% in pyrexia</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Slight paresis, livid, papilledema</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>February</td>
<td>1</td>
<td>External hematuria, dusky, dark, yellow</td>
<td>79.6/98.2</td>
<td>66/68</td>
<td>18.1/18</td>
<td></td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>T. 97.8/97.8 R. 66/68. R. 18/10.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>Anorexia, nausea, Sore throat</td>
<td>97.6/97.8</td>
<td>68/70</td>
<td>20/20</td>
<td></td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>Sore headache, livid. Coldness. Tender at night. Tenderness</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>5</td>
<td>Dusky, papilledema, hypostasis</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>6</td>
<td>Anorexia, hæmoglobinuria, obtundation</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Liver, muscular, hypostasis</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
BRAIN: Slight increase in size.


Spleen enlarged 4/2, dark red.

Liver: Small, esp. in d. lobe. R. lobe dark red, no coagulation.


KIDNEYS: Convoluted tubules filled with granules. Slight, epithelium straight tubes. Hyaline casts.

Liver: A. Yellow, lobules small. Fibrin granules, detritus, some fat drops at periphery. Pigment granules. Dense cells infiltrating the detritus. Intervenous vessels collapsed. Intervenous three blood. Renal tissue tissue infiltrated with cells, (as lobules large, cells small, rare vacuoles. Protoplasms more bulky, fat-drops large cells, nucleus thin cell.

B. Red. Lobules enlarged, cells swollen, pigmented, Bilirubin crystals.

Central veins especially small. Blood filling mostly small capillaries.

Blood in renal small, smaller than normal. Generally infiltrated with cells. Only once was hierachy delineated by formation of membranous Bile ducts. Branches, ascends, but with prickle from cels, without adhesion cylindrical epithelium. These show budding projection with flat epithelium. at other times duct with prolaged epithelium were found.
<table>
<thead>
<tr>
<th>Date</th>
<th>Event</th>
</tr>
</thead>
<tbody>
<tr>
<td>1878</td>
<td>Till lately very intermittent. Three weeks, light-colored urine, nausea, vomiting, weakness. Weak, haggard, flushed, jaundice. T. 100°. P. 84.</td>
</tr>
<tr>
<td>May 5</td>
<td>Came. (Death.)</td>
</tr>
</tbody>
</table>
Kabbel
24 M. 19\% hours pm.

(Smith),

Brain 9 oz.
Drops enlarged at base

Heart 7 1/4 oz.
Spleen 3 3/4 oz.
Liver 8 1/2 oz. Yellow

Capsule pockmarked
R. lobe few raised yellow patches
above portal surface of lobe.

Section: R. lobe posteriorly
Green yellow masses. Globular
anteriorly deeper. Specks of
fibrous tissue.

L. lobe green yellow homogenous
Spleen, liver, white, ducts present.

Liver, A. Red no liver structure

B. Yellow. Coarse well-formed
nucleated liver cells, huddled
Together in parallel disintegrated
Masses of fibrous tissue
Masses of yellow pigment
1878

October 20
Weakness, Headache, Anorexia

Well-nourished, collapsed.
Sharp headache. Abdominal pain.
Moderate jaundice. T. 105.8. Erythematous Tongue very dry, batting - blips
Lungs: widespread rales. Heart normal.
Liver enlarged, palpable beyond 3 cm. Spleen
Spleen enlarged 7 cm. above
No masses & palpable beyond.

After 10 p.m. very restless. Toward
Muscular twitching & tremor. Frequent
Frequent bloody diurethea, unconscious.
Stools & urine. Almost answers question.
Excited, sharp wry. Shortly falls

October 24
P. Very weak. Spleens weak. T. 102.6/106.3
Upper limbs very rigid. Active contraction
Outliers over benevolent. P. lung.
Brain: moist.

Stomach: adherent.

Liver: flat, grey, congestion free. White, pigmented patches oedema.

R. Spleen: congestion oedema.

Heart: normal.

Arterial sclerosis of mitral valve.

Liver: somewhat enlarged. Flabby. Very much bile stained; on section: some, brownish, dark, not branched parts in the yellow. Somewhat contracted, bulging yellow areas.

Spleen: dark-green, black bile.

Spleen: enlarged, two-fold, congested.

Kidneys: capsule adherent.


Liver: advanced degeneration. Fine granular detritus, fatty degeneration of fat cells. Fibrin deposits in the yellow. Strands of fibres enclosing detritus 1 have these liver cells in the red.
<table>
<thead>
<tr>
<th>Date</th>
<th>Event</th>
</tr>
</thead>
<tbody>
<tr>
<td>January 12</td>
<td>Slight jaundice</td>
</tr>
<tr>
<td>17</td>
<td>Jaundice.</td>
</tr>
<tr>
<td>19</td>
<td>T 98.4 Toraceclean, no pain.</td>
</tr>
<tr>
<td>26</td>
<td>Jaundice deeper, Jaundice less</td>
</tr>
<tr>
<td>31</td>
<td>Tinner with mental confusion, Kerry.</td>
</tr>
<tr>
<td></td>
<td>Deep jaundice. Drowsy, drowsy mild</td>
</tr>
<tr>
<td></td>
<td>Tongue clean. Appetite good. T normal.</td>
</tr>
<tr>
<td></td>
<td>Oedema, fainter</td>
</tr>
<tr>
<td></td>
<td>Tongue clean. P.T normal in seep, Jaundice deeper.</td>
</tr>
<tr>
<td></td>
<td>Difficulty to keep in bed</td>
</tr>
<tr>
<td></td>
<td>T 98.2. urea 100 mg.</td>
</tr>
<tr>
<td></td>
<td>Weight 7.825</td>
</tr>
<tr>
<td></td>
<td>Headache.</td>
</tr>
<tr>
<td></td>
<td>T 98.4. P 128. R 22. hr 110. unconsious.</td>
</tr>
<tr>
<td></td>
<td>Maltose syrup from mouth</td>
</tr>
<tr>
<td></td>
<td>Statue, Cheyne-Stokes, breathing P 120</td>
</tr>
<tr>
<td></td>
<td>T 10.2 12 2 4 6</td>
</tr>
</tbody>
</table>
|            | 103°5 104° 102°4 105° 105°
dump, hypertrophy, a few adhesions
Heart, normal. Surface ecchymosis
Spleen 5½ oz. Normal

Kidneys, one 2½ oz. deemed normal

Lungs 24½ oz.
Soft, capsule wrinkled
Sects incision, yellow patches, irregularly distributed, not connected with ducts

Spleen contained a little
Green bile
Nuclei patent

Analysis (Gumpee)

in 600 gms 7 gr Leceni
108 gr Tyrosin

Dr. Borthwick
Kidneys, cloudy, yellowish, epithelium, hyaline change in stroma
Liver, bile-ducts cysts, capsulated by microgranules

A. Yellow-paint. 1. Nive, cells swollen, nuclei ni diochich, central-fuel granules.
2. Few rows of cells with distinct outlines. Masses of fuel granules in remains of stroma
3. Only fuel granules, pigment, shrunken fibres.

B. Transition. Similar to A. But among debris are small submucous cells (leucocytes) staining well with lofwood

C. Red-paint. 1. Border outlining marked by cell infiltration around the boundary from interstitial vessels, periphery of masses of highly refractive cells in stroma
2. Nuclei arranged in tortuose channels, central fuel granules

Thyroid (a) Total vessel infiltration is leucocyte epithymic tissue. Intercellular spaces are many bile channels with bile stroma and epithelium (b) liver cells shrunken, homogeneous, without visible nuclei arranged in small tortuous tubes often becoming continuous with bile canals when cut transversely there seem like bile ducts, a circle of cells surrounded by membrane Chance from liver cells to epithelium as in A. Air can be seen (c) bile cells only fat granules, leucocytes + adynmic fibrous tissue infiltrating liver stroma
<table>
<thead>
<tr>
<th>Date</th>
<th>Event</th>
</tr>
</thead>
<tbody>
<tr>
<td>1882</td>
<td>Mother dead, neglected &amp; badly fed.</td>
</tr>
<tr>
<td>September 27</td>
<td>Fever, weakness, anemia, jaundice, constipation, vomiting. Later, right delirium.</td>
</tr>
<tr>
<td>27</td>
<td>Bilia pigment. Bilirubin stained urine.</td>
</tr>
<tr>
<td>28</td>
<td>Jaundice continues. T. 96°.4/100°.2</td>
</tr>
<tr>
<td>29</td>
<td>Less vomiting. Bilirubin stained stool.</td>
</tr>
</tbody>
</table>
| | Death.

Pendlebury Hospital

Abstracts for 1882. 45

22 hours 30m.

Bone and skin, fresh, due to fall out of bed before admission to hospital.

Lungs. L. Incapacitated. with adhesions in pleural cavities.

R. Occupied. Numerous small haemorrhages.


Spleen. 3 oz. enlarged, normal.

Kidneys. 4 lb. 2 oz. Center- pale yellow. Stools ripe from hae. Pyramids congested.

Stomach. Duodenum. Anterior perforation material, not purulent, pale yellow.

Liver. 12 lb. 10 oz. (curled).

Capsule wrinkled.

Suture lines cut back fine. Prenetum in red base.

Section. Yellow + red.


Red bloods visible, red center. pala-peripherly.

Brain. As lenticular nucleus.

Hatmotic upper convolutions.


B. Yellow. Large bloods.


Leucocytes in wall of portal veins.

Bile capillaries full of epithelium.
advanced pregnancy.

No puerperal fever or septicemia.


Heart muscle. Fat and granular degeneration.
Ctenidium

Dry 2 Aug.

Little yellow substance

Red. 900 cells only. Branchlet detritus
<table>
<thead>
<tr>
<th>Date</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>13</td>
<td>Abdomen normal, liver dull.</td>
</tr>
<tr>
<td>14</td>
<td>Face colourless. Markedly slept.</td>
</tr>
<tr>
<td>26</td>
<td>Blemish could be a scar. Documented.</td>
</tr>
<tr>
<td>27</td>
<td>Abdomen fully distended. Stomach full, no air. Eyes, hands, face, etc. Knees drawn up. Light trauma.URYING ORDERED THE FOLLOWING TREATMENT AMONG THE TREATMENT RECOMMENDED.</td>
</tr>
<tr>
<td>28</td>
<td></td>
</tr>
</tbody>
</table>


Kidney normal or, session: especially in helix along convoluted tubules. Medulla on tubular tissue normal.


Kidneys: 149/4 oz. On the mesentery wall marked.

Liver: pneumonia, many white spots.

Bladder: Paper thin.


Abdomen: dry.


Yellow spots from any size on dark ground reddish. Bowman's hollow. Burettes in yellow part larger, not definite, in red part definite. Larger tumescent, edges grey, centre red-brown.

Gall bladder: contents a little bile.

Stomach: very acid.

Rat: white warts.

Lungs. Firm spots are haemorrhagic.

Trachea: wide tubular shape.


Adipose tissue normal.


B. Sick, blood, sparry, fine, vascular or fat.

Lumen: thick, uneven, or fat.

Intestinal lining increased. Mostly by leukocytes infiltration. Intestines normal.

Some highly affected places shrunken.

Muscle: white, not yellow. Adipose: yellow.


Stomach ducts, postmiliar, fine pieces of cells and villi. Leucocytes.

Brain: Blood vessels connective tissue, fine granules, few cells, no haemorrhage.


Transcutaneous crys.

No pigment crystals.
Shopkeeper

Death after 8 days illness

His
Besides well preserved epithelial, fatty cells others in granular or fatty degeneration, especially in the center of the lobules.

Red. Changes more advanced
Receptors begin to disappear, capillaries with distinct nucleated epithelium in the region of cellular infiltration. In well preserved part extension of bile ducts

Microorganisms in ducts or vessels.
1887

October 7

Anorexia, yellow skin. Abscess also in right
Spitzage, continuous vomiting. Ipecacuanha, Ether.

23

Mustard stopped.

25 Pari in cap. Back of head, Gally. Restless
Several short attacks of unconsciousness.

Tantalus deeper. Forbade water.

July 7

Unconsciousness near, touch.
Vomiting, deep respirations.

27

Hospital. Unconscious. Very restless
Jumps of one moment to other.

28

Recurrent feeling of unconsciousness.
R. 34
T. 96.8 (Rectum) P. 76. Strong, deep.
Expression passed. Pupil dilated less
Sensitive.

28

Tonic delirium.

28

28 1 A.M.

28

Contraction in the upper limbs
R. 36. P. 7th.
?

Pupil insensible.

30/40. P. 140.

Death

B. Red. Vessels filled. Liver not only fatty, detritus & pigment. Glisson's capsule, and cells partly in small cell infiltration. partly from proliferation of duct epithelium as long tubes with large cells, having finely granular protoplasm. Very like normal liver cells.

