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Stewart Lecturer in Pathology, Melb. Univ.; Junior Assistant Pathologist, Melbourne Hospital; Acting Pathologist, St. Vincent's Hospital, Melbourne.

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HISTOLOGICAL STUDIES COMPARED WITH THE WASSERMANN REACTION.

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MANY years ago Fournier,[1] basing his conclusions mainly on clinical grounds, said that inherited syphilis was the great underlying cause of infant mortality, and since his day an increasing body of pathologists, basing their conclusions on certain macroscopic and microscopic appearances, have come to hold the same view; many clinicians, however, still doubting both the accuracy of the conclusions and the reliability of the standards from which they have been drawn.

The advent of the Wassermann reaction seemed to offer a means of obtaining further statistical information bearing on this question, and also a reliable method of checking the value of the criteria which have been adopted; accordingly I have examined miscrosopically sections of the liver and spleen in 100 cases coming to autopsy at the Children's Hospital, Melbourne, the series being practically consecutive, only those being discarded in which putrefactive changes introduced a possible source of error in histological or serological findings.

The blood in each of these cases was submitted to independent examination by means of the Wassermann reaction, and in all but a few cases it was obtained post mortem either from the heart or the subclavian vein, the work of Nauwerck and Weichert[2] having shown results obtained under similar conditions to be reliable, while Obregia and Bruckner,[3] who examined cerebro-spinal fluid kept in unsterilised vessels teeming with putrefactive organisms at intervals during a period of over six months, found that the syphilitic antibody is extremely resistant to putrefaction, a positive reaction being weakened or transformed into a negative one in but a few cases.

In no case was the result of the Wassermann reaction known until the histological sections had been examined and the findings noted, a diagnosis of syphilis being made if both liver and spleen showed the presence of interstitial fibrosis of varying age and activity, together with a thickening of the coats of the smaller arteries and activity of their endothelium. No case was regarded as positive which did not show the presence of these vascular changes. Schultz[4], who recently made a series of comparative studies of tissues stained by silver methods to show the presence of S. pallida and ordinary histological preparations, regarded these vascular phenomena as the essential indication.

A survey of the comparative results in this series after they were brought together has convinced me that the spleen, which is so rapidly and markedly affected by most conditions of general toxæmia, is not a thoroughly reliable guide in the histological diagnosis of syphilis. Its smaller vessels, particularly the central arteries of the malphigian bodies, readily shows signs of irritation in association with local suppuration elswhere in the body, in the

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A survey of the comparative results in this series after they were brought together has convinced me that the spleen, which is so rapidly and markedly affected by most conditions of general toxæmia, is not a thoroughly reliable guide in the histological diagnosis of syphilis. Its smaller vessels, particularly the central arteries of the malphigian bodies, readily shows signs of irritation in association with local suppuration elswhere in the body, in the

absence either of a positive Wassermann or of appearances suggestive of syphilis in other organs, while some degree of overgrowth of the fibrous frame-work was present in all but two cases in the series. On the other hand, however, the presence of the histological appearances adopted as diagnostic of syphilis in the liver may be regarded as very reliable, as the following figures will show.

Of the 100 cases examined, 54 gave a positive Wassermann reaction, 17 a partial reaction, while 29 were negative. The histological findings agreed very closely with these, for the whole 54 cases with a positive serum action were returned as positive histologically, while of those giving a partial reaction 14 were diagnosed as certainly positive, and 3 as doubtful, but probably positive; but further details of the histological peculiarities of these cases will presently be mentioned. Turning now to the cases giving a negative serum reaction—29 in all—of these, 14 cases were diagnosed as positive histologically, 5 as doubtful, and in 10 cases the histological and serum diagnosis agreed. Now, of the 14 cases diagnosed as positive, 5 had definite clinical manifestations of syphilis, and 3 of these were under mercurial treatment at the time of their death, thus leaving 9 cases with an absolute disagreement between the histological and serum diagnosis and without clinical signs in support of the histological diagnosis. But Rubens[5] has shown that many cases with inherited syphilis of a latent type only present a positive Wassermann reaction coincidentally with the onset of clinical signs, previous results having been negative, and it is more than probable that these 9 include some such cases.

Appended are the results in tabular form:—

Wassermann Reaction.		Histological Diagnosis.			
		Positive.	Doubtful.	Negative.	Total.
Positive	54	54	...	...	54
Partial	17	14	3*	...	17
Negative	29	14†	5	10	29
Total	100	82	8	10	100

* Probably positive. † Five with definite clinical manifestations.

Now, a consideration of any of the recorded investigations shows that many cases of syphilis do not give a positive Wassermann reaction, and some correction must be applied to any statistics based on this reaction. The Committee in charge of the Victorian investigation into the prevalence of syphilis have adopted as the basis for their report a correction of 40 per cent (*i.e.*, that 60 positives represent 100 cases of syphilis). If this be correct, it is obvious that there is a vastly greater measure of agreement between the histological standards of syphilis adopted and the Wassermann reaction than there is between clinical standards and the reaction.

But it may be objected that these results taken from cases chiefly of acquired syphilis do not apply to the inherited form of the disease, and, although the figures are on a small scale, Kaliski[7] con-

cludes that 98 per cent. of infants and 50 per cent. of children with hereditary syphilis give a positive reaction. The necessary correction for this class of case then lies somewhere between 2 per cent. for infants and 50 per cent. for older children; so that, even adopting these figures, the results of the present investigation are well within the limits suggested by Kaliski.

The Partial Cases.

A great difficulty is always experienced in the interpretation of partial reactions, but a consideration of the histology of the cases giving such a reaction in this series seems to make it clear that a partial reaction means the presence of a syphilitic process in a quiescent or retrogressing stage, for the majority of these cases show a retrocedent tendency, as evidenced by—

(a) hyaline degeneration of the thickened wall of the small arteries; or

(b) the absence of signs of activity in the over-grown fibrous tissue of Glisson's capsules in the liver.

Again, three of these cases which had given a definite history of syphilis, and had been under treatment for short periods, all showed very definitely this retrocedent tendency.

Syphilis as the great Underlying Cause of Infant Mortality.

