Indications.

	Number.	Cured, mother.	Death, mother.	Death, child.	Stillborn.
Contracted pelvis ..	40	33	7	6	2
Abnormal presentation	3	1	2	2	1
Post-maturity ..	12	12	..	..	..
Atresia vagina and rigid cervix ..	7	7	..	2	..
Osteomalacia ..	8	8	..	1	..
Impacted twin ..	2	1	1	..	..
Procidentia ..	1	..	1	..	1
Old multipara ..	2	2	..	..	..

Parity.

Parity.	Number.	Deaths.		
		Mother.	Child.	Stillborn.
1st ..	45	5	2	4
2nd ..	14	3	..	..
3rd ..	9	1	..	1
4th ..	4	..	..	1
5th ..	..	..	..	..
6th ..	..	..	..	..
7th ..	2	2	1	1
8th ..	..	..	..	..
9th ..	1	..	..	..

Age.

Age.	Number.	Deaths.		
		Mother.	Child.	Stillborn.
14 to 20 years.	30	4	5	2
21 to 30 years.	38	6	2	..
31 to 40 years.	6	..	..	..
41 to 42 years.	1	1	..	1

FŒTAL ASCITES.*

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ON account of their rarity and clinical importance, we venture to record the following two cases.

Case No. 1.

Post-mortem findings.—Baby A., male, weight 4 lbs. 14 ozs., length 16 inches, appeared normal and fairly well developed in external appearance. Abdomen:—protuberant, flanks bulged out, shifting dullness and fluid thrill were both present. Genital organs showed no abnormality. Anus patent, admitting the tip of the little finger. There was no evidence of subcutaneous œdema in any part of the body. On opening the abdominal cavity about 10 ozs. of slightly yellowish, clear fluid was drawn out; no flakes of lymph were found. The peritoneum as well as omentum were smooth and glistening everywhere. Coils of intestines were free. Liver, normal, capsule smooth. There was no evidence of portal obstruction which was particularly looked for. Spleen—normal. Kidneys and adrenals—normal, the former presenting a lobulated appearance. Bladder was not distended and contained only 6 c.c. of clear urine. Placenta weighed 2 lbs., looked big and œdematous, cotyledons friable and shaggy; cord 19 inches in length also œdematous, and friable, was attached marginally to the placenta. There was no fluid either in the pleural or pericardial cavities. Heart and lungs showed no abnormality.

Histological findings.—Liver, spleen, kidneys and suprarenals showed normal appearances, except for the presence of numerous leucocytes and hæmorrhages in some areas within the connective tissues; capillaries were engorged. This was specially marked in the liver. Peritoneum showed no sign of inflammation. Heart, lungs and thyroid showed a normal appearance. The skin and subcutaneous tissue did not show any œdematous changes. The placenta and cord showed evidence of œdema. Sections stained for *Treponema pallidum* (Levaditi's method) gave negative results. Sections stained for bacteria (MacCallum's method) showed none.

Chemical examination of the fluid.

Quantity	.. 10 ozs.
Colour	.. clear pale yellow.
Sp. gr.	.. 1015.
Albumen	.. copious.
Sugar	.. nil.
Bile pigments	.. present.
Bile salts, acetone and sediments.	Di-acetic acid—absent, a fair number of R.B.Cs. and a few pus and endothelial cells.
Rivalta's test positive.	

Cytological examination of the fluid.

	Per cent.
Polymorphonuclears ..	.. 50
Lymphocytes ..	.. 40
Endothelial cells ..	.. 10

Culture of the fluid—sterile.

Obstetric history of the mother.

Mrs. A., Hindu, Bengali, æt. 26, 3rd para: admitted with a history of 8 months amenorrhœa and swelling all over the body. Examination on admission: vertex presenting, cervix

*Being a paper read at the Medical and Veterinary Research Section of the Indian Science Congress, held at Nagpur in January 1931.