Stomach. Small cell infiltration in the interstitial tissue of the mucosa.
November 22
Admitted to Syphilis (Vesicle) Hospital with Leucopenia. Infected with Levisi major populer by syphilitics and some uncoited fluence. Two had same syphilitic pain + sleeplessness. 3. Normal. 4. Infected Eburnation. After 15 minutes had a little montenimation. Untied for 2-3 days then returned. After 42 minutes the had some symptoms of Adenolb pain + rect. No sweating; floor in between project beyond 96 ambrosia. Some lassitude at other cases well, till Grew weaker. 5.96 Pnum. Pupils slightly dilated, rectified.

February 7

February 8
9/98.3 Pnum. 100. Nasal weak slanting. Dosed till morrow Retd to Commune Hospital.

February 9
102 Retd. Deated second time instructed.

February 10
Heart wall, soft, myocardium yellow fatty.

Lungs. Hypostatic pneumonia in lower lobe, edema.

Liver 35 oz. Red with heparin. have small yellow spots. Thymus in capsule, capsule wrinkled. Tissue jelly firm but bile gall bladder content; a fluid-like fluid, containing epithelial cells, not pus cells.

Spleen, increased.

Kidneys normal size, yellow. Cortes, enlarged, pyramids infected. Echurns to internal, brain normal.
December 15

Indian purulice, carotid, aortic

Pulm. moist and s. belly

Vomiting, regurgitation of food.

Inbred, wind affected, constipated

Skene somewhat enlarged, heart cleared

Bladder hic labeled. No passage.

Pupils dilated. Vascula Knei rapid

Increased. Acute anemia. Triticum

Nipidity of lower limbs, to that passive

Movement are difficult. Sensation

Kricking. Sometimes restless, loosing

Speaks incoherently, talks to her mother.

Tied movement with difficulty from

R. 30. Scler. P 60. T. 96.8

T. 98.6/196.5 P. 72. 120. Found rice deeper

Loo. T. 6. Not palpable. Constitution is

Obviated. More still.

T. 98.6 P. 140. Sincerely perceptible

Hematemesis. Desperation. Microcosm

Stool pale yellow, no pupils Nattiez. R. 40.

Faint hemorrhage from nose.

When

Died at 70 a.m.
Potbelled, flattened pericardium
Lungs, congested.
Bronchial lining dark red.
Heart, normal size.
Aorta: yellow. 3-6 strokes.
Small ecchymoses on peritoneum.
Section: yellow, bile marked.
Red, firmer. Whole surface, red and white, diffusely stained by blood, gray.
Spleen: density firmness.
Duct: patent.
Spleen: retracted liver.
Soft, grayish.
Kidneys: enlarged. Soft, costly, broad.
Such, mucous membrane thick. Soft.
Blood: slightly yellow, saccus.
Brain: soft and pale. Irreducible dilated.

Cellular infiltration: yellow, mucosa, mucus-like.
Kidneys: salt, change; epithelium.
Liver: fresh. Granules drops.
Section, bundle fibres filled.
Fibre between the capillaries.
Mucous, preserved cells; yellow.
Fibrin, small, blood, infiltrate around the portal canal. Epithelium capsule increased.
Three chambers, mucin, and
Small accumulations of mucoid tissue.
Cells (2. Repetitieux).
Isolucini: hyposis, vitalicity.

Bacteriological investigation:
Bacteria only once seen (Gram stain).
Cultivation yielded only innocuous organisms.


February 5. Removed inspissated feces.

February 7. Suggestive signs of ascites. Reduced liver and spleen. Fossa iliaca and fossa of the ilium.

February 8. Palate small, frequent. Died 6 a.m.
Rheumatic fever, endocardial endothelioma.
Heart muscle softening, heart more yellow.
Spleen 5x2 x 3 cm. pale red. soft.
Kidneys normal size.
Liver 3 kg.3 g., capsule unbroken.
Spleen yellow to greenish yellow.
Adipose part soft. liver lobes red.
Kid. yellow, red.
No bacteria.

Rosenheim. 10.7.
**1891**

June 14. Went to visit the scene of an accident. Collisions with morning. After leaving home fell sick. Vomited several times, jaundice.


27. Jaundice, yellow, bile.


Jaundice two attacks of articular rheumatism.

<table>
<thead>
<tr>
<th>NAME</th>
<th>Burschardt's Case</th>
</tr>
</thead>
<tbody>
<tr>
<td>ADDRESS</td>
<td>Basel</td>
</tr>
</tbody>
</table>

**POCKET CHART.**

FOR BEDSIDE CASE TAKING.

Compiled by ROBERT SIMPSON, L.R.C.P., L.R.C.S.

John Wright & Co. Publishers, Bristol.

COPYRIGHT.
<table>
<thead>
<tr>
<th>DAY OF MONTH</th>
<th>16</th>
</tr>
</thead>
</table>

**Fahrenheit Scale**

<table>
<thead>
<tr>
<th>DAY OF DISPOSAL</th>
<th></th>
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</thead>
</table>

<table>
<thead>
<tr>
<th>Pulse</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Resp.</td>
<td></td>
</tr>
<tr>
<td>Motions</td>
<td></td>
</tr>
</tbody>
</table>

**Remarks**

- **June 1891**
- **Temperature Chart**
- **24** 26 27 28 30 1
- **Pulse**
- **Resp.**
- **Motions**

- **Remarks**: 
  - 80
  - 76
  - 80
  - 140

- **Pocket Chart**
  - For Recording Disposal
  - Compiled by Robert Simpkin C.R.G. L.R.C.P.
  - Copyright 1891
Liver 33 1/2 oz.
Saffron change to red atrophy in liver.
Fatty changes in brain, kidney, skeletal
muscles.
Ecchymosis in serous membranes.
Hemorrhage in stomach, diaphragm,
ovaries, uterus + tympanitis.
Multiple hypostatic pneumonia.

Liver A. Yellow. Cells swollen.
Fat droplet large + small.
B. Red. appearance only,
detritus of small brown fasciculi.
Brain kidney, spleen, in fat
Gastric mucous membrane coated
Swelling.
<table>
<thead>
<tr>
<th>Date</th>
<th>Spinal</th>
<th>Jaundice</th>
</tr>
</thead>
<tbody>
<tr>
<td>June</td>
<td>Yes</td>
<td>Jaundice</td>
</tr>
</tbody>
</table>
| June | Jaundice proteinuria. Stools pale
      | Diminished liver dulness |
| June | Enlargement of spleen. No anemia |
| July | Fatigue, weakness, no pain, no vomiting |
| July | P. B. 7. 98% no nervous symptoms |
divir 15.0g

Zellwgr. mit Atrophie

Schildr. disseminated with pus

Duct, healthy - patent

divir. 4. Yellow parb. Complete granular degeneration

B. 4th part. Marked increase of myogenic interlobular tissue

Multiplication of small ducts

No regeneration of cells

No micrococci visible.

Schildr. Bacilli in the pus, wall infiltrated with leukocytes.
<table>
<thead>
<tr>
<th>Date</th>
<th>Events &amp; Observations</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 Jan</td>
<td>Cold from exposure</td>
</tr>
</tbody>
</table>
| 4 Jan | Influenza, skin yellowish, 
|      | liver, brain, 
|      | deepening, 
|      | hepatic pain, 
|      | apathy, stupority |
| 31 Jan | Hospital, jaundice deep, 
|       | jaundice, 
|       | blood, 
|       | Pupils: small, active, 
|       | stupor, aphasia, 
|       | P: 76, T: 97.4, 
|       | Spleen: 
|       | mob margin, 
|       | Liver: dulness 
|       | 1in above rib, 2 in. 
|       | Costal pain: nausea, 
|       | vomiting bilious nausea, 
|       | Somnolence, delirium, 
|       | vomiting, 
|       | Coma, persistent 
|       | Pupil: small, active, 
|       | Nostrilness: diminished |
| 2 Jan | Death. |
Toronto General Hosp.

24 hrs. pm.

The man. Felt dead a few days.

R. Colitis.

Spleen large, soft.

Kidneys. Artery enlarged.

Liver 25 cm.

Haemorrhages.

Dark red, with yellow patches.

A. Yellow. Bushy preserv. under capsule.
Cells normal, intimate, in their granular
Vacuolated with fat. Separated; bile
with leucocytes. Formation of endothelial cells.
Gall capillaries enlarged. Karyokinesis frequent.
In nearly normal cells.

Infarcted vessels. Cells eosinophile or enlarged.
Concentric tissue necrosis and villous, apparently fully enlarged.

B. Red. Tumors much containing
Granular: necrosis of endothelial cells.
Cells lining vessels (villi). Infarcted. Enlarged granular, radially stained:
May lie free in the lumen. Blood
Arterioles in liver lobules venous.

In microorganisms:

1. Cellular elements inside hepatic
And endothelial cells, in yellow pods.
Strongly eosinophile, spherical,
Separated by tone from protoplasm.

Inclusion Cell. [Lecyto, hypostasosis]

2. Endothelial cells may be traced
In lesions from flat cells to large
Cubical cells with lobulated nuclei.

Invasion of the margins of lobules.
1882

November

22. Lancets from Esthbro Country, 3 weeks ago, slightly out of health; with no definite complaint. Appetite good.


Urine

30.03.

Darker color.

30.04.

Little albumin.

30.07.

Little pigment.

30.08.

Slight pyrexia.

2.00 p.m.

Diabetes.

11.00 p.m.

11.00 p.m.

Stools.

24.

Slight pyrexia.

30.09.

1.00 a.m.

30.10.

Died at 4 a.m.
Bladder. Normal.

Kidneys. Fig. 1703. Arteries much thickened.

Liver. Advanced atrophy, notably in cells near granular masses or much shrunken, in definitely scattered or oblong, fiballinar spongiiform or aggregated.

No increase of fibromatous fibrosis. Portal canals normal. The fibular matrix seems to consist of shrunken capillaries, of which nuclei cannot be distinguished. No bile ducts to be made out clearly except in portal canals where the epithelium is thinned, a distinct after necrocyte, scattered here and there, not enough to substantiate a theory of inflammation. Tissue bloodless. Bacteria found.

Kidney. Cloudy swellings. Healthy. Fatty degeneration of convoluted tubules. No nuclei to be seen. Some change less marked in straight tubules. Some nuclei seen.

Pants. None. Fatty, fatty and granular degeneration. Striated, well marked.

Stomach. Salem. Malpighian bodies marked.

Duodenum. Scattered obturated cells near granular masses or much shrunken, in definitely scattered or oblong, fiballinar spongiiform or aggregated.

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<th>Date</th>
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</tr>
</thead>
<tbody>
<tr>
<td>Jan 31</td>
<td>Diagnosed with dyspepsia. Face was yellow and sunken from fever. Headache.</td>
</tr>
</tbody>
</table>
| Feb 1 | General malaise, marked jaundice, 

TJ: 78.8, CR: 88.8; T: 99.8. Instestinal bladder, bile free. |

Became very prostrate, delirious at night, vomited food. |

Mar 16 | Boneyel found, Peritonism, on Billy's arm. |
|        | Note: |
24

Middlesex Hosp.

350 c.c. urine, albumin, pus, some red blood corpuscles and projecting large yellow urachal tubules.

Subcutaneous little brown bile.

Peritoneum about 400 c.c. fluid.

Some grey tubular formations on test tubes, surrounded by pigment.

Stomach content altered blood, haemorrhagic stools.

Small quantities of ascitic fluid.

Subcutaneous tubercles.

Heart and lungs ecchy mic.

Kidneys enlarged, cloudy, pouting.

Spleen small.

Dr. A. Red. Reticulated connective tissue with many nuclei.


At magnum spaces from Annulus of luga cells.
1884

November 13

Purging, turns of hands, especially
when sitting, bold came on suddenly

Pallid, conjunctivae became yellow.

Vomited, lay down, answered
no to all questions. Skin bright, face
blisters, difficulty of respiration

Vomited

Incontinence of urine

Vomiting, tongue white fur.

Incontinence, headed, violent
spasms from anorexia, faint

Slight than two deceased, in last
death.
March. Coffee ground, material
Scrap. Studded with hemorrhagic patches
Nek. 2 oz. Slin
Endocardial petechiae at L. Ventricle.
Stomach content. Coffee ground fluid.
Liver. 7 lb. Slin
Vesicle
Kidney. 17 1/2 oz. Enlarged. Full of Blood.
Duod. 3 1/2 oz. Smooth.
Pan. Yellow, yellow, yellowish
Protein. R. live mass, yellow
mass. Whites white, loose, jelly
Rest Other. Brown dense tissue
Hemorrhage. Many points & streaks of fat-staining tissue
Pelvis. Bile, orangish brown bile
duct. Patent.