Coming now to look more in detail at the effect of syphilis in causing infant mortality. If we put out of consideration for the time being all those cases in which the histological findings did not absolutely agree with the serum reaction or clinical phenomena, the facts are still very striking, for to the 54 cases giving a positive reaction must be added 5 cases with definite clinical evidence of syphilis but giving a negative Wassermann reaction, and 3 similar cases giving a partial reaction, thus bringing the total number of undoubted cases of infection up to 62 (per cent.). Fournier[5] says that "many of these children carry death in their birth," and certainly the process must start in some cases very early in intra-uterine life. For example, case No. 20 in this series shows in the liver of a child fourteen days after birth large tracts of old fibrous tissue completely inactive and undergoing retrogressive changes. So it is that very many of these cases die early during the first year. Of 44 cases in this series dying during the first twelve months, all, without exception, were diagnosed histologically as syphilitic—that is to say, all were "fibrotic"; and among these 44 infants, 26 gave a positive Wassermann reaction and 10 a partial reaction, while 8 (of which 3 were clinically definite cases of syphilis) gave a negative reaction.

Cases Dying under Twelve Months.

Total number	44
Histologically positive	44
Wassermann positive..	26
Wassermann partial	10
Wassermann negative	8*

* 3 definite clinical cases of syphilis.

But further light is thrown on the problem of the influence of syphilis in bringing about early death by a consideration of the ages and of the immediate cause of death in the 10 cases in which there was neither clinical, serological, nor histological evidence of syphilis present. Only 3 of these cases were under 4 years of age, and 4 of them were over 8, while in every case death was due to severe illness such as sarcoma of kidney (1 case), acute nephritis (1 case), cerebral abscess (2 cases), general peritonitis after operation for gastrostomy (1 case), general tubercle (1 case), rheumatic endocarditis (2 cases), broncho-pneumonia, with death on the fourteenth day (1 case).

These histories stand in marked contrast to those of the previous series (*i.e.*, cases dying under one year), in the majority of which death was ascribed to gastro-enteritis, broncho-pneumonia of short duration, rapid generalised tubercle, or mild infective disorders, while a few followed small surgical operations.

The Resistance of the Congenital Syphilitic.

Not only is syphilis responsible for much infantile mortality, but also it leads to diminished resistance to mild infective diseases, and to many sudden deaths with only slight lesions to be found post mortem. The present series abounds with illustrative examples in which the organs showed the characteristic changes and the Wassermann reaction was positive. For instance, one may quote case No. 20* a child 14 days old, with sudden collapse and death three hours after the complete cessation of a small umbilical hæmorhage, the only lesion found post mortem being a small hæmorrhage into the thymus gland; or, again, cases 39, 73, 76, with death in the first twenty-four hours from the onset of a broncho-pneumonia, with only slight lesions post mortem; or, as an example of yet another class, case No. 92, ill seven days with cough, epistaxis, and signs of cardiac dilatation, the only lesion found post mortem being a dilated heart with no valvular lesion; or, again, case No. 35†, with death after a short period of coma and acetonuria, the only gross lesion noted post mortem being œdema of the brain.

The Relation of Generalised Tubercle and Gastro-Enteritis to Fibrosis and Vascular Changes in the Liver.

It is commonly objected by those who do not accept the views advanced in this paper that the histological standards adopted are merely those of chronic inflammation, and might be caused by any chronic infective process such as tubercle or gastro-enteritis. That such may be the case in the spleen I have already stated; but that general vascular changes or fibrosis are produced by tubercle is contrary to all experience, not only in children, but in cases dying in adult life. The material in this series bearing on the point is too limited to warrant much attention, but all the cases of generalised tubercle, with one exception, gave a positive or partial Wassermann reaction, and showed generalised vascular changes; or, in other

* The numbers refer to appended table. † Wass. R. negative—clinical history of syphilis.

words, they were cases of tubercle grafted on to congenital syphilis, and running a downward course because of the devitalising influence of the inherited disease. The negative case (No. 17) did not show generalised arterial change in the liver, the only arteries affected being in the neighbourhood of the tubercular foci.

The relationship, however, between gastro-enteritis and syphilis and between gastro-enteritis and fibrosis and fatty changes in the liver is by no means so clear.

An examination of the appended table will reveal many cases showing chronic ulceration of the mucous membrane of the bowel with fibrosis of the outer coats and arterial change, in association with a positive Wassermann reaction and the following changes in the liver:—

1. Fibrosis in Glisson's capsules and between the cells.
2. Arterial changes of the " syphilitic " type.
3. Fatty degeneration.
4. Lysis of liver cells.

The last two changes were often so extreme as to leave few normal liver cells visible in any microscopic field. The difficult question to settle is whether the toxins elaborated in the diseased bowel are the direct cause of these latter degenerative changes in the liver, or whether the changes in the liver and bowel are but part of the same syphilitic process, the gastro-enteritis being merely an expression of metabolic disturbances consequent on abolished or diminished function of the liver.

The material available is not enough to settle the point, which needs further post mortem, clinical, and experimental investigation. It does, however, provide sufficient evidence to justify one in regarding the latter view as at least probable, chiefly because, in the majority of cases, the fatty change in the liver has been apparently of slow evolution and because the vascular changes affect the small arteries to a very much greater extent than the branches of the portal veins.

The Relation of Microscopic Appearances to Clinical and Post-Mortem Standards.

Although it has been pointed out that in many cases the process must start quite early in intra-uterine life, in others the inception is apparently delayed till a much later period of gestation, or even till some time after birth. Such cases may not present much enlargement of the organs nor gross fibrosis at the post mortem examination, and may easily be missed till microscopic sections are examined, the growth of new tissue not being sufficient to cause an alteration in the consistence of the organ sufficient to be evident to the examining finger. But more striking still is the fact that over half the cases do not present at all the classical picture of congenital syphilis, the only hint given to the clinician in many of them being the poor resistance of the child, and the abnormal course run by some inter-current complaint. Schultz[5], who also draws attention to

this fact, was struck by the frequency with which he found S. pallida in sections of the organs of chidren dying soon after birth with no clinical manifestations of the disease, and points to the frequency with which the diagnosis is missed by both the clinician and the pathologist, and says that "if one requires complex changes in lungs, liver, and spleen he will miss many luetic cases in young children."

A general consideration of the material dealt with leaves nothing to be added to the description of the morbid anatomy of congenital syphilis published by Professor Allen[10] in 1904, but it establishes the accuracy of the criteria he then enunciated, and consequently of the conclusions he then drew from post mortem experience with regard to the prevalence of syphilis. He then stated: "My figures must be taken for what they are, and no more general meaning must be read into them. They do not apply to the living hospital patients; they do not apply to the general community." The same remarks apply, but with greater force, to my own figures; first because the Children's Hospital, perhaps, less than any other may be taken as an index of the general population; and more important still, as Fournier, Schultz, and others have pointed out, death rapidly thins the ranks of the congenital syphilitic in the first few years of life, and so the figures derived mainly from children under 5 years must necessarily show an abnormally large percentage affected with the disease. Nevertheless, whether they belong to a hospital population or not, the loss of so many children is extremely serious from the national standpoint. The fact that out of the 100 cases now submitted considerably more than half were syphilitic should merit the most careful consideration by those interested in public health.