Plate I.

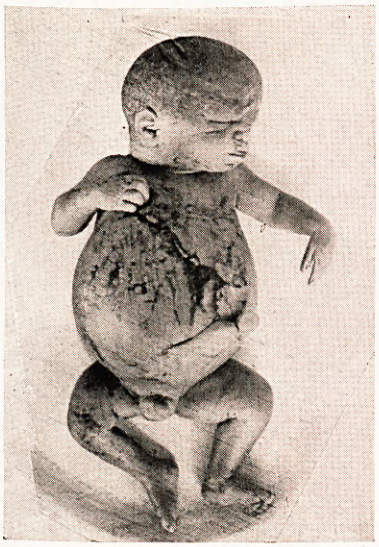

Fig. 1.

Case 1.—General appearance of the fœtus showing the well-marked ascites and œdematous condition of the cord.

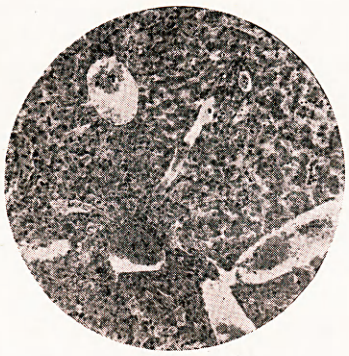

Fig. 2.

Case 1.—Photomicrograph of liver showing engorged capillaries and hæmorrhagic areas (low power).

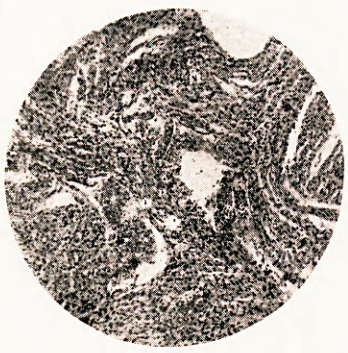

Fig. 3.

Case 2.—Photomicrograph of the heart showing (*a*) hæmorrhage between the muscle fibres, and (*b*) congested capillaries (low power).

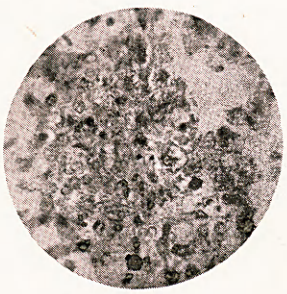

Fig. 4.

Case 2.—Photomicrograph of a hæmorrhagic area of the heart muscle (high power) showing megaloblasts, leucocytes, and red corpuscle infiltration.

fully dilated, head low down. Fœtal heart sounds not heard. Urine loaded with albumen. Blood pressure $\frac{175}{140}$ mm. Hg. Pulse 100, resp. 22 per minute, temperature 97°F. She was delivered of a dead fœtus within half an hour of admission without any artificial aid.

Previous history.—First baby born at full term but died of tetanus, a fortnight after birth. Second baby 1 year ago born at 8 months, died 2 hours after birth. Puerperia on both occasions were uneventful.

Blood.—Hb. 55 per cent., R. B. Cs. 3,600,000, W. B. Cs. 12,000, polymorph. 75 per cent., small mono. 22 per cent., large mono. 7 per cent., eosin. 1 per cent. No malaria parasites seen. Anisocytosis was present. Wassermann reaction negative. Chopra and aldehyde tests for kala-azar were negative.

Urine.—Sp. gr. 1018. Albumen present, 25 per cent. Sugar a trace only. Bile, acetone, di-acetic acid and phosphates—*nil.*

Microscopically.—A few pus cells.

N.B.—Evidently the mother suffered from albuminuria of pregnancy complicated with a moderate degree of anæmia.

Case No. 2.

Post-mortem findings.—Baby S., male, weight 6 lbs. 7½ oz., 17 inches in length corresponding in development to a 9 months' fœtus.