Liver. 2. Yellow, cells normally
arranged. granular or fatty.
3. Red: largely distended nuclei
4. Distinct, discrete, pericellular
5. Hepatocytes, vacuolated
6. In parts, vascular
7. Tissue, subnuclei, granulation tissue.
8. Distinct increase of fibrous
traces around portal canals
9. Cell periostitis around others.
Many duct formations outside.

Report: Committee
"Red atrophy of British Autism.
Identical with R. Taylor's case"
February 16th: Febr. well, complained of headache.

February 15th: Sudden left leg pain. Doctor strongly advising immediate return to hospital.

11:15 AM: Acid stomach, much discomfort to abdomen. Much fever present.

February 14th: Pupil median, normal. Moderate pinnae. Deep respiration.

February 13th: 79.3°F. Quiet night, patient very restless. Fever.

Pupils equal, dilated. Abd. membranes tended.
Liver: not adherent. Left congested,  
its border lost posteriorly and two patches  
red-brown and distinct air. In right  
such patches are numerous. Hard normal.  
Kidneys: Renal walls well. Capsules  
adherent at points.  
Stomach: distended, contained about  
white dark-brown watery fluid.  
Intestines: mucous membranes pale.  
containing parts chymous, partly furred  
dark-brown mucus.  
Brain: membranes. Considerable atonia  
and moderate oedema.

Brain: no hair cells, but only  
smaller large fat granules, and  
thin fine detritus.  
Stomach: beautifully marked with  
fine points, without alines of  
finetissue.  
Heart: muscles, yellow, no atriation.  
perforated by fine granular  
empty details.  
Voluntary muscles. Presence of tail  
degeneration.
Some symptoms: dyspeptic symptoms
then vomiting, diarrhoea, anorexia
facial edema, coma, contractures of joints
incontinence of urine and other nervous
symptoms. 

Diagnosis: Brachial.
Blocky acini: no desmoses present.
Great increase of Paneth's cells, degeneration into "intra-lobular accumulation of amorphous cells.

31/4/203

Red cells present: tough, no distinct lobules,
with yellow nodules, soft.

Liver is yellow. Hyperplasia of Paneth's cells.
Some mitotic figures change atrophy of cells, increase of connective tissue.
No biles, no bile-ducts, no fat granules.

B. Red. Round cellular connective tissue, near the portal canals.
Branched tubers with epithelial lining.

Which the thins denote:
Degeneration of the liver substance.
26 J pregnant 5 mo.

Eclampsia, first pregnancy.
Gratuitous fear of this pregnancy. Tried to reduce abortion.

Slight cutaneous jaundice for several weeks.

Six days. Dealt.
Spleen slightly large

Kidneys enlarged, congested
Arteries enlarged.

Liver about 1/2

Hasy. Capsule wrinkled

Redo yellow.

Stomach: Not seen or yellow

Hepatic veins look dark red pruny.

Kidneys: Chronic desquamative nephritis with congestion.

Dwarf: Cells much shrunken, albumin, oil globules (2) at periphery, shrunken cells, oil globules, free nuclei; lymphoid cells (3) cells entirely destroyed (4) lymphoid infiltration at periphery

Infiltration is greater in portal zone.

Sometimes amoeboid forms connective tissue; cells nucleos affected in portal zone.

Blood corpuscles: Some intra-lumen meshes, intercell vesicles.

Wall of hepatic artery thickened.

Duct filled with epithelial cells.

Thickened, not evidently multiplied.

Tissue of cups take hole.

Not very

Microscopic: ni dome veno.
Liver enlarged 2x. 63/203.

Dark yellow.

Full bladder content scanty, duct empty.

Phrenic nerve muscles prominent.

Kidneys: cloudy.

Liver cells in parts destroyed, and in these parts interstitial growth arranged the fat cells devoid of numerous micrococci. The diplococci found are very disordered in the liver parenchyma.
February 1884

8 months, slight, Vicuaria, jaundice, no pain. Child: melena, vomit;

Syr. c. Cist. for 6 weeks.

March 1884

Tachicardia, deep, xeropus. no appetite.

Rectal: dilated, opaque, vulva.

Bowels confined, stool, liquids, no appetite.

Syr. gent. 25 cc. R. lobe mid, lymph nodes.

25 cc. R. lobe mid, lymph nodes.

(Nept. Ameliora. 5th. & 6th. M. Osier.)

Reinforced, bowel haemorrhage.

26

Vesicle above haemorrhage,Jaundice less

(Syr. Cist. 5th. & 6th.)

28

Jaundice.

April 1884

5

Vomited 41 blood. Sore lachrymal.

17

Dehydration.

20

Vomited 4 times. Death.
Vena

Victoria St. spat. Chelsea

July 1886. 2. 191

Bleeding. raw. mount. section preprepa.

Head 44 cm adherent.

Drops congested

Brain normal

Spleen 144 oz. hard.

Stomach cedemus premorna fluid

Liver congested

Kidneys 2 1/2 oz. congested

Liver 10 1/2 oz.

R. lobe large. Mucous with yellowish tincture

L. lobe yellow. Generally

duo cells much atrophic and contain granular debris. In many on this i vague. Nucleus indistinct. There are fleeces or nodules by small roundles. There is interstitial hepatitis and yet at this stage of infected tissue in these partly many bile capsillaries no microorganism.
1882

June 6

Unsic. Much bile pigment in stool.

14

Consciousness impaired. Some dyspnea. Patient breathes more rapidly.

21

Death.

Brain: Fatty degeneration.


Spleen: Enlarged; capsule thickened.

Liver: Small, flat, soft.

Surfaces: Bunched, pinkish.


Thromboses: Smallui. Mucosa major.

Knee: Haemorrhages.


Liver: Hypertrophic. Left lobe.

Brain: Right oedema.


Kidneys: Fatty degeneration. No interstitial infiltration.


Liver: Cells almost vanished. Intestinal tissue cells, cells with acini formed.

Kidney: Tubular structures.

Brain: Cells probably collapsed.

Thalamus: Brain: Cells, apparently multiplied. The capillaries: Presently collapsed. The tissue then an accumulation of small cells.

In the left kidney, we can trace the gradual increase of the interstitial tissue in the lobules and the destruction of the cells from within without. The tubules: Central lobe: Rich in cells, blood vessels, blood vessels. The interstitial tissue: Rich in connective tissue, blood vessels, blood vessels, blood vessels. The destruction due to hypertrophy.
<table>
<thead>
<tr>
<th>Date</th>
<th>Event</th>
</tr>
</thead>
<tbody>
<tr>
<td>1890</td>
<td>December, 1890 influenza, not quite well again</td>
</tr>
<tr>
<td>July</td>
<td>22 Red rash, all over. 14 days headache, anaemia, faint forenoon.</td>
</tr>
<tr>
<td>August</td>
<td>13 Resumed work 9-10 days.</td>
</tr>
<tr>
<td>September</td>
<td>sick. Meningitis. Vomiting, continued stomach ache.</td>
</tr>
<tr>
<td>October</td>
<td>3 Food eaten upper part abdomen.</td>
</tr>
<tr>
<td></td>
<td>Hospital. back redness patches on necks arms, chest, upper extremity</td>
</tr>
<tr>
<td></td>
<td>Anorexia, thirst, sore tongue. Sore throat.</td>
</tr>
<tr>
<td></td>
<td>Liver, left lower ribs. Spleen not palpable.</td>
</tr>
<tr>
<td>November</td>
<td>1 Evisceration on 30th</td>
</tr>
<tr>
<td></td>
<td>Semicereiform. Death.</td>
</tr>
</tbody>
</table>

Leucemia, Synovitis not found.
Stomach, upright, filled with dark, mucous, pseudomembrane and haemorrhages.
Large bowel injected. Faeces pale grey.
Liver 31 oz. Fini.
A lob. brownyellow, fleshy, anteriorly. Red, coagulated Portal sinus free from mucus
Ach, patent
Spleen 10 oz. Fini.
Pancreas firm almost cystic masses.
Kidneys (each 7 oz) firm.
Lungs: Paridiia pleura collapse anteriorly.
Emphysema
Heart normal.

Liver: Scarring. Mitochondria advanced with degeneration of crista.
Sections: Fibrin network rich in small nucleated cells, haem.
Thick layer of round cells.
Impart lobules with fatty, cells dense, round-celled infiltration
Pancreas fibrous. No new growth.
Kidneys no thing of note.
<table>
<thead>
<tr>
<th>Date</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>January 1</td>
<td>Influenza.</td>
</tr>
<tr>
<td>January 13</td>
<td>Repeated.</td>
</tr>
<tr>
<td>February 20</td>
<td>Albumen.</td>
</tr>
<tr>
<td>March 25</td>
<td>Albumen.</td>
</tr>
<tr>
<td>March 28</td>
<td>Albumen.</td>
</tr>
<tr>
<td>April 1</td>
<td>Albumen.</td>
</tr>
<tr>
<td>April 5</td>
<td>Albumen.</td>
</tr>
<tr>
<td>April 11</td>
<td>Albumen.</td>
</tr>
<tr>
<td>May 17</td>
<td>Albumen.</td>
</tr>
<tr>
<td>May 17</td>
<td>Albumen.</td>
</tr>
<tr>
<td>May 17</td>
<td>Albumen.</td>
</tr>
<tr>
<td>May 17</td>
<td>Albumen.</td>
</tr>
</tbody>
</table>

1890

- Fever, subject to brain attacks, no fever.

- January 1: Influenza.
- January 13: Repeated.
- February 20: Albumen.

---

**Mr. Connell**

- 51 F widow

---

**Montréal Med. T. 1892 (April)**
Not macerated. Ascites & fulness.
Liver 38 oz. Haemorrhagic yellow-brown
Ranke's white spots.
Duct patent.
Spleen 67/2 oz.
Kidneys: Slightly congested.

Liver. Marked hyperplasia. Connective tissue especially around portal spaces, containing vessels & bile ducts. Some cells normal or atrophic & surrounded by white fat or mottled. The cells contain bile pigment but no crystals of haematochrome. The whole arrangement of cells is completely obliterated.
1876

August 29

Accident: Asp. Nick worn three times
Tennis injury. Painful injury. No swelling. No bruising. No atrophy of jaw

Sept 1

Amputation done. Relieved from pain

Spleen hard. No albumen. 17.5. 9 p.m. 96 Bp.

Muscles firm. No loss of weight.

Serum: dark. No distention. No albumen.

7.

Serum: dark.

13. Left side of body.


18. Tongue dark. Food well

Tennis injury. No albumen. No fever.

Spleen hard.


20. Urine yellow.

21. Died at 3 am.

Temperature normal. Abdominal

Urine very abundant.
Liver - smooth surface. Yellow nodules of fatty infiltration.

Spleen - small, white fibrous patch on surface.

Kidneys deep brown, affected.

Stomach - normal.

Lungs -

Brain -

Liver, decreased size due to tissue fibrosis. Small cells disorganized replaced by yellow brown granules.

Semen: globules.

Cystic-like haematoxilin.

After hardening with phosphoric acid, no semen or crystals.

Stroma became a small, cell-like structure-like an adenoma.

Liver cells became more distinct.
### 1883

<table>
<thead>
<tr>
<th>Date</th>
<th>Event</th>
</tr>
</thead>
<tbody>
<tr>
<td>March 11</td>
<td>Faint. Had cold and weak feet. Feeling ...</td>
</tr>
<tr>
<td>March 20</td>
<td>Hospital. Deep faint. He had .......</td>
</tr>
</tbody>
</table>

**Diagnosis:**
- Fainting
- Cold
- Weakness
- Diaphragm
- Spleen
- Mental confusion
- Palsy
- Nails plugged
- Fullness
- Kelin
- Not able to speak
- Pupil dilated
- Swollen:
  - Face
  - Eyes
  - Navel
Liver. 4 9 oz. Weight.
- Dark yellow copper colour mottled.
- Lobules distinct, separated by grey substance.
- Gallbladder distended, contains yellow fluid.
  Ducts patent.

Liver. Domes surrounded by connective tissue. No sili.
- Fibro liver.
- Cells distinct, visible. Nuclei visible.
- Hepatic ducts numerous. No haematoxilin.
- No typhoei.
- Spots that might be groups of microcoeci.