In conclusion, my best thanks are due to Dr. Hiller (who performed the Wassermann reactions) for many suggestions, but especially to Dr. H. D. Stephens, Pathologist to the Children's Hospital, Melbourne, who obtained for me both the opportunity of carrying out the investigation and also access to the clinical records of the cases examined.

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No.	Age.	Histological Appearances.			Serum Reaction.	Mercurial Treatment.	Clinical History.
		Liver.	Spleen.	Bowel.			
1	10 days ...	Capsule not greatly thickened. Increase in fibrous tissue in Glisson's capsule. Some pericellular fibroblastic infiltration. Vessels thick-walled, endothelium active. Definite periarteritis.	Capsule thickened. Vessels thick walled with active endothelium. Perivascular accumulations of fibroblasts.	Not examined	+ (ante-mortem.)	.....	Premature child. Mother dying of phthisis. Regarded clinically as typically syphilitic.
2	9 months	Capsule thickened. Increase in fibrous tissue in Glisson's capsules. No active fibrosis. Vessels thick-walled, endothelium active.	Great increase in fibrous stroma. Vessels thick-walled. Endothelium active. Some fibroblasts in pulp.	Not examined	+	10 days ...	Diarrhœa and vomiting intermittent for 6 months. Marked syphilitic facies. Family history—6 miscarriages, 1 still-born child, 3 others apparently healthy.
3	2 years ...	Thickened capsule. Some fatty degeneration. Great increase in fibrous tissue in Glisson's capsule, chiefly old; vessels thick-walled with active endothelium, though vascular change is patchy.	Diffuse fibrosis, chiefly old. The vessels are thick-walled, and show hyaline change. The endothelium is active.	Not examined	—	.....	Mongol idiot. Ricketty, with open frontanelle. Death from bronchopneumonia. 10 days' illness.
4	1 month ...	Increase of fibrous tissue and fibroblasts in Glisson's capsules. Some pericellular fibroblasts. Vessels are thick-walled. Endothelium active. Some congestion.	Some increase in fibrous stroma. The vessels are thick-walled, showing both endarteritis and periarteritis.	Not examined	Partial ...	.....	Hypertrophic stenosis of pylorus.
5	3 months	Capsule slightly thickened. Extreme fatty degeneration. Marked fibroblastic activity in Glisson's capsules. Vessels thick-walled, with very active endothelium. Perivascular cell accumulation.	Capsule thickened. Vessels thick-walled with marked endarteritis and periarteritis. Diffuse fibrosis.	Not examined	+	Treatment for some months.	Marked syphilitic stigmata. Gastro-enteritis.
6	3 years ...	Well marked multilobular fibrosis. Some activity at present. Vascular changes irregular. Doubtful, but probably positive.	Vessels thick-walled. Endothelium active. Very little perivascular cell accumulation. Capsule thickened and some increase in fibrous stroma.	Not examined	—	?	Nephritis.
7	17 days ...	Cirrhosis active and chiefly pericellular. Vascular changes patchy, but some vessels are thick-walled, with active endothelium and perivascular irritation.	Diffuse fibrosis and fibroblastic infiltration. Vessels thick-walled, with active endothelium and definite periarteritis.	Not examined	+	.....	Constipation—bowels never opened naturally since birth, never thriven, small child, rash on buttocks and labia.
8	.....	Active fibroblastic infiltration in Glisson's capsules and between cells. Vessels show very active endothelium and fibroblasts in their middle and outer coats.	Capsule slightly thickened. Diffuse fibroblastic infiltration. Vessels thick-walled, fibroblastic infiltration of outer coat. Great activity of endothelium.	Not examined	—	.....	Not obtainable.

No.	Age.	Histological Appearances.			Serum Reaction.	Mercurial Treatment.	Clinical History.
		Liver.	Spleen.	Bowel.			
	12 years ...	Active fibro-blastic infiltration of Glisson's capsules. Vascular changes irregular. Endothelial activity more marked than changes in outer coats.	Capsule thickened. Large areas of caseation. Increase in fibrous framework. Vessels —endothelium active, fibrosis of outer coats, perivascular cell accumulation.	Not examined	+ (ascitic fluid.)	.....	Hodgskin's disease.
10	1 year and 2 months.	Some increase in fibrous tissue in Glisson's capsules. Active fibroblasts. Microscopic tubercles. Vascular changes are definite.	Capsule thickened, irregular overgrowth of fibrous tissue of stroma. Vascular changes irregular, some showing thick walls and active endothelium	Not examined	+	.....	Generalised tubercle.
11	10 years ...	Very little increase of fibrous tissue. No vascular changes.	Capsule thickened. Vessels are thick-walled and hyaline. No active fibrosis or peri-articular irritation. Some activity of endothelium of arteries	Not examined	—	.....	Cerebral abscess sequent. On middle ear suppuration.
12	4 years and 9 months	A little fibroblastic activity in Glisson's capsules. No changes in smaller vessels. Some fibroblastic infiltration and endothelial activity in the larger arteries. No engorgement.	Capsule thickened. Some increase in fibrous stroma. Patchy thickening of vessels with endarteritis.	Not examined	—	.....	Pericarditis { 1 year's Endocarditis { duration. Pneumonia.
13	.....	Liver—multilobular and pericellular fibrosis. Vessels fibrosis of outer coats. Endarteritis well marked.	Thickened capsule. Increase of fibrous framework. Vessels thick-walled, with well-marked endarteritis and perivascular fibrosis.	Not examined	+	.....	Diabetes. Rapid onset of coma and death. No suspicion of syphilis clinically.
14	.....	Capsule greatly thickened. Increase of fibrous tissue of varying age in Glisson's capsules. Pericellular cirrhosis. Vessels thick-walled. Endarteritis, but very little periarteritis.	Well-marked fibrosis and thickening of walls of arteries which have undergone hyaline changes. Patchy endarteritis.	Not examined	+		No history obtainable, but gland sent for examination with specimens, was an old organising gumma.
15	.....	Thick capsule. Great increase of fibrous tissue in Glisson's capsules. Foci of active growth. Vessels thickened, some showing hyaline change. Endarteritis.	Capsule thickened. Great increase in fibrous stroma. Vessels hayline degeneration of thickened walls. Endothelium active.	Not examined	Partial ...	Treated in Out-patients' Dept.	Regarded as a syphilitic; anæmic.
16	9 years ...	Slight thickening of capsule. Slight fibrosis. No vascular changes.	Some fibrosis. Thickening of coats of all the small vessels. Hyaline change in outer coats. A few vessels show activity of endothelium.	Not examined	—	.....	Purulent meningitis. Duration of illness, 3 days. Considered at post-mortem to be probably syphilitic. Thymus enlarged. Liver and spleen enlarged and firm.