Extremities well developed. All the signs of free fluid in the peritoneal cavity were present. No evidence of subcutaneous œdema anywhere in the body. Genital organs quite normal. On opening the abdominal cavity about 300 c.c. of straw-coloured fluid escaped. No flakes of lymph present. The peritoneum, both visceral and parietal, was smooth and glistening. The intestines were free from adhesions, the large intestine contained meconium. The bladder contained only a few drops of clear urine. Placenta 14 oz., normal, did not look œdematous. Cord 17 inches long, was inserted marginally to the placenta.

Histological findings.—Liver, spleen and kidney showed normal appearance except for some areas of hæmorrhage within the internal organs. Megaloblasts were seen in the liver and spleen within the blood vessels. Heart—(*i*) there were areas of hæmorrhage, large and small, in between the muscle fibres, (*ii*) megaloblasts were seen in fair number, leucocytes were numerous, polymorphonuclears predominating.

Obstetrical history of the mother.

Mrs. S., Hindu, Bengali, æt. 24, 4th para: admitted in pains; labour lasted 12 hours 40 minutes, the second stage lasting only 15 minutes. Baby lived only for 1 hour after birth. Examination on admission: vertex presenting, head low down, cervix fully dilated, membranes were intact and were ruptured artificially followed by immediate delivery. Presentation was left occipito-anterior. Previous labours—normal.

Urine.—Clear.

Blood.—Hb. 50 per cent., R. B. Cs. 3,250,000, W. B. Cs. 25,000, polymorph. 88 per cent., small mono. 8 per cent., large mono. 2 per cent., eosin. 2 per cent. No malaria parasites seen. Chopra and aldehyde tests were both negative. Wassermann reaction negative.

N.B.—Moderate degree of anæmia in this case also is evident.

Review of Literature.

A single case of general fœtal dropsy is found in one of the two cases which represent the only clinical records of individual instances of the occurrence of fœtal disease and deformity in the Hippocratic writings. Albucasis (936-1013 A.D.) recorded a case of dystocia due to accumulation of fluid in the fœtal thorax and abdomen. Jacques Guillemeau (1550-1613) in his works described cases of fœtal ascites. Portal (1703) noted a case of fœtal ascites with distension of bladder. Philippepeu (1703) wrote on fœtal hydro-thorax and ascites. Guillaume Mauquest de la Motte (1655-1737) described cases of labour complicated by ascites and inflammatory peritoneal effusion. Mauriceau (? 1709) who was himself a subject of fœtal disease described cases of fœtal ascites, variola and syphilis. Levret (1703-1780) recorded a case of fœtal ascites. Galetti (1778) also described specimens of fœtal ascites and hydro-thorax. Later, notably among many others, Hohl of Halle, Virchow, Fordyce, and Ballantyne all wrote on fœtal ascites, but to Ballantyne credit is due for the most painstaking accounts of antenatal pathology and fœtal diseases.

A pure form of fœtal ascites has been seldom observed. It has been found most commonly as general fœtal dropsy associated with hydrothorax and sometimes with the dropsy of the mother. Large quantities of free fluid have occasionally been found in the macerated fœtuses.

As to the causation of the disease the following have been enumerated by various authors:—(1) Obstruction to the portal circulation by perihepatitis, syphilitic gumma, tumours pressing on the portal vein. (2) Chronic peritonitis (rarely acute) evidenced by firm vascularisation and adhesions between the different abdominal organs. Peritonitis is one of the manifestations of syphilis in the fœtus. (3) Tumours or a distended bladder pressing on the large vessels in the abdomen. (4) Hypoplasia of the urinary apparatus (Opitz). (5) Defects in the circulatory system, e.g., absence of the ductus venosus Arantii with heart failure. (6) Escape of fluid from a distended bladder into the general peritoneal cavity producing ascites (Olshausen). Ballantyne collected 17 cases of ascites with a distended bladder. Fordyce believed that ascites was the

result of bladder conditions. Sometimes "apparent" ascites may be due to a persistent cloaca (Cruickshank). In many cases no "apparent" cause for the ascites could be found.