Inspection. Bilious Currants.
<table>
<thead>
<tr>
<th>Date</th>
<th>Events</th>
</tr>
</thead>
<tbody>
<tr>
<td>July</td>
<td>Grippe</td>
</tr>
<tr>
<td>March</td>
<td>Jaundice, which diminished (5 weeks well)</td>
</tr>
<tr>
<td>September</td>
<td>Hospital, fitful, headach, no confusion, delirium, late, mild delirium</td>
</tr>
<tr>
<td>October</td>
<td>24 - Violent delirium, urination, unconscious; 25 - Stool, delirium</td>
</tr>
<tr>
<td>November</td>
<td>30 - Temperature, sudden fall, clayey stool; 31 - Coma, 2.41 acidosis, stools, sweating</td>
</tr>
<tr>
<td></td>
<td>Death</td>
</tr>
</tbody>
</table>
Dung, Angina, Hypotonia.
Old & pleural adhesions.

Knee: Soft

Pustule on skin.
Spleen: 94oz. firm.

Liver: 67oz. Surface green.
Substance: Green yellow.
Gallbladder: Distended.
Bile: brown, normal.
Duct: Normal.
Intestines: Normal.

Liver: Micronodular, tumour upon embryonic cells displaced, pigmented, focal granular affectation.
1881

April 11

History of fever, vomiting or alcohol.

April 11

Eighth day, anorexia, malaise, typhoid fever. Nausea, vomiting.

April 11

Fifth day, agitated, slight night delirium.

April 11

Duration of delirium. Bitting, Sentini, under chlorplam.

April 11


April 11

P. 78. Kentn. Deprive, retarded, insensible.

April 11

Eyes continually rolled.

April 11

Eye involuntary rolled.

April 11

At 6 a.m. to lower deck.

April 11

Skin red and felt.

April 11


April 11

Reddened, jaundice (icterus). Body of abdomen.

April 11


April 11


April 11

10.30 a.m. T. 105.4

April 11

Exhaustion till

April 11

Slight at 3:30 p.m.
Liver 32 oz.
Capsule thinned, cord, lobes.
Under part of liver much decomposed.
Section, white, red yellow with black red spots.
Lobes, pure yellow.
Habbly mixed in B. lobes.
Scleromec, recrunt positive.
Damp, blood stained fluid in pleura.
Scleromec Engaged.
Kidneys Engaged.
Brain 31/2.
Heart soft friable.
<table>
<thead>
<tr>
<th>Date</th>
<th>Event</th>
</tr>
</thead>
<tbody>
<tr>
<td>July 10</td>
<td>At breakfast pain in living and improper ileus.</td>
</tr>
<tr>
<td>July 11</td>
<td>All sellow, nausea, no pain</td>
</tr>
<tr>
<td>July 13</td>
<td>Jaundice had increased, sent to hospital.</td>
</tr>
<tr>
<td>Aug 1</td>
<td>Skin hot-dry, with clayey odour, 98°. P. slow, full. Abdomen</td>
</tr>
<tr>
<td>Aug 2</td>
<td>Hepatic pain, liver dulness</td>
</tr>
<tr>
<td>Aug 3</td>
<td>T. 99°. Slighter liver dulness in right upper abdomen</td>
</tr>
<tr>
<td>Aug 4</td>
<td>T. 99°, P. rapid, full.</td>
</tr>
</tbody>
</table>

**Test:**
- Adrenaline
- Albumen: traces
- Bile pigments: slight
- Renal: traces
- Typhoid: deposit
Face shows scar like lupus

Dwgs. basal congestion

Spleen enlarged

Kidneys

Liver 34 oz.

Fatty, friable.

Olive green.

On section:

R. lobe - Bullred with green yellow patches. Red parts more abundant - firmer

L. lobe - No bulding

Surface green - yellow

Ecchymoses under dermis surface.
<table>
<thead>
<tr>
<th>Date</th>
<th>Events</th>
</tr>
</thead>
<tbody>
<tr>
<td>July 15</td>
<td>Diarrhoea; chlorosis for renal colic (33)</td>
</tr>
<tr>
<td></td>
<td>Has been feeling badly in breast.</td>
</tr>
<tr>
<td></td>
<td>Chill fever; 103.5°; haemorrhaging from the back, chest, painful. 99°</td>
</tr>
<tr>
<td>28</td>
<td>Pneumonia; breathing antine.</td>
</tr>
<tr>
<td></td>
<td>Vomiting, haemorrhaging stool; 103°; dyspnoea; slight oedema</td>
</tr>
<tr>
<td>29</td>
<td>T. 98.5°; least nausea.</td>
</tr>
<tr>
<td>30</td>
<td>Hands cold, slight deep.</td>
</tr>
<tr>
<td>31</td>
<td>Slight, tremulous; hands deep.</td>
</tr>
<tr>
<td>Aug 1</td>
<td>T. 97.5°; lice; dullness less (very fat)</td>
</tr>
</tbody>
</table>

**Aug 1**
- **Death.**
39
Man
M (Michael)

Lumps. Hypostasis

Stomach content dark fluid

Kidneys. dark. Spleen hemorrhaged.

Liver, 31 1/2 oz. soft.

Dura. Normal structure. Some connective tissue fibrinocoels, oil globules, traumatic detritus.
17 days before death. Malaria. Anemia.
Slight jaundice.
T. 99° 3. Liver dulness 1/2 in. beyond ribs.
8 deep lata. Liver not felt. Spleen large.
Conscious. Unconsciousness.
Spasms. Tonic delirium.
Convulsions.
Health.
Hemorrhage under serous membranes

Lungs: edema

Spleen

Kidneys: necropsis

Liver: venules small

Intracerebral hemorrhage.

Red spots: not palpable. Disappearance.

Bile ducts: patent.

Muscle: slight degeneration.

Kidneys: cloudy swelling.

Some oil drops.

Liver: cells filled with fat.
Duckworth 8 St Bartholom's Hosp. Laneet 1892. 1. 630 F M

1891

Sepr 26 Suffered yet another Child almost delivered.

September 27

Vomiting - Mucous Bilia continued

Oct 9

Semi-l. Hypochondrium
growns. Pressure in head.

20

Hospital. Biliary. Answer delayed.

23 P. Ven. R. normal. Tom's normal.

Dec 26. Sphincter hard

Unconscious.SX. 1872.

Die pigment.

24 le. albumen.

Bilirubin. SX. 1072.

25

Alkali.

Unconscious.

Chlorides.

Flexibility.

Presence of albumen.

Bilirubin urine no. lust.

Analysis.

1670

P. 120. Tense. R. 60

Sphincter loose but normal.

Liver dullness 142 in umbilical 60 rib.

Splenec at all time line.

Death.
Liver, hemoglobin: 75% normal. 

Heart: Some atheroma. 

Spleen: 503. Diff. different. 

Kidneys: Natural size. Linty-thin, no ischipe. 


Spleen: Yellow patches in red. 

Kidneys: Interstitial fibrosis change. 

Liver: Cells mostly disintegrated. 

Renal cells swollen and shrunken. 

Round-celled infiltration round portal vessels. 

Acetate (in fresh section).
19

Parent's daughter.

1. Sudden nausea, vomiting, pain in abdomen. Vomitus faeculent.
2. Sudden unconsciousness.
3. Stomach rigidity and tenderness.
4. Pupils equal and active.
5. Acute delirium. Turning of eyes.
6. Right swelling of spleen.
7. Death.
Heart large flabby.


Spleen enlarged.

Kidneys: Eventually yellow.
Small echymoses.
Also in duodenum and mesenteric hiccups.

Liver: Normal size.
Odour yellow, light at lower edge.
Centre of lobules yellow. Pronounced periphery grey. Margin in a few part periphery red (from extravasation or infection).

Brain: oedema and anaemia.
1885

August


2. Slight fever. Urine dark brown, but no albumen.


September

5. Slight fever. Urine dark brown, but no albumen.


7. Slight fever. Urine dark brown, but no albumen.


10. Slight fever. Urine dark brown, but no albumen.


17. Slight fever. Urine dark brown, but no albumen.


Edematos aspectus general

Heart, fatty 703

Spleen, enlarged left.

Kidneys, fatty, artery, enlarged

Periarticular deformity

Alimentary canal, normal.

Liver, 30 1/4 oz.

R. 905, many yellow spots

L. 750, few - -

Spleen, over 1 1/3, friable bile
February

   Stools: regular.

20. Temperature steady, 99.6°F.

22. Jaundice distinct, yellowishbrown in color.
   Stools: clay-colored.

24. No rales, rales at base.


27. Jaundice, icterus.

March


3. Jaundice.

4. Restless, fever.

5. Jaundice.

6. Jaundice.

7. Jaundice.

8. Jaundice.


10. Jaundice.

11. Jaundice.

12. Jaundice.


15. Jaundice.


17. Jaundice.

18. Jaundice.


20. Jaundice.


22. Jaundice.

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15. Jaundice.


17. Jaundice.

18. Jaundice.


20. Jaundice.


22. Jaundice.

23. Jaundice.

24. Jaundice.

25. Jaundice.


27. Jaundice.


29. Jaundice.

30. Jaundice.

31. Jaundice.
Cerebral vessels, moderately dilated.

Lungs: adherent at upper. Caseous + calcareous tubercles.

Constitution: Old man.

Liver: dull. Yellow and bile yellow tone.

Spleen: dull. Spleen.


Ulcers: small.

Liver: diffuse fatty degeneration.

No microsaccularis.
Mutter was devenegen in Asylum. Menses regular.

1884

January

Six weeks: 'Cold', hoarseness, cough.

Three weeks: jaundice, depression, Zephrin, vomiting, delirium

12-15: Constipation


16: Constipation. Obstructed. Vomiting

17: Vomit green. No pain. Quiet. Sudden illusions - very drowsy

18: Drowsy - Coma

Constipation (after 06. Con. 492)

Retention of urine.

Died: dark.

No reflex.
Lungs: unexpected
Heart: normal
Liver: not enlarged
Kidneys: healthy

Diary 25-03:
- Bright yellow
- congestion. Green-yellow with red patches
- Gall bladder empty
- Duct: patent

Liver: Fatty degeneration yellow
- Pigment. Some cells distended with oil globules
- Ischemic or hepatic necrosis
1882
April 22

Given to alcohol. Had been drinking.
Took a good dinner then lay down at 9 o'clock.
At night breathing rapid.

23
At 10 a.m. (at 6 a.m.) Stiff
abnormal vomit or severe

24
Reflux vomit

25
Tumbled. Feels worse.

27
Violent delirium. Frightened
(hospital) 6 p.m. Pupils wide, sensitive.
Stiff. Limbs rigid. Teeth clenched.
Tongue dry, black. No haemorrhage.
P. 118. D. t. Incoherent. T (rectal) 97° 8
Disorientation much diminished

Retention of urine

6.00 p.m. Pupils wide insensitive
T. (rectal) 97°. Frightened speech. Tanno
Maddening. Blood-foam

8.30 p.m. R. 30. P. 130. T. (rectal) 97°
Convuls

9.30 p.m. P. 113. T. 97°

11.15 p.m. No urine.
Hepatichic.

Brain: normal

Lungs: edema

Spleen: large, dark

Kidneys: fatty, soft.

Liver: 32 oz.

Yellow spots on dark chocolate

Lobules: indistinct

Portal canals: seemed numerous

Spleen: contained alit in

Black bile

Liver: Section of a few normal cells, fatty, degeneration, and debris.

Spleen:

Section: normal. A few red blood cells, veins intact

Afferent cells, granular, fatty, hemorrhage.

Inky-heliotrope mass; of fatty granules. Free nuclei and cholesterol material.
August
1861

Temperature Chart

December
1861

No. 77, B. S. 1861

Pulse
Resp
Motions

Remarks:

Slight general rotation. Clandestine colic.

Several deep.

Stool chairy.

Vomiting.

Severe meteorism. Episodic tenderness.

Pain.

R. Ovaries.

Weak.

Frequent Rous; Occipital Head pain.

No sleep.

Long sleep.

Motions.

Sleepy. Tense.

Incontinent. No sleep.

 coaches.

Slight moderate with little diarrhea.

4 months before. Body not ill.


Vomiting.

Auscultation.

Croupous.

Liver. Extensive fatty degeneration and destruction of cells.

Spleen. Pancytopenic crystals on the surface.

Kidneys. Enlarged.
- Left kidney, cloudy, pyramidal, inflammatory, haemorrhage in pelvis & calyces.
- Right kidney with protein fatty change.


Bladder. 11/2 inches in greatest length. Soft, deeply icteric with red parts.

Urine. Clear, yellow, bile.

Intestines. Content clayey, slightly yellow.
Pancreas large, purulent infiltration.