No.	Age.	Histological Appearances.			Serum Reaction.	Mercurial Treatment.	Clinical History.
		Liver.	Spleen.	Bowel.			
17	1 year and 4 months.	Capsule thickened, slight increase of fibrous tissue in Glisson's capsules. Some microscopic tubercles. No general vascular changes. Slight fatty degeneration.	Capsule greatly thickened. Areas of fibrosis with more active cell infiltration. Vessels—walls thickened and hyaline. Marked endareritis.	Not examined	—	.....	Tubercular lymph glands. History incomplete.
18	3 weeks ...	Active fibroblastic infiltration of Glisson's capsules and between the cell columns. Vessels thick-walled, with very active endothelium.	Capsule thickened. Increase in fibrous tissue of framework. Active fibrosis. Vessels show thick walls with definite endarteritis.	Not examined	+	.....	Father had syphilis. Child died of cellulitis of leg in 36 hours.
19	5 months	Thickening of capsule. Multilobular and pericellular cirrhosis. Vessel walls thickened. Endothelium active. Some fatty degeneration.	Great increase in fibrous stroma. Vessels thickened. Perivascular fibrosis. Endarteritis with a tendency to thrombosis in some of smaller vessels.	Not examined.	+	.....	Cerebro-spinal meningitis. Ill 3 weeks. Terminal broncho-pneumonia.
20	2 weeks ...	Increase in fibrous tissue in Glisson's capsules ,but no sign of activity. Vessels thick-walled, with hyaline change in walls. Some proliferation of endothelium. Active pericellular fibroblastic infiltration.	Diffuse fibrosis more active than in the liver. Vessels thick-walled, showing little degeneration and active endothelium.	Not examined	+	.....	Umbilical hæmorrhage with sudden collapse 3 hours after hæmorrhage had been completely controlled. Petechial hæmorrhages in pleura, hæmorrhage into thymus.
21	.....	Increase in fibrous tissue in Glissons' capsules. Vessels thick-walled, with marked endarteritis. Pericellular fibroblastic infiltration. Slight fatty degeneration.	Not examined	Not examined	+	.....	
22	.....	Not examined	Capsule slightly thickened. Marked fibrosis and thickening of vessels, which show definite endarteritis and periarticular fibrosis.	Not examined	+	.....	Sub-acute abscess of left kidney.
23	.....	Capsule not thickened. Well marked, multilobular and pericellular fibrosis; great thickening of vascular walls, and active round cell infiltration around vessels—marked endarteritis.	Capsule greatly thickened. Diffuse fibrosis. Thickening and hyaline change in vascular walls. Endothelium active.	Not examined	Partial ...	.....	
24	4 years and 6 months.	Fatty liver with active fibrosis of and thick vessels, but no endothelial activity. No periarticular cell accumulation.	Some fibrosis with thick-walled vessels, but no endothelial activity.	Not examined	—	.....	Not syphilitic. Old æsophageal stricture following KOH. Gastrostomy and general peritonitis. History of gastro-enteritis intermittent for some years.

No.	Age.	Histological Appearances.			Serum Reaction.	Mercurial Treatment.	Clinical History.
		Liver.	Spleen.	Bowel.			
25	10 days ...	Fibroblastic infiltration between cells. Endothelium of capillaries very active. No great thickening of arteries.	Capsule thickened. Great fibrosis, vascular thickening and endarteritis.	Atrophic	Partial ...	.....	Miserable child—red about anus. Fed on condensed milk. Hæmorrhagic effusion on dura under parietal bone. Umbilicus not healed.
26	.....	Fibrosis. Thickening of vascular walls with marked periarteritis and endarteritis.	Diffuse fibrosis. Marked thickening of walls of all small vessels. Endothelium active. Periarterial fibrosis.	Not examined	Partial ...	.....	
27	3 years and 4 months	Slight increase in fibrous tissue in Glisson's capsules of varying age. Vessels show thickening of outer coats and activity of endothelium.	Capsule thickened. Diffuse fibrosis. Vessels as in ilver.	Not examined	—	.....	
28	9 months	Increase in fibrous tissue in Glisson's capsules. Active foci of fibroblasts among older fibrous tissue. Vessels thick-walled. Endothelium active.	Not examined	... Not examined	Partial ...	.....	Tubercular meningitis, generalised miliary tubercle. Nothing suggestive in family history.
29	2 months	Fatty liver, with some multilobular and pericellular cirrhosis. Activity round vessels, which are thick-walled. Endothelium active.	Capsule not thickened. Diffuse fibrosis. Vascular walls thickened. Endothelium active. Patchy periarteritis.	Not examined	+	.....	Congenital syphilitic asthenia. Gastro-enteritis since birth.
30	4 months	Some fibrosis, chiefly pericellular. Vessels thick-walled with marked endarteritis.	Great thickening of the capsule. Diffuse fibrosis, with marked thickening of vessel walls and endarteritis.	Marked ulceration of colon. Sub-acute inflammation with old syphilis of small bowel.	+	.....	Catarrhl enteritis and colitis for 1 month; cough 7 days before death. Definite signs of broncho-pneumonia 2 days before.
31	7 years ...	Some old fibrosis, with more active foci. Pericellular fibrosis and fibroblasts. Vessel walls hyaline and thickened. Endothelium active.	Diffuse fibrosis with thick-walled vessels. Endothelium active.	Not examined	+	.....	Rheumatic fever when æt. 4. Some endocarditis then. 3 weeks before admission sudden left-sided heimplegia, without loss of consciousness. Some mitral endocarditis. Sudden death. Post-mortem pulmonary œdma, with right-sided cortical hæmorrhage.
32	7 months	Slight increase of fibrous tissue in Glisson's capsule. Thick-walled vessels with endarteritis and periarterial cell accumulation.	Diffuse active fibrosis. Vessels thick-walled and hyaline. Endarteritis.	Not examined	—	?	Rash since birth. Considered syphilitic. Death from broncho-pneumonia. Ill 2 days. Sudden heart failure. History of 1 miscarriage previous to her birth.