External appearance in most of the cases was normal, except in a few cases where the anus was either absent or imperforate. Maceration was present in a fair number of cases, the peritoneum generally showed signs of inflammation. In the thoracic and abdominal organs no characteristic lesion was noted save some occasional malformations of the urinary apparatus such as dilation, atrophy or absence of the kidney and ureter on one side, distension of the bladder, or occlusion of urethra. Imperfect development of the genital organs was noted in a few cases.

Practically no characteristic change was found in any organ or tissue. The placenta was œdematous in a few cases.

Obstetrical significance.

Fœtal ascites has been observed to occur mainly in primiparæ and 2-paræ. Nearly all cases are premature, mostly between 7 to 8 months. Majority of the cases are vertex presentations, next to these come breech, transverse, face, brow presentations in order of sequence. Hydramnios has been noted in many of the cases. Uterine pains are usually weak from over-distension of the abdomen. Though difficulty is encountered in the delivery of the child from distension of its abdomen, which might necessitate artificial aid from simple traction to mutilating operations, yet fœtuses are often born spontaneously owing to prematurity and the compressibility of the abdomen when filled with fluid. Rupture of the uterus is extremely rare. Babies with ascites, no matter how they are delivered, are either born dead or die soon after birth. Congenital ascites has been observed to occur in the children of the same mother.

Summary.

(1) Dystocia from this cause is of rare occurrence, and when it does occur the true nature of the obstacle is apt to be overlooked. In both of our cases there was no dystocia. (2) Labour is generally premature, most cases occurring near about 7 months. In our cases both labours were premature. (3) None of the causes such as malformations of the urinary apparatus or a distended bladder, congenital syphilis, or portal obstruction were present in our cases. Both the mothers had a moderate degree of anæmia, while one of them had albuminuria in addition. Both were multiparæ. (4) Though there was no apparent evidence of peritoneal inflammation, the cytological examination of the fluid showed the presence of albumen and a high percentage of polymorphonuclear leucocytes which is suggestive of an inflammatory process. The cause of the inflammatory nature of the fluid is unknown and needs further investigation.

We express our indebtedness to Major P. Flemming Gow, F.R.C.S.E., D.P.H., I.M.S., Professor of Clinical Gynæcology and Obstetrics, for the specimens and clinical notes, and also to Major G. Shanks, M.D., I.M.S., Professor of Pathology, and to Dr. T. Sur, M.D., for giving us every facilities to publish this paper.

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HOW THE STRONGLY POSITIVE WASSERMANN CASES SHOULD BE REPORTED.

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I HAVE seen in my long experience many Wassermann cases reported as being 100 per cent. positive in the usual dilution of 1 in 10, continuing to be 100 per cent. positive in spite of treatment, this causing great despondency on the part of the patients, some of whom give up treatment as they think the disease is incurable, and doubts in the mind of the attending physicians as to the reliability of the test or the efficacy of the mode of treatment. This induced me to test the strongly positive sera in different dilutions, namely, 1 in 10, 1 in 20, 1 in 30, 1 in 40 and so on up to 1 in 240. I have been all along since 1912, when I first began to do this work, using a 1 in 10 dilution and reporting those as 100 per cent. positive that gave a 100 per cent. positive reaction with sera in a 1 in 10 dilution. But when I examined the strongly positive sera in different dilutions as stated above I saw that a certain serum which was reported 100 per cent. positive in 1 in 10 was really 100 per cent. positive also in 1 in 240. In order to explain how this examination was carried out, I must describe in brief my method of doing the Wassermann test.

Patient's serum.—The patient's blood is taken and is allowed to clot. The serum oozes out in half an hour and it is then centrifugalised or allowed to stand over-night in the ice chest. The serum is decanted and then heated to