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CLINICAL REPORT OF THE SCHOOL OF MIDWIFERY AND GYNAECOLOGY OF HONG KONG UNIVERSITY.

by

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By courtesy of the Director of Medical Services the work of this Clinic is carried out at the Tsan Yuk Hospital (T.Y.H.) and the Government Civil Hospital (G.C.H.). The former contains 45 obstetric and 15 gynaecological beds, whereas the latter has 16 obstetric and 8 gynaecological beds.

The classification of the Obstetric section of this report is the same as that adopted by Queen Charlotte's Maternity Hospital, London, and is the one suggested by the Committee for Unification of Obstetric Hospital Reports which was appointed by the Royal Society of Medicine. I wish to record my thanks and appreciation to the Medical Committee of Queen Charlotte's Maternity Hospital for allowing me to use their system of classification.

My predecessor, Professor R. E. Tottenham, vacated the Chair in May, 1935, and until my arrival in November of that year Dr. Pillai acted as Head of the Clinic. The Chair was founded by the Rockefeller Foundation in 1924 and Professor Tottenham was appointed to it in that year. I should like to take this opportunity of congratulating him on the present position of this Clinic and to state that the prestige with which it is held is due in large measure to his guidance, organisation and teaching. As a newcomer to the East my task has been considerably alleviated by the fact that the Clinic was founded by one who has not failed to impress upon his colleagues the principles of and the modern attitude towards Obstetrics and Gynaecology.

I wish to express my thanks to the Director and members of the Government Medical Department, Matrons, Sisters and Nurses for their co-operation.

W. C. W. NIXON.

OBSTETRIC DEPARTMENT.

During the year 1936 there were 2,367 admissions and 2,245 deliveries.

An attempt has been made to differentiate between cases which have had no ante-natal care and those that have.

A. *Booked Cases.* Patients having attended the ante-natal department on two or more occasions are included in this group.

B. *Emergencies.* Cases admitted without previous ante-natal care or sent in from outside Doctors or Midwives are classed under this heading.

A. Patients Booked in the Ante-natal Department.	*T.Y.H.*	*G.C.H*
1. Delivered in Hospital (*a*) Discharged	62	79
(*b*) Transferred	0	1
2. Baby born before arrival	0	0
3. Discharged undelivered	9	3
4. Died (*a*) after delivery	0	0
(*b*) undelivered	0	0
	71	83

Total of "Booked" Cases = 154

B. Patients admitted without previous ante-natal care or sent in as Emergencies by Outside Doctors or Midwives.

	T.Y.H.	*G.C.H.*
1. Delivered in Hospital (*a*) Discharged	1452	618
(*b*) Transferred	1	4
2. Baby born before arrival	6	5
3. Discharged undelivered	73	36
4. Died (*a*) after delivery	14	3
(*a*) undelivered	1	0
	1547	666

Total of "Emergency" Cases = 2,213

Total admissions to T.Y.H. = 1,618 and G.C.H. = 749

Total admissions to the Clinic = 2,367
Total deliveries = 2,245

Of the 154 Booked Cases, 63 were Primiparae 91 were Multiparae.

Of the 2,213 Emergency Cases, 751 were Primiparae, 1,462 were Multiparae.

NUMERICAL SUMMARY OF CASES DELIVERED IN HOSPITAL. ADMITTED FOR TREATMENT OR AFTER DELIVERY.

PRESENTATIONS.	T.Y.H.	G.C.H.	Total.
Persistent Occipito-posterior	41	23	64
Breech	52	21	73
Face	2	0	2
Shoulder	2	5	7
Brow	1	0	1
Twins	14	6	20
			Total
V. 1 (L.O.A.)			1524 (67.58%)
V. 2 (R.O.A.)			549 (24.35%)

COMPLICATIONS.			
Ante-partum Haemorrhage :—			
(*a*) Accidental	5	0	5
(*b*) Placenta Praevia	2	8	10
Post-partum Haemorrhage	33	18	51
Manual removal of placenta	4	0	4
Albuminuria with Toxic Symptoms	34	53	87
Eclampsia	5	1	6
Puerperal Pyrexia	108	41	149
Hydramnios	7	0	7
Caesarean Section	5	2	7
Prolapse of cord	4	2	6
Cardiac disease	1	2	3

ABBREVIATIONS.