No.	Age.	Histological Appearances.			Serum Reaction.	Mercurial Treatment.	Clinical History.
		Liver.	Spleen.	Bowel.			
33	1 year ...	Slight increase of fibrous tissue in Glisson's capsule of varying age. Vessels show endarteritis. Thickening of walls, some hyaline, some showing active infiltration. Slight fatty degeneration.	Thickening and cellular infiltration of capsule. Fibroblastic infiltration of coats of all smaller vessels, with marked endarteritis and periarteritis.	Not examined	—	.....	Five others in family. 2 miscarriages, 1 stillborn; coryza for 3 months; mentally deficient; chokes when taking food. Breast fed. Died suddenly. Only lesion noted post-mortem was œdema of brain.
34	9 months	Fibro-fatty, active fibro-blastic infiltration, chiefly in Glisson's capsuels. Vessels thick-walled. Endothelium active. Miliary tubercles throughout section.	Diffuse fibrosis. Vessels thick-walled. Endothelium active. Periarticular cell accumulation.	Not examined	Partial ...	Treatment for some weeks in O.P.	Congenital syphilitic. Generalised tubercle. Both parents gave + Wasserman R. 2 miscarriages; 5 children, all with positive Wassermann reaction. Ill 2 days before death with broncho-pneumonia.
35	11 years	Some increase in fibrous tissue in Glisson's capsules, both old and active. Vessels thickened. Marked endothelial activity. Active pericellular fibro-blastic infiltration.	Some thickening and hyaline change in walls of small arteries. Endothelial activity. Perivascular fibrosis.	Not examined	—	.....	Ill 1 month with pericarditis, endocarditis. Death from broncho-pneumonia.
36	11 years ..	Marked multilobular and pericellular cirrhosis. Vessels show great thickening of walls, only a few showing any signs of activity.	Extreme diffuse fibrosis, very little trace of spleen pulp being visible. Vessels for most part have hyaline walls. A few show active endothelium.	Not examined	+	.....	Syphilitic ænemia. Enlarged spleen and liver. Death following copious hæmatemesis and malæna. Said to have been well till a few days before death.
37	2 years and 6 months.	Multilobular fibrosis, but condition of vessels is doubtful. A few are thick-walled and hyaline, a few show endothelial activity.	Capsule slightly thickened. No great fibrosis, but vascular changes are well marked.	Not examined	+	.....	Death from pneumonia on the third day. Nothing suggestive of syphilis clinically, or in family history.
38	7 years ...	Multilobular cirrhosis Vessels thick-walled. Endothelium active. Some perivascular fibrosis.	Capsule thickened. Slight fibrosis. Vessel walls thickened and endothelium active.	Not examined	+	.....	No suspicion of syphilis. Ill 17 days. Measles. Lobar-pneumonia, empyema.
39	11 weeks	Multilobular and pericellular fibrosis, not showing much sign of activity. Slight fatty degeneration. Vessels have thick walls, with hyaline degeneration. Patchy endothelial activity.	Slight fibrosis. Great engorgement and hæmorrhage. Vessels thick-walled and hyaline. Endothelium generally active.	Not examined	+	.....	A seven-months child. Death from broncho-pneumonia on third day.

No.	Age.	Histological Appearances.			Serum Reaction.	Mercurial Treatment.	Clinical History.
		Liver.	Spleen	Bowel.			
40	13 years ...	Marked multilobular and pericellular fibrosis. Vessels very thick-walled. Endothelial activity patchy in distribution.	Capsule slightly thickened. Diffuse fibrosis. Larger vessels have hyaline walls. Active fibro-blastic infiltration with endarteritis and periarteritis on smaller ones.	Not examined	+	.....	Born in Kew Asylum Deaf and dumb. Syphilitic purpura. Syphilitic. ænemia. Gastro-enteritis. Ill 1 month.
41	1 month ...	Fibro-fatty. Extreme degree of fatty change. Vessels thick-walled with active endothelium. Perivascular cell accumulation.	Capsule thickened; increase of fibrous stroma. Vessels as in liver.	Not examined	+	.....	Syphilitic family. Others under treatment in Out-patients' Department. Death from chronic suppurative meningitis. 3 weeks' illness.
42	4 months	Multilobular and pericellular fibrosis of liver. Vessels show marked endothelial activity.	Diffuse fibrosis. Vessels thick-walled with active endothelium.	Not examined	—	Hyd. c Cret. and Grey oil for 2 months.	Regarded as syphilitic. Mother looks syphilitic. 3 previous children died in infancy. Gastro-enteritis of 3 months' duration.
43	8 months	Fibro-fatty. Some active foci of fibrosis multilobular and pericellular, with hyaline change in coats. Endarteritis.	Capsule greatly thickened. Diffuse fibrosis. Vessels as in liver.	Engorgement of sub-mucous vessels. Slight ulceration of colon.	—	.....	Entero-colitis intermittent Fatal attack only lasted 24 hours. ? Status lymphaticus. Thymus very large, post-mortem.
44	9 weeks ...	Old pericellular fibrosis, with multilobular still showing signs of activity. Small vessels show marked irritation of endothelium.	Capsule thickened. Slight fibrosis. Vessels show fibrosis of outer coats, and activity of endothelium.	Not examined	+	.....	Death 36 hours after onset, of strangulated hernia. Operation note: "Cæcum and appendix in sac—not injured beyond repair. Returned. Child greatly shocked." Post-mortem wound healthy, no peritonitis; no gangrene of contents of sac.
45	4 years ...	Pericellular fibrosis, but no vascular changes	Capsule thickened, fibrosis, and some changes, particularly in central arteries.	Not examined	— (ante-mort.)	.....	Sarcoma of kidney.
46	1 yea and 3 months.	Some increase in fibrous tissue. Vessels doubtful.	Capsule thickened. Fibrous framework decidedly increased. Vessels doubtful.	Not examined	Partial ...	.....	Broncho-pneumonia. Death on 14th day.