Peritoneum. Purulent infiltration in white posterior wall.
February 22
Jaundice noticed. Jaundice.
Slight vomiting.
February 23
Soft clay-stomach stool.
P. T. normal.
February 24
Jaundice deeper. Swollen.
Jelly-like yellowish color.
Liver edges well under ribs. Rumen diminished.
Restless. P. 60.
February 25
Jaundice, deep jaundice.
Icterus more marked.
Jaundice milky color.
T. Subnormal. P. 100/120.
No vomiting.
Cale
Hemorrhage into kidneys and intestines.

Kidneys: Each 17 g.

Spleen: 1420 g.

Liver 70 g. Healthy

Urine: Ammonia and a little light fluid

Duets patent

No trace of hepatic tissue only granular detritus.

Blood: Red corpuscles are normal.

Incapable of forming toluidine

Heme leukocytes.

Crystals of Tyrosine

Cholesterol.
Two new boys jaundiced - one sick leave from the ship.

Jaundice
By catheter
4 p.m.
8 q. 1035

Jaundice
Bile present
3 p.m.

Jaundice
Bile present

Jaundice
3 p.m.

Jaundice
Bile present

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Jaundice
Bile present
Dungs, congested.

Heart 7 1/4 oz. Fibrous clot on R. side.


Kidneys 10 1/2 oz. Granular & fatty.

Gastro Pyle, capillaries injected.

Stomach & Bowels, congested.

Contents 'coffee-ground'.

Liver 30 oz. Pale & flabby.

Dark-green bilious.

Gallbladder dull.

Scanty & faint.

Kidneys. Pigment & fat cells.

Liver granular matter.

Pigment. Oil globules.
1872

<table>
<thead>
<tr>
<th>Specimen</th>
<th>Date</th>
</tr>
</thead>
<tbody>
<tr>
<td>403. Soft-white</td>
<td>21/12/03</td>
</tr>
</tbody>
</table>

Cf. above fully reported.
38. M  
Blackemith

1882  
Formerly heavy drinker, abatement four months.

August

   Pale & crepuscule. P. quick. Well nourished
   Fever during the night. P. 90/80. T. 105°

   High sensation. Backache.

5. Severe headache. Feels better. Conjunctiva
   Yellow. P. 94. T. 102° 5

6. Fever during the night. Sweating. T 98°4

   Ankle, Elbow & H. Knee. P. 100. T. 103°
   Pain in lower abdomen

   Midriff clear. Swelling of forearms
   Call for Dypnoeic. P. 40. P. 90 Thoracic
   Ecchymosis ext. med. ext. H. arm

6pm: Caeruleus inst. wandernig
   Asa
Sanisburg

Lumps: congested
Heart: normal, no petechiae
Spleen: soft, dilated
Kidneys: echymoses, soft, pulpy
Stomach: large echymoses & lessers, curvatures

Liver: 48 oz.
Very lumpy, softened
Capsule stripped easily
Lobulation not so marked as
normal but still distinguishable.

Kidneys: Air sacs.
Artery cells swollen & bile stained

Liver: intense fatty degeneration
Cells chiefly masses of fat
Branches, no nuclei visible.
<table>
<thead>
<tr>
<th>Age</th>
<th>Sex</th>
<th>Occupation</th>
<th>Date Died</th>
<th>Cause of Death</th>
</tr>
</thead>
<tbody>
<tr>
<td>24</td>
<td>F</td>
<td>Peasant</td>
<td>8 mos.</td>
<td></td>
</tr>
</tbody>
</table>

**Medical Notes**

- Sudden paralysis, prostration, death.
- Vomiting.
- Hospital: death: head injuries, jaundice, liver failure.
- Comment: Staph. Pneumonia, death.

**Laboratory Tests**

- Urine: 68 cc
- Blood: Albumin positive
- Total solids: 10.75
- Urea: 1.25%
- Hemoglobin: 19.50 cc
Extravasation in pericardium + pleura

Heart, muscles pale

Lungs: Hyperaemia, oedema

Spleen: Pale, flabby

Kidneys: anaemic, costly; colour

Extravasation in lung; spleen's

Uterus: Flabby, pale, yellow, stunted

Pancreas: Flabby

Liver: Stab, yellow, fatty, die

Brain: Marked fatty, debriso.
Day 6: Jaundice
Day 7: Dead, inacervated pelvis
Day 10: Nekirin; Chronic Appendicitis
Day 11: Incomplete. T. 98°.4 St.ctor 2.120. Sph.
Dilated pupils, anaesthesia conjunctiva, tympanitic, abdominal tenderness
Haus delusorily perceptible.
Inconsp. Incontinence of urine. Cura.
T. 98°/100.4

Ur. 4.336%.

Death.
Throat muscle slightly rough.
Spleen: slightly diminished.
Uterus: endometrial.
Kidneys: arteries: bile stained.

date: 1703. orange.
Liver cells finely granular (alterations degeneration)
No microabscesses
an epithelial necrosis
Kidneys: like the liver

Stomach, Gastritis Mycose
Bacilli in vessels (endothelium)
attached membrane
Inflamma very few bacilli formed

Mucosa liver also play a segment
Coated the gastritis with
a tone main poisoning.
Temporary pain over liver, then Culture Fever

Typical culture Fever for four weeks
Sudden violent pain over liver & full bladder
Slight cough.
Rectal temp. 106.8
Sopor
Vomiting
Ratte.
Liver normal size, color and texture.
Bile ducts not visible.
Dark red, paler, yellow.

Spleen adherent to the surrounding tissue. Spleen lobes dark green, shrivelled.
Number of cases duct closed by stones.

Thrombocytopenia in partial marrow thrombosis.

Spleen enlarged.

Kidneys acute parenchymatous nephritis.

Cells in condition of acute atrophy.
<table>
<thead>
<tr>
<th>Date</th>
<th>Text</th>
</tr>
</thead>
<tbody>
<tr>
<td>1881</td>
<td>April 7</td>
</tr>
<tr>
<td></td>
<td>5/8/12 in hosp. of Dr. H.</td>
</tr>
<tr>
<td></td>
<td>10.20. a.m.</td>
</tr>
<tr>
<td></td>
<td>Deep yellow</td>
</tr>
<tr>
<td></td>
<td>T 93, O 76, R 20</td>
</tr>
<tr>
<td></td>
<td>Liver tender</td>
</tr>
<tr>
<td></td>
<td>Stomach</td>
</tr>
<tr>
<td>13</td>
<td>Intestines</td>
</tr>
<tr>
<td></td>
<td>Soft, tender</td>
</tr>
<tr>
<td>14</td>
<td>Spleen</td>
</tr>
<tr>
<td></td>
<td>T 98.6/100.4</td>
</tr>
<tr>
<td></td>
<td>Complaints: Intense pain in lower abdomen, burning sensation, and desire to urinate</td>
</tr>
<tr>
<td></td>
<td>Stomach and back</td>
</tr>
<tr>
<td></td>
<td>Stomach</td>
</tr>
<tr>
<td></td>
<td>Slight fever</td>
</tr>
<tr>
<td></td>
<td>british</td>
</tr>
<tr>
<td></td>
<td>Flushing and headache</td>
</tr>
<tr>
<td></td>
<td>5 p.m. dose</td>
</tr>
</tbody>
</table>

1880 Summer. Supposed from haemoptysis. No proof for Rupheus phosphorus.
Liver, 1994 oz.
Acute atrophy and signs of contraction.

Ecchymosis, pyocele, numerous
inflammation
- Mediastinal cellular tissue
- Pericardium
- Peritonaeum

Heart: Fully degenerating pyocele.
Kidney: Fully degenerating pyocele.
Urinary temperature. Slight audible systolic murmur.

Depression. Slight melancholia for some weeks.

Chill of station. Sausage Chateu (by other also) at supper.

Glaucous. Slight jaundice.

Improved. Jaundice nearly gone.

Severe headache and evening.

Discourages in morning. Kneel, sitting.

Recognized nobody.

Anesthetized. But in hand and head as if pained.

No corneal reflexes. Pupils wide, reactive.

Splen not enlarged.

Liver dulness not depressed by change.

Jaundice not increased. No pain.

Like liver or tenderness.

Deprit.
only partial permission could be obtained.

Haemorrhage on omentum, bowels.

Kidneys: Infarctions.

Liver soft: Pulpy mass, easy to sm.

Liver structure almost obliterated. Splenius fat globules.

Diagnosis confirmed by Professor Greenfield.
January 11. Asthma, jaundice, pleurisy, vomiting occasionally, clear yellow stool. Dr. X gave drops but would not for word.

E. M. taken meal, same.

After some days, had some constipation. Well nourished. Jaundice brighter. T. 98°


13. #4

Belly under 20.3. 
Brain 420 g. Soft & flabby. 
Lungs: a good deal of capillary icterus. 
Pant 140 g. Muscle pale & flabby. 
Stomach: contents offensively, 
for small icterus. 
Intestines: Icchymacae, sheets of blood 
visible, contents like paste. 
Liver 1300 g. By pericardium white, 
and had fallen away. 
Fleshy white and surface flabby. 
Diffused yellow patches & icchymacae. 
Sections yellow, soft, thick & gummy. 
Kidney (2) In Pratt. Blended with adhesions. 
Liquefaction in yellow part. 
L. Iliaca: distinct in red. 
Red part, no firmer than normal; 
shell depressed. 
Right shell more diseased. 
Spleen: content colorless. 
Duck: Brown. 
Spleen 2430 g. Larger & firm.
November 19: Admitted from horseman, divided injury of glands in front of neck.

December 3: Stomach sore on breast. Anemia. Heel sore.
11: T. 102.2/100.8. Systenma on left.
14: Papules on trunk. (21) Immunization.

8: Pulse 120. R. Immunization.
16: 101.5 above external auditory meatus.
17: 103/100.4

26: Minor abscess below sternum.
Super. Attachment of sternum. Roland.
R. Hypertension.

Wound.
Heart. Epicardial ecchymoses.
Muscle stiff, fatty.
Lungs. Subpleural ecchymoses.
Kupffer cells.
Spleen enlarged, 17 lb.

Liver, 4203. Surface wrinkled.
Small ecchymoses.
Red-brown with small yellow spots.
Black content moderate.
Spleen: Undercut, yellow, firm, smooth, under microscope, flat cells, spiky lining and chain present.

Intestine - Colorless, few ecchymoses.

Kidneys Enlarged. Cortex Enlarged.
Pyramids Engorged.

Cranium. Blood stains confluent in lower quantity in subdural space.
<table>
<thead>
<tr>
<th>Name</th>
<th>Age</th>
<th>Sex</th>
<th>Diagnosis</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jüntzel</td>
<td>36</td>
<td>M</td>
<td>Carbon Monoxide Poisoning</td>
<td>Attributed to illness due to inhalation of vapours in the works.</td>
</tr>
</tbody>
</table>

14 days jaundice

Symptoms could be noted and answered impatiently
disappearance diminished before death.

Leukopenic symptoms noticed;

anatomy does differ from those in phosphine poisoning, but no phosphorus is found in
the blood or the liver.
This passed.

28

Scurvy: Suffered principally the small joints.-Blisters in the hands and feet.

Jan. 19

Scurvy: Suffered principally the small joints.-Blisters in the hands and feet.

Threw cold about 8 months ago.

was drinking brandy for about a month at Christmas last year.

St. Mary's Hospital.

[Signature: Wilson]
Truncus Ileostomatis
Bowels: Unaffected
Kidneys: Unaffected

Liver (16 oz below average)
Capsule puckered
Sections dull yellow
lobules indistinct

Spleen: hardened a little
vivid Ammon's fluid
Nuets patent, Mobile

[No further text]
<table>
<thead>
<tr>
<th>Date</th>
<th>Event</th>
</tr>
</thead>
<tbody>
<tr>
<td>February 3</td>
<td>Vomiting &amp; Nausea, Abdominal pain, Deep drowsine, unconsciousness, death in coma.</td>
</tr>
<tr>
<td>April 26</td>
<td>Meeze jaundice, headache, nausea, vomiting, jaundice</td>
</tr>
<tr>
<td>May 3</td>
<td>Jaundice, Infection</td>
</tr>
<tr>
<td>May 5</td>
<td>Coma, Paralysis of limbs, Twitching</td>
</tr>
<tr>
<td>May 6</td>
<td>Death</td>
</tr>
</tbody>
</table>
Strong psychical disturbance.

Two forms occurred from asthmatic.

Tomnucu thinks it a neuropathic case.
26 | M | Volunteer

Continuous psychic excitement

Symptoms of fast and weak, cardiac
Dark brown urine, lassitude
Emaciation, weakness, diarrhea
Vomiting
Considerable increase of liver, heart valvul
Fever; after fever dulness suddenly
Diminished in appetite passed
Under the ribs, at first melanosis
but this passed away

Greencolor
Delirium, tremor, delirium amnestic
Hemorrhages

Death. Oedema of lungs.
dye analyses.

an Albuminoid like Albuminoid.
Peptone.
Sarcolic Acids.
Amino-fatty Acids (Mamm).