A.P.H. = Ante-partum Haemorrhage.
A.P.V.D. = Ante-partum Vaginal Discharge.
A.R.M. = Artificial Rupture of Membranes.
F.H.N.H. = Foetal heart not heard.
P.F. = Promontory felt.
P.N.F. = Promontory not felt.
P.R.M. = Premature Rupture of Membranes.
G. = Good.
F. = Fair.
P. = Poor.
S.B. = Stillborn.
M. = Mother.
C. = Child.
A. = Alive.
D. = Died.
C.S. = Caesarean Section.
B.W. = Baby's weight.
P.M. = Post mortem.
H.S. = Haemolytic Streptococcus.
D.A.A. = Discharged Against Advice.
D.C. = Diagonal Conjugate.

CASES TREATED IN THE HOSPITAL BEFORE LABOUR.

Reg. No.	Age	Gravida	Maturity	Disease	Result	No. of days in Hospital before delivery or discharge	Remarks
T.Y.H.							
BOOKED							
1011	37	8	19	Tropical anaemia. Amoebic dysentery. Splenomegaly ...	Improved	6 weeks	
EMERGENCY							
577	36	3	30	Pneumonia.	G.	4 days	
926	19	1	36	Vitamin B deficiency. General oedema	G.	7 ,,	D.A.A.
959 (a)	26	3	8	Hyperemesis gravidarum. Retroverted gravid uterus ...	G.	13 ,,	Manual replacement.
1483	19	1	11	Hyperemesis gravidarum. Toxaemic	Good	24 ,,	
1557 (a)	42	10	27	Intestinal obstruction by omental band	Died	7 ,,	Aborted.
G.C.H.							
EMERGENCY							
217	25	1	34	Mitral stenosis	G.	25	Still-birth.
383	41	10	8	Hyperemesis Gravidarum	G.	9	Transferred.
440	23	1	26	Malaria & Broncho-pneumonia	G.	1	
464	27	2	40	Ankylostomiasis	G.	2	

CARDIAC DISEASE.

3 cases.

No Mothers died.

1 Baby was stillborn, a mortality of 33.3%.

Reg No.	Age	Gravida	Maturity	Lesion	Degree of failure of Compensation	Days in Hsp. before Delivery	Method of Delivery	Result M. C.
T.Y.H.								
EMERGENCY								
879	40	2	40	Mitral Regurgitation & Mitral Stenosis	Severe	1 day	Normal	G. G.
G.C.H.								
217	25	1	34	Mitral Stenosis	Marked...	25 days	Normal	G. G.
608	24	1	40	Mitral Stenosis	Moderate	2 days	Normal	G. S.B.

HYDRAMNIOS.

7 cases.

1 Mother died, a mortality of 14.8%.

2 Babies died, a mortality of 28.6%.

Reg. No.	Age	Gravida	Maturity	Girth of Abdo	Treatment	Result M.	Result C.	Remarks
T.Y.H.								
EMERGENCY								
169	32	1	37	40 inches.	Nil.	G.	D.	Syphilis.
534	26	8	40	40 inches.	Nil.	G.	G.	
519	22	2	36	40 inches.	Nil.	G.	D.	Foetal Ascites. Syphilis.
463	24	8	40	——	Nil.	G.	G.	
572	33	6	40	——	Artificial rupture of membs.	G.	G.	Generalised oedema.
693	28	8	36	40½inches.	Nil.	G.	G.	Twins.
1430	29	1	40	37½inches.	Nil.	D.	G.	P.P.H. Pre-eclampsia.

CASES OF ALBUMINURIA ADMITTED FOR TREATMENT.

87 cases.

No Mothers died.
8 Babies were stillborn and 3 died, a mortality of 12.6%.