No.	Age.	Histological Appearances.			Serum Reaction.	Mercurial Treatment.	Clinical History.
		Liver.	Spleen.	Bowel.			
47	10 months	Pericellular fibrosis. Some fibro-blastic activity in Glisson's capsules. Vascular changes, chiefly in larger vessels, which are thick-walled, and show active endothelium. No fatty change.	Capsule thickened. Slight increase in fibrous stroma, Some thickening of vessels with irritation of endothelium.	Ulceration of colon with mono-nuclear leucocytes in ulcerated area. Round cell accumulation in sub-mucosa. Vessels thick-walled. Endothelium active.	+	.....	Bowel trouble since birth. 8 other children. Brother died, æt. 2 months. Wasermann +. Fatal attack of acute gastro-enteritis, lasting 1 week. No acute changes, post-mortem.
48	4 years and 6 months.	Multilobular fibrosis. Fatty degeneration. Vessels thick-walled, hyaline, with marked endarteritis.	Capsule thickened. Diffuse fibrosis. Vessel walls thickened and hyaline. Endothelium active.	Not examined	+	.....	Diphtheria with retained tube. In hospital 6 months. Then contracted measles with bronchitis, and died on the second day.
49	4 months	Multilobular fibrosis of varying age. Vessels thick-walled, with endarteritis and periarteritis. Uniform engorgement. No fatty change.	Capsule greatly thickened. Diffuse fibrosis. Vessels as in liver.	Not examined	+	.....	Cough for 5 weeks. Gastro-enteritis 4 days before death. 7 lb. weight. Death from malnutrition, no lesion being found in bowel, post-mortem.
50	4 months.	Fibro-fatty liver. Multilobular fibrosis. Vessels are doubtful.	Capsule greatly thickened. Marked fibrosis and thickening of coats of vessels which show endarteritis.	Ulceration of mucous membrane. Muscle thickened. Chronic inflammation.	Partial ...	.....	Entero-colitis, 1 month's duration. No other children. No miscarriages.
51	7 months	Fibrosis—vessels thick-walled, endothelium active. Old multilobular and pericellular fibrosis.	Capsule thickened. Increase in fibrous framework. Vessels thickened. Endothelium active.	Chronic inflammation of colon and small bowel, with sub-acute exacerbation. Great ulceration. Chronic fibrosis of muscle coats. Vessels as in liver.	—	?	Entero-colitis of 3 months' duration. Clinically regarded as syphilitic. Nothing suggestive in family history.
52	10 months	Fibro-fatty. Multilobular and pericellular fibrosis. Foci of marked activity. Vessels show endarteritis and some fibrosis of outer coats.	Diffuse fibrosis. Vessels fibrosis of outer coats and activity of endothelium.	Chronic inflammation with engorgement of sub-mucous vessels, and fibrinous exudate on surface. Vessels thickened.	+	.....	Entero-colitis.
53	2 months	Old fibrosis. Fatty liver. Vessels thickened with endarteritis and periarteritis.	Capsule thickened. Vessels thickened, and as in liver. Extreme diffuse fibrosis.	Old fibroid changes in outer coats. Sub-acute enteritis.	Partial ...	.....	
54	6 weeks ...	Old multilobular fibrosis. Some increase of fibroblasts. Between columns of liver cells. Vessels thick-walled, with definite endarteritis.	Capsule thickened. Great increase in fibrous stroma. Vessels thick-walled, with definite endarteritis.	Old fibrosis of muscle coat, with thick-walled vessels. Sub-acute inflammation.	+	.....	Ill since birth. Undersized perionychia of right index finger. Enteritis. Broncho-pneumonia.

No.	Age.	Histological Appearances.			Serum Reaction.	Mercurial Treatment	Clinical History.
		Liver.	Spleen.	Bowel.			
55	6 months	Fibro-fatty, fibrous tissue, chiefly old. Vessels thick-walled. Endothelum active.	Not examined	Not examined	—	.....	Broncho-pneumonia. Empyema, 2 months.
56	6 weeks.	Extreme fatty change. Great increase in fibrous tissue in Glisson's capsules. Patchy endothelial activity.	.....	Some ulceration	—	.....	Ill 3 weeks. Entero-colitis, but only slight lesions found post-mortem. Small, poorly-developed child.
57	5 months	Multilobular fibrosis. Pericellular fibroblasts. Vessels thick walled-with marked endarteritis. Some increase in fat.	Capsule thickened. Diffuse fibrosis. Vessels as in liver.	Sub-acute enteritis and colitis.	Partial	.....	Entero-colitis. Ill for 2 months. No suspicion of syphilis clinically.
58	8 months	Fibrosis. Thick-walled vessels, with endarteritis.	Capsule thickened. Diffuse fibrosis. Vessels—fibrosis of outer coats.	Not examined	+ (ante-mortem.)	.....	Ill 3 weeks. Colitis long continued. Mild inflammatory changes at post-mortem.
59	9 months	Fibro-fatty liver. Old fibrosis with more active areas. Smaller arteries are thick-walled with endarteritis.	Capsule slightly thickened. Slight fibrosis. Patchy endarteritis of vessels, which are thick-walled.	Not examined	+	.....	Entero-colitis. Broncho-pneumonia. Slight mitral endocarditis, recent. Ill 3 weeks.
60	7 months	Capsule thickened. Increase of fibrous tissue in Glisson's capsules. Pericellular fibrosis active. Vessels thick-walled. Endothelium active	Capsule thickened. Diffuse fibrosis. Vessels as in liver.	Not examined	+	.....	Erysipelas and general peritonitis. Ill 1 week. Chronic mild entero-colitis.
61	3 months	Multilobular and pericellular cirrhosis. Vessels thick-walled, with endarteritis.	Fine fibrosis. Vessels thick-walled. Endothelium active.	Not examined	—	—	Blue since birth. No anatomical lesion found, but heart dilated. Six other healthy children. No history of vomiting.
62	1 year and 7 months.	Capsule greatly thickened. Old multilobular fibrosis with activity. Some pericellular fibrosis. Vessels thick-walled. Endarteritis. No fat.	Capsule thickened. Marked diffuse fibrosis and vascular thickening with endarteritis.	Old chronic fibrosis in muscle coats. Ulceration of mucous membrane. Vessels as in liver. Terminal engorgement.	—	.....	Entero-colitis. Acutely ill 7 days. No suspicion of syphilis clinically.
63	3 months	Extensive fatty change. Active fibroblastic infiltration in Glisson's capsules. Vessels show fibrosis of outer coats and, activity of endothelium.	Capsule thickened. Diffuse fibrosis, and vascular thickening with endarteritis.	Chronic fibrosis of muscle coats and mucous membrane; little glandular tissue remaining. Terminal engorgement. Vessels as in liver. Great overgrowth of endothelium in sub-peritoneal lymphatics.		.....	Acutely ill 8 days. Entero-colitis. Colon showed evidence of chronic change at post-mortem.
64	1 year ...	Extreme fatty change. Old fibrosis with very active foci in Glisson's capsules. Vessels thick-walled. Periarteritis and endarteritis.	Capsules thickened. Diffuse fibrosis. Vessels as in liver.	.....	Partial ...	.....	Entero-colitis. Ill 9 days.