Tyrosin.
Aromatic Oxycarbons.
Xanthene bodies 3 traces.

Xanthene due to decomposition.
Nicolein (Saustuin Pashun's Acid).
v. Kessel has been found twofold in the urine (Bajrusky).
22. October 22
Fever, nausea, has felt fatigued
Tuesday: Great malaise of stomach
Some indigestion. Epistaxis. Diarrhoea
Persistant headache.
Small, thin. Face typhoid. Tongue brown
Abdomen, distended. Lenticular rose spots
Gurgling connected with R. Side fossa
Rales bibasilar apicis. Subepigastrial bruit.
P. 110/70 102/2

23. Right intercostal. Pain in pressure
in the fossa. Yellowish, oozing toward liver

24. Restless. The right delirium. P. Small
quick 100/70. 101/3. Feeble abdomen
Deaf pain on. Tender. Dulness about
R. Upper rectaloma line 8"/4"

3\4. in right gluteal fold

Frequent diarrhoea. Dark brown
Vomiting. No froth appear.
Restless. Tries to get up.

25. 11/0.

Nied
Dinner congested, almost dried.
R. cavity by thick false membranes.
Gastritis, general motility disturbed.
2. Large caseous nodules in middle.
Hypertrophy of a collection of small tubules at the apex. Concomitantly false membranes almost consolidated.
2. Stomach very small like a pigeon's chest.
Muscle-like strands, soft, flaccid.
Stomach slightly dilated, pale, lichen.
Colon a voluminous bundle of lumbricated large fibers (paler, firmer) at level of cæcum.
Rectal passage 1 cm. apex.
Follicles not hyperplastic.

Liver 61\% of body weight.
Section dark yellow, nodules.
Under the capsule some yellow tubercles slightly prominent.
Ducts prominent.
Lymphatic glands caseous.
L. not 20 cm. Nodules not caseous.
Scattered tubules, but some with amyloid bodies; pyramids congested above.
Bladder. Many small ulcers.
1879

Supposed to have been some fevers from an herbalist Dr. P. Another child ill

October 9

Severe cramps, headache, abdominal pain.

October 9

Sore throat, feverish sleeplessness.

October 9

Two days, fright delirium.

October 9

Day awake, left knee, back, headache, shakiness.

October 9

Knees, face pale, slight oedema hands.

October 9

P. very rapid, irregular. Abdomen tense.

October 9

Swarthy ascites, slightly bronchitis.

October 10

Liver large, 5" upper border. Yellow urine.

October 10

T. 104 1/104

October 11

98 2/102 6

October 11


October 12

Starved, slept. No delirium, diuresis.

October 12


October 12

Much fever. Liver much less.

October 12

Framed stools, Porten urine.

October 14


October 15

Died, slightly delirious.
Lungs, nearly solid.
Heart, white, spherical, Hermes
Spleen, small.
Stomach, content, homogenous.
Kidneys pale, cut, dull white medulla angosted.
Birr 16.02. P.M.

Kidney cells very sally.
Liver, white, toned.
Oment, yellow, reddish, muddy.
Mucous membranes, transparent.
Cells from intestine thin, changed to fat.
Portions of mucous membranes round fat drops.
Notable fat drops.
Homogeneous connective tissue.

Thrombosis present in section shown with identical changes, and history of maleic acid jaundice.
no pancreas
No clinical symptom of import

Liver enlarged, cloudy
Yellow to green

Microscopic masses in bile ducts
Bacteria surrounding vessels.
No jaundice
Fever, water general symptoms, with abdominal pain + ascites
No jaundice, no fever
Ascites tapped. Improvement for a day.
None from vomit, collapse and death
Petechiae in skin, muscles, peritoneum. Intestinal lining. Recent hemorrhage of R. Ileo vein.

Liver: 330 g

Connective, wall of Portal Vein & surrounding tissue in new tissue. Smaller branches and also the arteries & veins.

Yellowish whitish yellow lobules, alternating with dark red. Diff. under Portion 5 x 7 x 3 cm.

Spleen: Enlarged.
Dear Stomach Distress Constipation

Tenesmus swelling of belly

Diarrhea dyspepsia

Abdominal pain.

Old man's legs

Lung apices dull brisk respiration

Abdomen as above.

Repeated aspiration

Gentle stimulant

Death.
<table>
<thead>
<tr>
<th>Liver</th>
<th>2002 smooth</th>
<th>Thickened capsule in parts</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spleen</td>
<td>Enlarged, adherent</td>
<td>Adherent gray brown</td>
</tr>
<tr>
<td>Bile</td>
<td>Bile with calculi</td>
<td>Dilated capillaries and</td>
</tr>
</tbody>
</table>

Thickening of vessel walls

Atrophy, endarteritis


22. Unconsciousness.

25. In bed. Sent to hospital.


A littlerigidity of the neck. Fingerskips string, peripheral stimulation, R. plantar stiff.


Vomitus, green. Appetite absent. Urine albumin, white blood, T. 100.

May 27. Cold improved.


Foth, ulcers. Rigid on neck.

No petechiae. Ht. 153cm. T. 98.6/104.4

Death on Monday.
Meniscal vessels full of black thick fluid,
Capsules melanin. Visceral substance haemosiderin.
Large fibrous head. Yellow deposit.
Intercellular and lamellar nuclei. Mitoses are extensive here; also protein in peritoneal capsule. On lesion. Mucous membrane affected.
Liver 335 oz. R. lobe flattened.
Spleen 13 oz. fatty but not very much.
Kidneys. Cortical tubules dilated.
Lungs R. alveoli elastic.
L. Alveoli less complete.
Numerous alveolar sequestrae.
Stomach: Empty.
Spleen 4 oz. firm.
No other change can be seen.
<table>
<thead>
<tr>
<th>Date</th>
<th>Events</th>
</tr>
</thead>
<tbody>
<tr>
<td>1878</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>Lacitade, sudden pain in lower limbs</td>
</tr>
<tr>
<td>10</td>
<td>Anorexia, headache, diarrhoea, vomiting</td>
</tr>
<tr>
<td>11</td>
<td>Anemia, jaundice</td>
</tr>
<tr>
<td>13</td>
<td>Hospital, anemia, jaundice, liver enlargement, normal size</td>
</tr>
<tr>
<td>14</td>
<td>Splenomegaly, white stools, yellow urine</td>
</tr>
<tr>
<td>16</td>
<td>Hematuria, microscopic blood, hematuria, diabetes, depressed 3-4%</td>
</tr>
<tr>
<td>18</td>
<td>Albuminuria, night urine, edema.</td>
</tr>
<tr>
<td>19</td>
<td>Nausea, headache, coma, delirium, death</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Name</th>
<th>Moses Carroll</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>54</td>
</tr>
<tr>
<td>Sex</td>
<td>M</td>
</tr>
</tbody>
</table>

Pocket Chart

For Bedside Case Taking

Compiled by Robert Simpson, L.R.C.P., L.R.C.S.

John Wright & Co. Publishers, Bristol

Copyright.
dumps' urine, copper
Heart - soft
Liver, 5 oz., yellow green
Constitutes albuminuria
Salivary a little bile

Kidneys, normal size

Albumuric Canalis normal

Discoe

Albuminormal
Many cells infiltrated with pigment
in the lobules spaces wide, at one point
only was cell infiltration seen
cells fairly badly

Kidneys
Tubular epithelium cloudy, granular
in part avulsive
Sub invasive capsular - connective tissue
Abstract

History of cases (published since 1876)

of acute diencephal atrophy verified by autopsy

but unaccompanied by a histological report.


Dreier Haus, Halle

Vinkler Kirche, Jakob, 1885, 2.
<table>
<thead>
<tr>
<th>Date</th>
<th>Event</th>
</tr>
</thead>
<tbody>
<tr>
<td>1878</td>
<td>Miscarried nine months previously</td>
</tr>
<tr>
<td></td>
<td>Six weeks' fetus decomposed</td>
</tr>
<tr>
<td></td>
<td>Died 3rd weeks - Deepening jaundice</td>
</tr>
<tr>
<td>2</td>
<td>Wanderous delirium, no pain</td>
</tr>
<tr>
<td>4</td>
<td>Bright jaundice, P. 108 R. 12 T. 98.6</td>
</tr>
<tr>
<td>5</td>
<td>Disgust Spleen dulness normal</td>
</tr>
<tr>
<td></td>
<td>Constant Vomiting</td>
</tr>
<tr>
<td>6</td>
<td>Scurvy, dark, lumpy</td>
</tr>
<tr>
<td>7</td>
<td>Headache</td>
</tr>
<tr>
<td>9</td>
<td>Foul perrectum</td>
</tr>
<tr>
<td>10</td>
<td>Jaundice less, feels better</td>
</tr>
<tr>
<td>11</td>
<td>Scurvy</td>
</tr>
<tr>
<td>18</td>
<td>P. 128 R. 13 T. 87°</td>
</tr>
<tr>
<td>19</td>
<td>Purple discoloration inside stumps</td>
</tr>
<tr>
<td>21</td>
<td>Liver dulness less</td>
</tr>
<tr>
<td></td>
<td>She is quite rational when objectives</td>
</tr>
<tr>
<td></td>
<td>To vapour by ammoniac</td>
</tr>
<tr>
<td>24</td>
<td>Died</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Date</th>
<th>Event</th>
</tr>
</thead>
<tbody>
<tr>
<td>1879</td>
<td>Tent was introduced to provoke abortion</td>
</tr>
</tbody>
</table>
Sternach eingerieben, all über der
Gerippe.

Niere: Haltig
Die [bleibt] keine Capsula / Wirtz

Spleen dunkel, Emphysem
Durch 34 Gr.

Haltig
Sauer, brunn

Leber, markiert, Blut-Einzelteil
in Exsudat. Kobolde, visibles
Sauer, brunn
Emphysem

Gallengalle enthieilt heiliges Wärme
Die

Leber, Venus. Einzelteil
enthielt aus flüssigem Blut
Mental distress for a year during husband's illness, ultimately melancholia with delusions.

1881.

The week before she was admitted to the Asylum 10th June.

June 10

Admitted to Asylum, and through still depressed. Much improved his side at night.

July 13.


August 13.

Jaundice.

14.

Rash. Spotted papules. General irregularity of surface, exucent.

15.

Bowel's confined. Rash fading on face.

16.

T. 98.4. Rash fading.

18.

T. 93.4. Better but very feeble.

22.


23.


27.

Jaundice due to fever. Jaundice marked. Eruption present and mixed.

August 1.

Jaundice intense.

2.

Jaundice and chills.

3.

Jaundice deep. Fever, yellow stains, hepatic tenderness, no lymph nodes.

Death at 3am.

Wants facial expression.

Features round smooth, immobile after the paroxysms. Delirious.
Br. Lateral ventricles dilated.

Liver at 30g. Zellweger's. Histol. 84% 
Marked at anterior base. Endocardium deep purple on R.

Lungs adherent at apices. Intervole of R. apex. Pulmonary nod hepatitis am

Spleen 4 % of

Duod. 37% of. Adherent over upper surface.
Rectum almost black in parts. in other scabrous yellow. Soft and pulpy. L. col. very thin.
Salt in bladder content 32. dark hide with duct patent.

Kidneys. R. Sec. 1. 60g. Capsule adherent. Substance deep brown. Cupolated.

Pale brown secre in lower bowel.
1882

Confinement of a monster, prematurely

March 7

Restless night, screaming, delirium. Vomiting, even bedwetting

Sudden coma

Inconscioius, occasional screaming

Cerebral vessels pulsating violently

Head to left, eyes turned up, fixed

Pupils dilated, insensitive

Arms flaccid, ataxia, tachycardia

Legs drawn up, much starting

Extremes of rigidity, ataxia

Respir., P. 28, R. 100 very small, T. 107.

Unic, decapitated, passed over bed.

Lata, complete AP and posterior

Ecchymotic marking ankles

Noddy fluid from mouth

Occasional profound, staring, flushing

Breathing laboured, irregular

P. small, rapid, T. 104.

Blood swarmmed with bacteria

W. fracture of skull.
Liver 30\% wt.

Red pleura stiff and yellow.

Multitude of small vessels on the surface.

Gall bladder tightly closed and almost full of mucous ducts patent.

Glands in lesser omentum swollen.

Spleen 5 oz. soft.

Brain soft.

Hodules present of phlebitic aneurysms of major arteries.