Reg. No.	Age	Gravida	Maturity.	History of Renal Disease	Albuminuria on admission	Albuminuria on discharge	Oedema	Headache	Eye Signs	Highest Blood Pressure	No. of days in Hospital before labour or discharge	Type of labour	Result M. C.	Remarks
T.Y.H.														
BOOKED														
445	28	1	35	Nil.	†	Nil.	Legs	Nil.	Nil.	136/76	4 days.	Forceps ...	G. G.	Pre-clampsia. Uterine inertia.
515	29	3	40	Nil.	††	Nil.	Legs	Nil.	Nil.	156/108	7 days.	Normal ...	G. G.	Pre-clampsia.
EMERGENCY														
1519/35	31	3	40	Nil.	†	Nil.	Body & legs ..	Nil.	Nil.	150/96	2 days.	Normal ...	G. G.	
1524/35	29	4	40	Nil.	†	Nil.	Nil	Slight	Nil.	136/92	2 days.	Normal ...	G G	Accidental haemorrhage.
1532/35	25	3	40	Nil.	††	Nil.	Legs	Nil.	Nil.	110/90	2 days.	Normal ...	G. {G. / G.}	Twins.
1541/35	23	1	38	Nil.	†††	Nil.	Legs & Vulva	Nil.	Nil.	164/104	8 days.	Normal ...	G. G.	
1	22	2	37	Nil.	†	Nil.	Nil	Nil.	Nil.	150/100	1 day.	Normal ...	G. G.	
10	19	1	39	Nil.	†	Nil.	Legs	Nil.	Nil.	160/116	3 days.	Normal ...	G. G	
73	17	1	40	Nil.	††	Nil.	Nil	Nil.	Nil.	120/86	2 days.	Normal ...	G. S.B.	
1521/35	25	3	38	Nil.	Trace	Nil.	Legs	Nil.	Nil.	140/78	2 days.	Normal ...	G. G.	
126	24	1	40	Nil.	†	Nil.	Nil	Nil.	Nil.	114/81	3 days.	Normal ...	G. G.	
148	40	2	40	Nil.	†	Nil.	Legs	Nil.	Nil.	194/120	4 days.	Normal ...	G. G.	
165	24	1	39	Nil.	Trace	Nil.	Legs	Nil.	Nil.	168/130	3 days.	Normal ...	G. G.	
220	30	3	40	Nil.	†	Nil.	Legs	Nil.	Nil.	128/102	2 days.	Normal ...	G. G.	
245	27	1	40	Nil.	††	Nil.	Legs	Nil.	Nil.	204/120	5 days.	Forceps ...	G. G.	
276	31	3	40	Nil.	†	Nil.	Nil	Nil.	Nil.	120/80	3 days.	Normal ...	G G.	
280	33	2	38	Nil.	†	Nil.	Legs	Yes.	Nil.	130/88	4 days.	Breech ...	G. S.B.	Placenta Praevia.
290	26	1	40	Nil.	†††	Nil.	General	Nil.	Nil.	196/136	1 day.	Normal ...	G G.	
387	31	1	37	Nil.	†††	Nil.	Legs	Nil	Nil.	148/120	3 days.	Normal ...	G G.	Pre-clampsia.
423	30	1	40	Nil.	†	Nil.	Legs	Nil.	Nil.	148/114	2 days.	Normal ...	G. G.	
428	24	1	40	Nil.	†	Nil.	Legs	Nil.	Nil.	120/85	2 days	Normal ...	G G.	
430	20	1	37	Nil.	†	Nil.	Legs	Nil.	Nil.	104/85	2 days.	Normal ...	G. G	
426	22	1	40	Nil.	†	Nil.	Legs	Nil.	Nil.	130/72	2 days.	Normal ...	G G.	
467	33	1	40	Nil.	†	Nil.	Legs	Nil.	Nil.	148/102	2 days.	Normal ...	G. G.	
516	33	5	40	Nil.	†	Nil.	Legs	Nil.	Nil.	146/102	4 days.	Normal ...	G. S.B.	
538	29	1	30	Nil.	†	Nil.	Body	Nil.	Nil.	150/108	2 days.	Normal ...	G. S.B.	Accidental Haemorrhage.
849	21	1	39	Nil.	†	Nil.	Legs	Nil.	Nil.	164/110	In labour.	Normal ...	G. G.	
881	22	1	40	Nil.	††	Nil.	Legs abdom wall	Nil.	Nil.	150/96	In labour.	Normal ...	G. G.	Puerperal Sepsis. D.A.A.
909	22	1	40	Nil.	††	Nil.	Legs	Nil.	Nil.	110/70	In labour	Normal ...	G. G	
1250	38	4	40	Nil.	†	Nil.	Legs	Nil.	Nil.	176/118	1 day.	Obstructed ...	G. S.B.	
1429	23	1	40	Nil.	†††	—	General	Nil.	Nil.	176/134	In labour	Forceps P.O.P.	G. G.	P.P.H. Hydramnios.
1491	22	1	38	Nil.	†	Clear	Legs Abd. wall	Nil.	Nil.	142/90	7 days.	Normal ...	G. G.	Twins.
1520	17	1	40	Nil.	†	Trace	Nil	Nil.	Nil.	114/86	1 day.	Normal ...	G G.	D.A.A.
1592	24	1	40	Nil.	Trace	Clear	Legs			150/90	In labour	Normal ...	G G.	
G.C.H.														
BOOKED														
279	33	6	37	Nil.	†	Nil.	Legs	Nil.	Nil.	116/70	12 days.	Normal ...	G. G.	Vitamin B1 deficiency.
293	27	3	40	Nil.	†	Nil.	Nil	Nil.	Nil.	94/70	2 days.	Normal ...	G. G.	Pendulous abdomen.
309	26	1	38	Nil.	†	Nil.	Legs	Nil.	Nil.	136/92	2 days.	Normal ...	G. G.	Pre-eclamptic.
372	27	3	37	Nil.	†††	Nil.	Legs	Nil.	Nil.	174/108	9 days.	Normal ...	G. G.	
459	26	2	39	Nil.	††	Nil.	Legs-Vulva ...	Nil.	Nil.	154/100	4 days.	Normal ...	G. G.	
500	19	1	39	Nil.	†	Nil.	Legs	Nil.	Nil.	114/56	1 day.	Normal ...	G. G.	
512	28	1	34	Nil.	Trace	Nil.	Legs	Nil.	Nil.	146/80	3 days.	Normal ...	G. G.	
527	21	1	41	Nil.	Trace	Nil.	Legs	Nil.	Nil.	164/110	4 days.	Forceps ...	G. G.	Delayed 2nd stage. Toxaemia. Subinvolution