No.	Age.	Histological Appearances.			Serum Reaction.	Mercurial Treatment.	Clinical History.
		Liver.	Spleen.	Bowel.			
65	5 months	Extreme fatty change. Old fibrosis in Glisson's capsules of varying age. Vessels show great irritation of endothelium and some fibrosis of outer coats.	Capsule thickened. Fine fibrosis. Vessels as in liver. Some hyaline change in coats of central arteries.	Old chronic fibroid changes in sub-mucosa and muscle coats. Terminal engorgement.	+	Treatment with Hyd. c Cret. for 6 weeks.	Poorly nourished child. Regarded as syphilitic. Acutely ill 17 days. Death from entero-colitis. Family history not obtainable.
66	1 year and 6 months.	Fibrosis old and active. Vessels are not characteristic.	Capsule thickened. Fibrosis. Vessels show activity of endothelium and thickening of coats.	Not examined	—	.....	Entero-colitis. Broncho-pneumonia.
67	3 years ...	Fibro fatty multilobular cirrhosis, with very active pericellular infiltration. Vessels thick-walled. Marked endarteritis.	Capsule thickened. Some increase in fibrous framework. Vessels as in liver.	Tubercular ulceration. Old fibroid condition. Vessels thick-walled. Endarteritis and periarteritis.	+	.....	Measles and otorrhœa, followed by tubercular meningitis. Tubercular ulceration of bowel. Old tubercle at right apex. Interstitial keratitis.
68	5 months	Fibrosis, old and active. Vessels thickened. Marked endarteritis.	Capsule slightly thickened. Diffuse fibrosis. Vessels as in liver.	Not examined	+	.....	
69	4 months	Fatty liver. Scarcely any normal cells now visible. Multilobular cirrhosis. Vessels thick-walled. Endothelium active.	Not examined	Colon—some ulceration. Marked catarrhal nflammation. Chronic catarrhal enteritis.	+	.....	Gastro entero-colitis of 4 weeks' duration. Infant life child.
70	7 years and 10 months.	Fibrosis in Glisson's capsules and between cells, chiefly old. Vessels doubtful. Some increase in fat.	Thick capsule. Old fibrosis. Vessels thick-walled. Hyaline doubtful.	Not examined	—	.....	Death from acute acidosis. No lesion found, post-mortem. Constipation and head-ache for 4 weeks. Urine contained diacetic acid and acetone.
71	5 months	Capsule thickened. Slight fibrosis. Marked fatty change. No active fibrosis. Vessels thick-walled, with marked endarteritis.	Capsule thickened Diffuse fibrosis. Definite endarteritis and fibrosis of outer coats of small arteries.	Some ulceration and subacute inflammation. Old fibrosis of muscle coat. Vessels as in spleen.	+	.....	Ill 8 days. Convulsions, intestinal toxæmia.
72	8 months	Fibro-fatty. Great increase in fibrous tissue in Glisson!s capsules. Marked atrophy of liver cells. Vessels are thick-walled. Endothelium active.	Not examined	Colon—ulceration of mucous membrane. Old fibrosis of muscle coat. Vessels as in liver.	+	.....	Entero-colitis inter-mittent for 2 months. Traces of blood in motions at times.
73	7 months	Fibrosis pericellular and in Glisson's capsules—some very active foci. Vessels thick-walled. Endothelium active.	Diffuse fibrosis. Vessel as in liver.	Not examined	Partial ...	.....	Broncho-pneumonia. Ill 1 day. Old history of diarrhœa and vomiting. 2 other children. 1 miscarriage.

No.	Age.	Histological Appearances.			Serum Reaction.	Mercurial Treatment.	Clinical History.
		Liver.	Spleen.	Bowel.			
74	1 year ...	Intense pericellular fibrosis. Some increase in fibrous tissue of Glisson's capsules. Some fatty degeneration. Vessel walls thickened Fibrosis and endarteritis.	Diffuse fibrosis. Vessels as in liver.	Not examined	—	.....	Scalds on face and forearm, not extensive. Died in 5 days. Chloroform for dressing, 3 times.
75	4 months	Fibro-fatty. Fibrosis, old in Glisson's capsules. No activity. Vessels thick-walled with active endothelium.	Capsule thickened. Great increase in fibrous framework. Vessels as in liver.	Old chronic entero-colitis, with sub-acute inflammation. Some ulceration.	Partial	.....	Broncho-pneumonia Entero-colitis.
76	4 months	Capsule greatly thickened. Great increase in fibrous tissue of Glisson's capsule. Active pericellular fibrosis. Vessels thick-walled. Endothelium active.	Capsule thickened. Diffuse fibrosis. Vessels as in liver. Hæmorrhage and engorgement.	.....	+	.....	Broncho-pneumonia. Ill 1 day. Heart failure and sudden collapse. No family history obtainable.
77	10 months	Extreme fatty change, few normal cells persisting. Capsule thickened. Increase in fibrous tissue with active foci. Vessels show endarteritis and fibrosis of coats.	Capsule greatly thickened. Great increase in fibrous tissue of framework. Vessels very thick-walled with mark-endarteritis.	Not examined	+	.....	Measles 2 months before death followed by entero-colitis and broncho-pneumonia. Was regarded as a congenital syphilitic. Family history not obtainable.
78	5 years ...	Fibrosis—chiefly multilobular with active foci of cell accumulasion. Vessels thick walled, hyaline Active endothelium	Some increase in fibrous framework. Vessels as in liver. Sub-capsular infarction.	Not examined	+	.....	Ill 1 month. Lymphatic laukæmia. Death from broncho-pneumonia on second day.
79	7 months	Capsule not greatly thickened. Increase in fibrous tissue of Glisson's capsules with foci of round-celled activity. Vessels thick-walled. Endothelium very active.	Capsule thickened. Great fibrosis, chiefly old. Vessels thick-walled, with active endothelium.	Not examined	—	.....	Ill 5 weeks, diarrhœa. Death from broncho-pneumonia. No lesion in bowel, post-mortem.
80	5 months	Fibro-fatty. Fibrosis of varying age. Small tubercles. Vessels everywhere show thickening of walls and endothelial activity.	Capsule thickened. Diffuse old fibrosis. Vessels as in liver. Miliary tubercles throughout the section.	Not examined.	+	.....	Entero-colitis. Ill 8 weeks. Tubercular broncho-pneumonia. Generalised tubercle, post-mortem.
81	8 years ...	Slight interlobular fibrosis, chiefly old, with a few round cells. Vessels are not characteristic.	Some increase in fibrous framework old. Vessels are not thick walled.	Not examined	—	.....	Ill 1 month. Olden endocarditis. Acute rheumatism and endocarditis. Heart failure.
82	4 years ...	Capsule thickened. Multilobular fibrosis of varying age. Active pericellular fibroblastic infiltration. Vessels thick-walled. Endothelium active.	Capsule thickened. Vessels show endarteritis and thickened hyaline walls.	Not examined	+	.....	Ill 6 days. Started with boil on scalp. Marked albuminuria. Post-mortem, septic broncho-purulent, meningitis. Hæmorrhagic exudate over cordex of brain on left side, and some atheroma of aorta.