Bladder containing excess of yellowish mucus containing strined and containing multitude of mucus.
<table>
<thead>
<tr>
<th>Day</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>24</td>
<td>Sudden malaise, fever, headache, jaundice, vomiting.</td>
</tr>
<tr>
<td>5th</td>
<td>Obliterated, liver healthy, virea</td>
</tr>
<tr>
<td>6th</td>
<td>Vomiting, slight nausea. No fever.</td>
</tr>
<tr>
<td>7th</td>
<td>Jaundice, nausea, constant diarrhoea, vomiting with blood, trismus.</td>
</tr>
<tr>
<td></td>
<td>Slumped face, close moment of faint.</td>
</tr>
<tr>
<td></td>
<td>Unconsciousness, deep unconsciousness. Symmetrical pulse, no pulse.</td>
</tr>
<tr>
<td></td>
<td>Pupil, insensible. P. 196.</td>
</tr>
<tr>
<td></td>
<td>Liver dulness, distinctly perceptible at 3½ feet. Auditory line. Delta marked.</td>
</tr>
<tr>
<td></td>
<td>Uterus firm. Symptoms of endometritis, &amp;c.</td>
</tr>
</tbody>
</table>

Health.
Pia Materia unruptured
Effusion in Ventricles
Sbjut. Soft
Echymoses in pericardium
Kidneys, pale
Liver, small, flat
Coronary yellow
Atelectasis of right ventricle
All over delirium
1885

SYPHILIS TWO YEARS BIPPE

July
25. Hand, Vomit bitter taste. Doporin
Rutin. P. 90. T. Normal
Constitution: Normal.
27. Conjunctivae yellow
Slight headache. Appetite.
29. Appetite, Hands swollen (Must have deg 2 years).
Drake, P. 90 T. 98.4.
30. Tumidc. deeper. Vomit. Cerebral, palsy
Liver, dulness less. T. 92.4.

Aug
Postulated: Evaluation.
Brui-,lces
Mechanical dysphagia.
Anorexia at night. Staph.
180. 43 102.5.

Extract
12 hours pm.

Liver under 3cm.
Hacmit. granular. Section.
sclerotic spots.
Gallbladder content. Green
Duct patent.
<table>
<thead>
<tr>
<th>May</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>14</td>
<td>Sudden bloody vomit</td>
<td></td>
</tr>
<tr>
<td>15</td>
<td>Jaundice, jaundice, jaundice, rapid</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Dyspnoea, coma</td>
<td></td>
</tr>
<tr>
<td>16</td>
<td>Rapid</td>
<td></td>
</tr>
</tbody>
</table>
Drops: Oedema, Angina

Stomach: enlarged, putrid

Liver: very small
Soft, incoherent
Hollows yellow, like hemp seed
Extravasation
Gall bladder: amber, brown, fluid.
50 Mr. American Merchant

Abstinence from alcohol.

In five years mental strain.

Indefinite symptoms, with vomiting, jaundice, weakness.

Deep jaundice, hemi-cona, muttering.

Vomiting constant, & passage of altered blood from rectum.

T. 98° F. 60. Weak irregular.

Conna.

Sudden.
Swiss under 320g.
Dark brown yellow.
Rough shair.
<table>
<thead>
<tr>
<th>Date</th>
<th>Event</th>
</tr>
</thead>
<tbody>
<tr>
<td>25 July 1887</td>
<td>Slank from May 1886 not inflammatory or explosive</td>
</tr>
<tr>
<td>31 July</td>
<td>Fever, last day of July, Malarias slight headache, anorexia</td>
</tr>
<tr>
<td>1 Aug.</td>
<td>Cold, grippe, nausea, epigastic oppression. Hospital. Somewhat emaciated</td>
</tr>
<tr>
<td>31 July</td>
<td>Skin eczema, T 100/101, D. 100 soft</td>
</tr>
<tr>
<td>1 Aug.</td>
<td>Cold, Flu, 101.3, Rested almost well in head, anorexia, epigastic pain</td>
</tr>
<tr>
<td>6 Aug.</td>
<td>Steppella, 99.5 - 101.3</td>
</tr>
<tr>
<td>7 Aug.</td>
<td>Fever, after rising temperature, no pain. Great headache, gastric trouble, T 102.2</td>
</tr>
<tr>
<td>7 Aug.</td>
<td>Sleepless</td>
</tr>
<tr>
<td>8 Aug.</td>
<td>Skin found nice. Headache great. Anorexia, ideas, answer very slowly, unwillingly, D. 120, Small. 101.3</td>
</tr>
<tr>
<td>8 Aug.</td>
<td>Dim vision, crystal body with teardrop</td>
</tr>
<tr>
<td>9 Aug.</td>
<td>Jaundice general, deeper, tongue dry, stools chalky, T 102.4/104.4</td>
</tr>
<tr>
<td>9 Aug.</td>
<td>Jaundice, epigastria, plugged, T 103.1-104.9</td>
</tr>
<tr>
<td>10 Aug.</td>
<td>Jaundice, depression, jaundice, yellow skin, face red, demin paranaemia, fibrin 104/104.9. Glands enlarged</td>
</tr>
<tr>
<td>11 Aug.</td>
<td>Jaundice worse, jaundice deeper</td>
</tr>
<tr>
<td>12 Aug.</td>
<td>Jaundice deeper, emaciated, painful gout and a circumscribed infarct of liver past yellow skin T 105.2, D. 160, urine malignant, most unpleasant odour</td>
</tr>
</tbody>
</table>

**Death, 105.6, 6**
Liver large, firm to touch
Ducts normal
Bile ducts normal
Gall bladder content, color, yellow bile.

Spleen enlarged, capsule wrinkled, resistant
Other abdominal viscera normal

Pleuric dissemination haemorrhagic spots
Lungs normal

Heart normal, 205.
<table>
<thead>
<tr>
<th>Year</th>
<th>Date</th>
<th>Symptom</th>
<th>Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>1888</td>
<td>April 8</td>
<td>Abdominal pain, two weeks</td>
<td>Weak jaundice</td>
</tr>
<tr>
<td>1888</td>
<td>April 12</td>
<td>Jaundice, morose</td>
<td></td>
</tr>
<tr>
<td>1888</td>
<td>April 13</td>
<td>Died</td>
<td></td>
</tr>
</tbody>
</table>
dew in 1803.
Soft. fulgy
Dirt yellow-green.
History of cases (published since 1876)

of recovery from illness diagnosed

Acute disorder Atrophy

84, 85 Howley 86 Michel 87 Mered 87 Loh 87 Müller 90 L. W. E. W. S.
<table>
<thead>
<tr>
<th>Date</th>
<th>Events</th>
</tr>
</thead>
<tbody>
<tr>
<td>June 14 7/12</td>
<td>F</td>
</tr>
<tr>
<td>1864</td>
<td><strong>Subjects to bilious attacks &amp; fainting</strong>&lt;br&gt;Ro mornig &amp; ekeeo&lt;br&gt;Recovered fr Smallpox int May.</td>
</tr>
<tr>
<td>July 3</td>
<td>Sudden severe hepatic pain&lt;br&gt;Right hepatic lobe enlarged; jaundice&lt;br&gt;Epicystic pain after food&lt;br&gt;Dorsa depresso, &amp; angios&lt;br&gt;Died suddenly from 1st liver nipple&lt;br&gt;After dei 12h 1st boro.</td>
</tr>
<tr>
<td>Aug. 10</td>
<td>Jaundice decrease&lt;br&gt;Deceased 1/2 mi</td>
</tr>
<tr>
<td>24</td>
<td>Anvalerent.</td>
</tr>
</tbody>
</table>
Henry
58 m
1869


Weak, jaundiced, Tongue dark grey.
Skin hot but muriatic. P. rapid. T. nearly
Intermediate. Intellignece Sluggish.
Respirations headache. hr. xii. tenderness
Liver Alkaline 1 ml.
Vomited haemorrhagic
Constipated. Stools bloody.

Recovered quickly.
Treatment: No. of doses: 1

hi 15 months
18 "

Liver dulness 3 ml.
- 4 1/2 ml.
1873

**January**
- 13: Intense headache, last ludicrous.
- 18: Fever, Euphoria.
- 20: Hospital, Convulsions, yellow, irritable.
- 21: Jaundice, general; liver, short fatigued.

**Unice, 90 a.m.**
- 8.90 a.m.
- Systolic 101.5

**Blood**
- Etiometry 1.0
- Hb 85%
- Liver: Severe, yellow, jaundice.

**Tympanum**
- Liver: 2/5 in. T. 100. D. 80/88
- Fever: Severe improvement. Sleep.

**February**
- 27: Fever, Rectal.
- 29: Jaundice, Jaundice.

**March**
- 14: Jaundice.
- 15: Jaundice.

**Bedlaher.** Proof, delirium.
1874

June 12 Repir, Cough, Postulation, Madache, Backache.
      Fever, Unic black.

15 Jaundice, Loss of Temperance;
      Sleepless, Anorexia, Constipation.

17 Liver Stagnant, Kidney enlargement.

June 18
      Burns, Plane, Jupiter, Apollo.
      Hemorrhoids, T. 101.5/102.4.
      Gumb Hysteria, Hoo Manoeuvre.

June 20
      Deeper jaundice, Liver shrunken, Kidney.
      Stipula movement and contractions.

June 21
      Delirium, Allergy.
      Slight bitter, Leucopenia.
      Liver under 100, Leucoma.

June 22
      Hoberman, 50000, Asthma.
      Liver dry, Pallor.

July 1
      Insomnia.

5 Insomnia, Jaundice.
    Liver, Vomiting, Fever, Jaundice.

17 Insomnia, Jaundice.
Felt ill in malariaous places but never had fever.

October

1. Sudden diarrhoea, no fever.
2. Slight fever, headache, weakness, anorexia, no grip, no chill.
3. Great headache and malaise.
   Stool: Brown, frothy, tawny, faeces.
   Jaundice: Cold skin.

Bilirubin. Stips at right side of sternum.
Nipple line, rachis from ribs.
7 cm to 10 cm above umbilicus, no fluid.
Spleen enlarged.

Stimulant treatment.
In a few days, relief and improvement.
Temperature normal. Bowels clear.
Liver dulness increased 1 cm daily, and in 10 dayssnore normal.
<table>
<thead>
<tr>
<th>Date</th>
<th>Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>April</td>
<td>Dark Stools. P. 120.</td>
</tr>
<tr>
<td>Date</td>
<td>Event</td>
</tr>
<tr>
<td>------</td>
<td>-------</td>
</tr>
<tr>
<td>April 21</td>
<td>Unwell, Vomiting. Vomiting continued, tannic acid in general malaise. Drier decreased from 2.2 in above navel to become impalpable. Intercurrent erysipelas of face. Drier again palpable.</td>
</tr>
<tr>
<td>July 12</td>
<td>Drier 2.2 in above navel.</td>
</tr>
<tr>
<td>Sept. 20</td>
<td>Nourishment for months by peptonized water.</td>
</tr>
</tbody>
</table>
History of cases (published since 1876)
in which no autopsy was made.

1876

June 30

F. Infatuated, jaundice for a few days.

Temperature 99.2, well-nourished

Anorexia, constipation.

Pallor, bilious urine, depression, no fever.

July

2

3.

P. 140. T. 104. 6.

Bilious ailments,

Unconscious movements, defecation

Muscular twitching

Dura dulness normal

4

Bruit dulness less

Convalescence

Death.
<table>
<thead>
<tr>
<th>Date</th>
<th>Event</th>
</tr>
</thead>
<tbody>
<tr>
<td>1878</td>
<td>Day after marriage.</td>
</tr>
<tr>
<td></td>
<td>Sky, return to health.</td>
</tr>
<tr>
<td></td>
<td>P. 100.</td>
</tr>
<tr>
<td></td>
<td>Dull, coma.</td>
</tr>
<tr>
<td></td>
<td>Black vomit.</td>
</tr>
<tr>
<td></td>
<td>Driven ditches 1½ mi. along edge of road.</td>
</tr>
<tr>
<td></td>
<td>Convulsions.</td>
</tr>
<tr>
<td></td>
<td>Health.</td>
</tr>
</tbody>
</table>

Constipation habitual. Ovarian Tumour.
Subsequently jaundiced a few days after marriage.
<table>
<thead>
<tr>
<th>Arqueroa</th>
<th>Havana</th>
<th>Con dedic. parti. Havana 1879 5 57</th>
</tr>
</thead>
<tbody>
<tr>
<td>20</td>
<td>M.</td>
<td>Veri. del. H. Sabast. 1880 2 197</td>
</tr>
</tbody>
</table>

Some drain is often communicated with decay.
This case of yellow fever in house within a few months last a month ago.