CASES OF ALBUMINURIA ADMITTED FOR TREATMENT. *(Continued).*

Reg. No.	Age	Gravida	Maturity	History of Renal Disease	Albuminuria on admission	Albuminuria on discharge	Oedema	Headache	Eye Signs	Highest Blood Pressure	No of days in Hospital before labour or discharge	Type of labour	Result M. C.	Remarks.
G.C.H.														
EMERGENCY														
538	19	1	38	Nil.	††	Nil.	Legs	Nil.	Nil.	134/76	2 days.	Uterine inertia	G. G.	P.O.P.
29	22	1	42	Nil.	††	Nil.	Legs	Nil.	Nil.	110/120	2 days.	Normal ...	G. G.	Perineal laceration.
57	41	4	41	Nil.	†	Nil.	Legs	Nil.	Nil.	172/140	3 days.	Normal ...	G. G.	Dobey's itch. External piles.
95	29	1	39	Nil.	†††	Nil.	Legs	Nil.	Nil.	160/120	1 days.	Normal ...	G. G.	Perineal laceration. Cervix badly lacerated.
172	25	1	35	Nil.	†††	Nil.	Legs	Nil.	Nil.	131/90	2 days.	Breech ...	G. G.	Diarrhoea and vomiting.
175(b)	26	2	40	Nil.	†	Nil.	Legs	Nil.	Nil.	130/80	2 days.	Normal ...	G. D.	
186	33	9	40	Nil.	†	Nil.	Legs	Nil.	Nil.	152/108	2 days.	Normal ...	G. G.	
194	33	7	40	Nil.	†	Nil.	Legs	Nil.	Nil.	106/72	2 days.	Normal ...	G. D.	
195	28	1	30	Nil.	†	Nil.	Nil	Nil.	Nil.	148/82	1 day.	Normal ...	G. S.B.	
204	30	5	39	Nil.	†	Nil.	Legs	Nil.	Nil.	126/72	2 days.	Normal ...	G. G.	
210	25	2	41	Nil.	†	Nil.	Legs	Nil.	Nil.	114/98	3 days.	Normal ...	G. G.	
214	28	1	40	Nil.	†	Nil.	Legs	Nil.	Nil.	150/90	2 days.	Normal ...	G. G.	
218	22	2	37	Nil.	†	Nil.	Legs	Nil.	Nil.	138/86	3 days.	Normal ...	G. D.	P.O.P.
240	36	6	38	Nil.	†	Nil.	Legs	Nil.	Nil.	152/90	4 days.	Normal ...	G. G.	
256	32	5	44	Nil.	†	Nil.	Nil	Nil.	Nil.	120/90	2 days.	Normal ...	G. G.	
265	36	8	41	Nil.	†	Nil.	Legs & Vulva ...	Nil.	Nil.	132/79	2 days.	Normal ...	G. G.	
265	35	5	42	Nil.	††	Nil.	General... ...	Nil.	Nil.	164/118	3 days.	Normal ...	G. G.	Twins. Bronchitis. Avitaminosis B Peripheral Polyneuritis.
269	32	3	36	Nil.	†	Nil.	Legs	Nil.	Nil.	154/86	In labour.	Forceps ...	G. G.	
281	35	6	42	Nil.	†	Nil.	Legs	Nil.	Nil.	150/100	6 days.	Normal ...	G. G.	
274	19	2	40	Nil.	†	Nil.	Nil	Nil.	Nil.	92/50	2 days.	Normal ...	G. G.	