No.	Age.	Histological Appearances.			Serum Reaction.	Mercurial Treatment.	Clinical History.
		Liver.	Spleen.	Bowel.			
83	11 years ...	Capsule thickened. Marked pericellular fibrosis, inactive. Vessels show endarteritis and fibrosis of outer coats.	Capsule thickened. Increase in fibrous stroma. Perivascular fibrosis. Vessels as in liver.	Not examined	+	.....	Convulsions since infancy. Meningo - encephalitis. Ill 1 day. Considered to be syphilitic at post-mortem.
84	9 weeks ...	Active fibro-blastic change in Glisson's capsules and between cell columns. Fatty liver. Vessels—endarteritis and fibro-blastic infiltration of outer coats.	Not examined	Not examined	+	.....	Gastritis and some enteritis for 1 week. Paralytic distension of the bowel. Post-mortem:— "Moderate colitis, considerable enteritis.
85	1 year and 7 months.	Capsule thickened. Fibrosis of varying age, chiefly multilobular. Some pericellular fibrosis. Vessels thick-walled. Some perivascular irritation and endarteritis.	Capsule slightly thickened. Increase in fibrous stroma, inactive. Vessels show endarteritis and hyaline walls.	Intense ulcerative colitis.	+	.....	Diarrhœa and vomiting, intermittent for 6 weeks Some blood in stools.
86	1 year and 3 months.	Increase of fibrous tissue in Glisson's capsule. Inactive. Vessels show irritation of endothelium and thickened hyaline walls. No tubercle.	Capsule thickened. Some active cell infiltration of deeper layers. Vessels thick-walled, and hyaline. No tubercle	Not examined	Partial ...	.....	Empyema. Ill 2 months first with tuberculous broncho-pneumonia.
87	13 years ...	Some increase in fibrous tissue around lobules. Vessels show some thickening of walls but no endothelial activity.	Slight fibrosis and thickening of capsule. No vascular change except in central arteries of malphigian bodies. In these, endothelium is active.	Not examined	—	.....	Acute nephritis. Suppression of urine.
88	1 year ...	Active fibrosis, but no endothelial activity in smaller arteries. Great fatty change.	Not examined	Not examined	—	.....	Ill 6 weeks. Diarrhœa, with slime and blood. Entero-colitis and broncho-pneumonia.
89	2 months	Capsule thickened. Great increase of fibrous tissue in Glisson's capsules. Vessels thick-walled and hyaline. Endothelium active. Great fatty change.	Great thickening of capsule. Vessels thick-walled and hyaline. Endothelium shows signs of irritation.	Not examined	Partial ...	Active treatment for 3 weeks.	Enteritis for 11 weeks. Malnutrition.
90	3 years and 9 months.	Capsule greatly thickened, old and recent fibrosis. Multilobular and pericellular vessels show fibrosis of coats, and endarteritis. Perivascular irritation.	Some increase in fibrous stroma. Vessels thick-walled, with active endothelium.	Old chronic fibroid change. Some ulceration of mucous membrane of colon.	—	.....	Acute anæmia. Only child, no miscarriage.
91	1 year ...	Old and active fibrosis in Glisson's capsules. Some pericellular fibrosis. Vessels thick-walled. Endothelium active.	Slight increase in fibrous framework. Vessels as in liver, with marked periarterial irritation.	Not examined	—	.....	

No.	Age.	Histological Appearances.			Serum Reaction.	Mercurial Treatment.	Clinical History.
		Liver.	Spleen.	Bowel.			
92	12 years ...	Multilobular and pericellular fibrosis. Vessels thick-walled, with hyaline change in walls. Endothelium active.	Capsule greatly thickened. Increase in fibrous stroma. Vessels as in liver.	Not examined	+	.....	Ill 7 days, cough, vomiting, and epistaxis. Mitral regurgitation. No history of rheumatism. Post-mortem; Dilated heart, no valvular lesion. Slight pericarditis.
93	4 years ...	Old multilobular fibrosis. Very little evidence of activity. Old pericellular fibrosis. Vessels show endothelial activity, with thick hyaline walls.	Capsule thickened. Old fibrosis Vessels thick-walled, hyaline. Endothelium of smaller vessels show signs of irritation.	Not examined	+	.....	
94	2 years and	Multilobular fibrosis of varying age. Vessels show activity of endothelium, fibrosis of outer coats, and perivascular irritation.	Capsule thickened. Diffuse fibrosis. Vessels as in liver.	Not examined	+	.....	Ill 6 days, tubercular meningitis.
95	1 year ...	Fibrosis in Glisson's capsules and pericellular, inactive for most part. Vessels show endothelial actitvity.	Capsule slightly thickened. Increase of fibrous stroma. Vessels show activity of endothelium.	Not examined	Partial ...	.....	Ill 6 weeks with measles. Temperature 100°. Broncho - pneumonia, but only slight lesions, post-mortem.
96	5 years ...	Capsule not greatly thickened. Multilobular cirrhosis of varying age. Active pericellular fibrosis. Vessels show endarteritis and fibrosis of outer coats.	Capsule thickened. Small vessels as in liver. Hyaline change in walls of larger vessels.	Not examined	+	.....	Acute nephritis 10 days after discharge from Diphtheria Hospital; uræmic, 2 days.
97	3 years ...	Old and active fibrosis in Glisson!s capsules and pericellular. Vessels thick-walled, with active endothelium. Some fatty accumulation.	Capsule thickened. Old diffuse fibrosis. Vessels as in liver.	Not examined	+	.....	Pneumonia. Death on the third day. Temperature never above 100°.
98	7 years ...	Multilobular fibrosis, old and active. Vessels thick-walled, with endarteritis.	Great thickening of capsules and fibrosis. Vessels show marked changes as in liver.	Not examined	+	.....	Was at school 4 days before death. Purulent meningitis. No focus found.
99	12 years ...	Extreme grade of multilobular cirrhosis, also some pericellular fibrosis. Vessels thick-walled, with endarteritis.	Capsule thickened. Great increase in fibrous stroma. Vessels thick-walled, and endothelium very active.	Not examined	+	.....	Old standing cirrhosis of liver with ascites. No history of alcohol. Death from toxæmia 10 days after performance of an omentopexy.
100	7 years ...	Capsule greatly thickened, slight fibrosis and active fibroblastic infiltration in Glisson's capsules. Vessel thick-walled Endothelium active.	Capsule thickened. Some increase in fibrous framework. Vessels show activity of endothelium and fibro-blastic infiltration of outer coats. Some hæmorrhage.	.....	—	.....	Diphtheria. Death from toxæmia. No family history obtainable.

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