Decrease temperature
Decreasing liver dulitude
<table>
<thead>
<tr>
<th>Date</th>
<th>Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>29</td>
<td>Skin yellow, no fever, no pain, uncommon vomiting, good appetite.</td>
</tr>
<tr>
<td>November 6</td>
<td>Sudden delirium, rapid heart rate, P. 80, T. 97°, difficult to nurse.</td>
</tr>
<tr>
<td></td>
<td>Liver dulness under 6&quot; rib only</td>
</tr>
<tr>
<td></td>
<td>Papiles dilated, Hepplein, Shotty White</td>
</tr>
<tr>
<td></td>
<td>Urine: Daffron</td>
</tr>
<tr>
<td>7</td>
<td>10 a.m. Spoke to his father</td>
</tr>
<tr>
<td></td>
<td>2 a.m. Completely unconscious,coma</td>
</tr>
<tr>
<td></td>
<td>Continuous, deep firm vomiting</td>
</tr>
<tr>
<td></td>
<td>Liver dulness normal</td>
</tr>
<tr>
<td></td>
<td>Death.</td>
</tr>
<tr>
<td>Date</td>
<td>Age</td>
</tr>
<tr>
<td>------------</td>
<td>-----</td>
</tr>
<tr>
<td>22 September</td>
<td>1891</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td>Date</td>
<td>Events</td>
</tr>
<tr>
<td>--------</td>
<td>--------</td>
</tr>
</tbody>
</table>
| 20 Dec | *Vomited* three times, *temperature* 100.4°F, *headache*.
| 21 Dec | *Vomited* four times with *ache*.
| 22 Dec | *Coughed*.
| 23 Dec | *Appetite*.
| 24 Dec | *Toilet*.
| 25 Dec | *Toilet*.
| 26 Dec | *Toilet*.

**Note:** *Headache* when moved or ed of cholera.
Sections of liver from case of Acute Liver Atrophy (case III)

The liver had been four days in spirit. A fresh section was made vertically through half the thickness of the right lobe from the posterior surface. The parts were turned aside and laid with the Spigelian lobe of the left lobe between them, and the drawings were confined to this part.
Acute Liver Atrophy
D.R.I. 27.2.94
A. Yellow area. x 480.97
Peri-aneurysm
Lobule showing Anhritic
Change and cellular
Infiltration.
Red area, x 97

Cervic. LAPWOOD:

Double arrangement of stroma preserved.

Thrombosed fibrous cells.
O. Uterine Carcinoma
Dense tissue with fibromyxoid proliferation of mon核 cells. Small cell infiltration all over.
[Handwritten text not legible]
Edwes x 62
Corneal
Corneal drusen
Fusiform
Yellow nodules
(Fusiform
Corneal drusen)
C. Red area × 435
Osmic acid
Single Bile Duct
which has been cut
at knuckles at
A B C D E.
C. Red Area x 230

Drawing of an area in which haemorrhage has taken place.
B. lusia. x230

dogwood. Drawing of vessel in one of the fibromuscles.
A. Kidney x 52. Osmic acid

The cells are better seen
than in the logwood section.

A. Kidney x 52. (Deposed)

The disintegration is barely advanced.
The drawing from a fact corresponding to W. Kidney A.
Shows here there a tubule cell in the space. The
stroma seems much increased but not fibrillated. There are purpse of
brown fragment granules here there. Somewhat they seem to
follow the course of the tubules.
(3) Stroma alone left:
No trace of tubular epithelium which has fallen out.

(a) More advanced:
Spaces mostly empty. Tubular remnants here.

(b) Closer capsule. Enlarged:
Tubules mostly frequent, but some only partly fill the spaces. Some of these are empty.
B. Kidney x 230

Innerven (a) Shown
a. Parity the integrated tubules
b. Adjacent tube in the cortex
c. Tubules with nucleated epithelium

B. Kidney x 230
Group of five spaces from (c) under capsule
Shown in different stages of this integration.
C. Red Area x 435
Gemnic Acid.

Sketch of wall of veic showing picked distribution of fat granules at thin wall.
Grave Jaundice

I have used the symptomatic term grave jaundice as an exact translation of the icterus gravis which connotes a condition of deep jaundice in which severe mental symptoms have developed usually in the form of an impairment of consciousness.

Case 3
Grave Jaundice during Pregnancy.

If you look at this woman of 34 years you see that she is very deeply jaundiced, the face having a deep green yellow hue. You see also that she lies rather listlessly, but if I ask her some simple question she makes an effort to rouse herself. She is not very successful and though she seems to grasp the meaning of the question she sometimes in a drowsy, drawling voice answers that she can hardly tell. When asked she puts out her tongue in a slow deliberate fashion and you see that it is rather thickly furrowed especially towards the edges. When we turn down the clothes you see again the deep yellow colour of the skin and you note that the abdomen is swollen and that there are about a score of spots with bloody crusts upon them.

Observing the abdominal swelling more closely you note that at the upper part the peristaltic movements of several coils of distended bowel can be seen through the apparently thin abdominal wall. The lower part has a smooth rounded outline which reaches as high as the umbilicus. On palpation we find that this rounded swelling is due to the uterus pregnant about seven months. In the upper part we cannot even though the walls are thin feel the edge of the liver but in the flank there is a sense of fulness which suggests distended colon. On percussion one at first thinks that there is no liver dulness. In the nipple line we have to go well above the rib margin and eventually a dull note is found over an area beginning half an inch below the nipple and extending 1 1/2 inches down. It is however not so intense as the ordinarily absolute note of the liver. The splenic area is not enlarged. If you look now at the lower limbs you see that the skin is closely set with minute red points which remind you of flea marks but many of which lack the central red point and also the rounded outline of the parasitic petechia.
The patient was admitted to the hospital two days ago and at that time her symptoms especially the mental obfuscation were even more marked. She could not answer questions at all though she evidently tried to and she was very drowsy yawning widely every few minutes when she was not actually asleep. The history before admission is that this is her fifth pregnancy and that during the last she had a transient jaundice which recovered spontaneously before she came into hospital to be delivered. Apart from a uterine prolapse for which she attended my outpatient department and wore a pessary she was well till about six weeks ago though she was very constipated and had at times some bearing down pain. Six weeks ago she began to vomit, usually ten minutes after a meal. Two weeks ago she became done up and had to lie occasionally. Her husband had noticed the yellow colour about her neck and the red spots on her skin afterwards. Six days ago she was confined entirely to bed, the jaundice became deeper and three days ago her manner became strange and she had incontinence of urine. She had no stool during the five days before admission. She has complained much of itchiness and has scratched herself.

We may now complete the clinical account. The pulse is a little quick, ranging from 104 to 112. The temperature has been usually subnormal, the limits being 97° and 99°. The urine has apparently not been scanty though owing to the incontinence of the bowel which we have partly produced the quantity could not be exactly measured. The urine in catheter specimen is dark amber in colour, frothy, slightly hazy but without deposit. S.G.1011. Urea .7%. Albumen in traces, epithelial casts are found microscopically and a few phosphatic crystals but no leucin or tyrosin. The Biliverdin reaction is intense.

The stools which we have been dark in colour, but it is not certain that this may have been dark blood.
The Cardiac area, beat and sounds are normal. The respiratory murmur is vesicular and without accompaniments. Ankle clonus is got to three or four jerks on each side and the plantar reflexes are lively. Proceeding on the theory that the symptoms were produced by a condition of poisoning we injected two pints of saline solution under the left breast and to get the evacuations in order and if possible introduce more saline solution ordered five ounces of saline solution to be given by the rectum every four hours while by the mouth we gave five grains of calomel followed by a tablespoonful of Mist. alba every two hours. Some of the saline enemata were returned some retained in a all about two pints of saline solution were retained in the first day. The bowel had not moved after an enema of soap and water was given and a copious brown formed stool provoked. Twenty-four hours after admission the liver dulness was even less occupying only the fifth interspace. She had a four pint saline enema and has since had passed a large quantity of dark brown stool and her urine partly without control. Today the condition is as you have seen slightly improved.

The after history of the case may for convenience be added here. It is a record of steady improvement. Next day the jaundice was visibly less and the patient was much improved in intelligence having now conscious control of both stools and urine and the liver dulness began to be more distinct and to extend. She had polyuria, four to six pints a day being measured for the next week. The saline enemata were omitted on the third day, the saline mixture on the sixth. On the fifth day the liver dulness had increased in intensity and the absolute area now measured two inches in the mammary line. The tongue was clean and the patient very hungry. On the tenth day the jaundice was much diminished the mind clear and the stools had become clayey in colour. The intestine still showed a tendency to gaseous distention.
This tendency to gaseous distention may account for the fact that she went into premature labour. On the evening of the tenth day she had been complaining of and crying out with pain in the back but it passed off and she slept most of the night. At 6 a.m. the membranes were found protruding from the vulva. She was transferred to the Maternity ward. The membranes were ruptured and a female child was born at 8.30 a.m. Both liquor amnii and vernix caseosa were markedly yellow but the child was not jaundiced. The placenta was born at 8.35 a.m. The bleeding during the third stage being free. Afterwards there was no excess. The patient had Liq. Ergot. The child had retraction of the episternal notch during inspiration and died in hours. The relative liver dullness was measured 4½ in. and was only 1½ above the costal margin. Two days later it reached the costal margin.

Though the labour had been absolutely spontaneous and though the jaundice steadily regressed the puerperium was not uncomplicated. On the third day the temperature rose and next day a fragment of decidua was expelled. On the sixth day the temperature again rose and on the ninth a large clot was expelled and it is interesting to note that we had an almost identical occurrence in a previous case of intense obstructive jaundice. After this the patient had a series of intrauterine douches but the temperature kept up till the nineteenth day and on several occasions there were rigors. The patient was able to go home on the twentietheighth day and I have since seen her as an outpatient for her prolapse. She then showed no trace of her liver illness.
Case 2.

About five years ago I was called one evening to see a primipara who had been delivered of her first child a few hours before. She was thought ordinarily rather bright become almost suddenly confused and could with difficulty rouse to answer ordinary questions. The bowels were said to have been moved daily with Cascara. In spite of this the colon was found loaded from end to end. The liver dulness was normal. By daylight next morning she was seen to be deeply jaundiced though this had not been noticed before. The patient gradually but speedily recovered under free purgation.

Case 3.
Case 3

Eleven years ago I was called one evening to a woman of 29 who had one child ten months before and had not lactated. She had been under treatment two months previously for secondary syphilis and had been apparently somewhat irregularly taking under mercurial treatment since. Three days before she had been jaundiced and she had been constipated for three weeks. Her husband had left her apparently well enough in the morning but on his return from work found her sitting in the middle of the floor with her hair down and unable to speak. After considerable effort I got her to put out her tongue but she repeated this as the answer to any question or rather on every rousing after she had heart and liver dulness normal. Next morning before going into hospital she was rather more easily roused but on admission was unconscious and could not be roused and she had occasional sighing and yawning. In the evening consciousness had partially returned. The liver dulness remained normal in extent for two days more though on the second its intensity was less all over. On the next day the intensity was even less and the extent reduced to three inches in the nipple line. She had distinct ankle clonus and a curious tache appearing as a line of "goose skin" and more easily elicited on the chest than on the abdomen. Next day the pulse had risen from 70 to 104 and the respirations were stertorous and long. She had several times gave a loud scream as if in pain. She died next morning just seven days after the jaundice had been first noticed. She vomited only once. The temperature was never over 76\degree.

The urine was bile stained, showed traces of albumen but no bacillus or typhosum. Urn 2.5%.
This last case gave me the opportunity of studying the morbid anatomy. The liver weighed 23 oz and on section showed extensive areas of orange-yellow color on a base of deep red. Microscopically the liver cells were extensively degenerated and there were widely distributed hemorrhages in the red parts.

These three cases raise in a most interesting way the questions of the diagnosis and treatment etiology of acute atrophy of the liver. In the third the provisional diagnosis made on sending the patient to hospital was confirmed by the subsequent course of the case and by the morbid anatomy. In the first the symptoms were analogous, deep jaundice, rapid mental obscurcation, constipation, low temperature, and liver dulness diminished both in extent and intensity. Was it also acute atrophy?

In both and in the middle case also we had as a possible etiological factor the marked constipation and the success of the treatment in the two cases tends to confirm our impression of the importance of this factor.

Personally I am convinced that the provisional diagnosis was as well established in the first case on her admission to hospital as it was in the last and also that this patient would have died had we not treated her as we did. In the second case matters had not advanced to any organic affection of the liver, but it seems not unlikely that had the case lasted longer it would have come still closer to the others than it actually does. It is possible that this may account for a slightly greater preponderance of the female cases of acute atrophy of the liver if this really exist.
Thesis on Acute Liver Atrophy

Twelve Drawings

R C Bruck MB