284	30	1	41	Nil.	††	Nil.	Legs	Nil.	Nil.	150/104	3 days.	Normal ...	G. G.	
314	24	2	40	Nil.	†	Nil.	Nil	Nil.	Nil.	170/94	2 days.	Normal ...	G. G.	
360	26	2	38	Nil.	†	Nil.	Legs	Nil.	Nil.	138/90	2 days.	Occipito posterior	G. G.	
366	21	2	38	Nil.	††	Nil.	Legs	Nil.	Nil.	160/64	3 days.	Normal ...	G. S.B.	Anaemia. Syphilis.
378	28	6	38	Nil.	††	Nil.	Legs	Nil.	Nil.	140/100	2 days.	Normal ...	G. G.	
382	36	4	40	Nil.	††	Nil.	Whole body ...	Nil.	Nil.	126/80	4 days.	Normal ...	G. G.	
390	23	1	42	Nil.	†	Nil.	Legs	Nil.	Nil.	146/91	3 days.	Normal ...	G. G.	
444	30	6	40	Nil.	††	Nil.	Legs	Nil.	Nil.	140/108	2 days.	Normal ...	G. G.	
448	19	1	40	Nil.	††	Nil.	Legs	Nil.	Nil.	138/64	3 days.	Normal ...	G. G.	Anaemia.
460	21	1	39	Nil.	††	Nil.	Legs	Nil.	Nil.	112/84	3 days.	Normal ...	G. G.	
467	28	1	37	Nil.	Trace	Nil.	Legs	Nil.	Nil.	180/90	3 days.	Normal ...	G. G.	
503	25	1	38	Nil.	Trace	Nil.	Legs	Nil.	Nil.	158/96	1 day.	Normal ...	G. G.	
508	28	1	36	Nil.	Trace	Nil.	Legs	Nil.	Nil.	128/65	1 day.	Normal ...	G. G.	
469	31	6	40	Nil.	††	Nil.	Legs	Nil.	Nil.	154/76	1 day.	Normal ...	G. G.	
492	26	4	38	Nil.	†	Nil.	Legs & Vulva ...	Nil.	Nil.	110/50	2 days.	Normal ...	G. G.	
521	21	1	32	Nil.	†	Nil.	Legs	Nil.	Nil.	100/70	1 day.	Normal ...	G. G.	
529	26	2	36	Nil.	†	Nil.	Legs	Nil.	Nil.	108/50	4 days.	Normal ...	G. S.B.	
539	24	3	37	Nil.	††	Nil.	Legs	Nil.	Nil.	144/90	5 days.	Normal ...	G. G.	Ankylostomiasis. Secondary anaemia. Syphilis.
545	27	3	38	Nil.	†	Nil.	Arms	Nil.	Nil.	136/90	2 days.	Normal ...	G. G.	
551	20	1	38	Nil.	†	Nil.	Nil	Nil.	Nil.	106/68	3 days.	Normal ...	G. G.	General contracted pelvis.
555	28	1	40	Nil.	†	Nil.	Legs	Nil.	Nil.	104/76	2 days.	Normal ...	G. G.	
559	25	2	44	Nil.	†††	Nil.	Legs	Nil.	Nil.	156/76	2 days.	Normal ...	G. G.	Avitaminosis B1.
566 (a)	31	3	42	Nil.	†	Nil.	Legs	Nil.	Nil.	184/46	In labour.	Normal ...	G. G.	Avitaminosis B1.
567	22	1	40	Nil.	†	Nil.	Nil	Nil.	Nil.	142/86	In labour.	Normal ...	G. G.	
568	23	1	37	Nil.	†	Nil.	Legs	Nil.	Nil.	150/90	In labour.	Normal ...	G. G.	

ECLAMPSIA.

6 Cases.

2 Mothers died, a mortality of 33.3%

1 Baby was Still Born and 1 died, a mortality of 33.3%

Reg. No.	Age	Gravida	Maturity	Condition on admission (including if in labour)	No before admission	FITS Total	FITS Onset	URINE Albumen on admission	URINE Albumen on discharge	URINE Quantity in first 24 hrs.	Oedema	Highest Blood Pressure	Headache	Eye Signs	No. of days in Hospital before delivery	Type of labour Induced	Result M.	Result C.	Remarks
EMERGENCY																			
674	29	1	38	Good (in labour) ..	1	2	Intra partum	†††	Trace	—	Legs	144/102	†	Nil	In labour	Normal	G.	G.	D.A.A.
998	28	1	40	Poor (in labour) ...	3	5	Ante partum	†††	Nil	53 ozs.	General	170/120	†	†	In labour	Forceps	D.	G.	
1245	20	1	40	Good (not in labour)	0	4	Intra partum	†	Nil	34 ozs.	Legs	160/100	†	†	1 day	Normal	G.	S.B.	
1482	26	1	40	Good (in labour) ...	0	1	Intra partum	†	Nil	35 ozs.	General	180/120	†	†	In labour	Normal	G.	D.	Puerperal Pyrexia
1507	26	1	40	Good (in labour) ...	0	2	Intra partum	†	Nil	19 ozs.	Nil	140/100	—	—	2 days	Forceps	G.	G.	
1186	24	2	44	Good (not in labour)	0	30	4 days post partum	†	..	88 ozs.	General	194/114	†	†	6 days	Normal	D.	G.	Twins.

ACCIDENTAL ANTE PARTUM HAEMORRHAGE.

5 cases.

1 Mother died, a mortality of 20%.

2 Babies were stillborn and 1 died, a mortality of 60%.

Reg. No.	Age	Gravida	Maturity	Condition on Admission	Albumen	Treatment	Result M.	Result C.	Amount of Bleeding Concealed	Amount of Bleeding Revealed	Remarks
T.Y.H.											
EMERGENCY											
1524/35	29	4	40	Good.	†	Tight binder.	G.	G.	——	20 ozs.	Melaena Neonatorum.
536	29	1	30	Condition fair. Oedema of body.	†	Vagina plugged. Tight binder.	G.	S.B.	——	15 ozs.	
800	27	5	38	Poor.	†	General.	G.	G.	——	10 ozs.	
887	35	10	32	Good.	Nil.	A. R. M. and plugging. ...	G.	D.	——	10 ozs.	
1119	26	2	34	Good.	††	General.	D.	S.B.	?	20 ozs.	P.P.H.

PLACENTA PRAEVIA.

10 cases.

In 2 cases the Placenta was Central.

In 5 cases the Placenta was Marginal.

In 3 cases the Placenta was Lateral.

No Mothers died.

4 Babies were stillborn and 1 died, a mortality of 80%.

Reg. No.	Age	Gravida	Maturity	condition on Admission	Variety	Treatment	Result M.	Result C.	Amt. of Bleeding	Remarks
T.Y.H.										
EMERGENCY										
280	33	2	38	Fair	Central.	External version & leg brought down	G.	S.B.	10 ozs.	
750	42	5	31	Good	Central.	Willett's Forceps	G.	D.	5 ozs.	
1078	25	2	37	Poor	Marginal.	Internal version	G.	S.B.	20 ozs.	
1597	36	3	33	Good	Marginal.	Legs brought down	G.	S.B.	10 ozs.	Breech presentation
671	28	1	38	Good	Marginal	Forceps	G.	G.	10 ozs.	
675	29	4	40	Good	Marginal.	General	G.	D.	10 ozs.	
903	30	7	32	Fair	Lateral.	Caesarean Section	G.	S.B.	20 ozs.	
1021	23	2	38	Good	Lateral.	Willett's forceps	G.	D.	10 ozs.	
G.C.H.										
329	41	11	36	Good	Marginal	Legs brought down	G.	G.	20 ozs.	
465	27	4	30	Good	Lateral	Binder & A.R.M.	G.	D.	20 ozs.	

FACE AND BROW PRESENTATIONS.

There were 2 cases of Face, and 1 of Brow.

No Mother died.

2 Babies were stillborn, a mortality of 66.6%.

Reg. No.	Age	Gravida	Maturity	Position	Treatment	Result M.	Result C.	Weight of Child lb.	Weight of Child oz.
T.Y.H.									
EMERGENCY									
746	27	3	34	L.M.P.	Nil	G.	S.B.	6	8
1052	26	3	40	L. M. P.	Manual rotation and forceps.	G.	G.	6	12
1049	35	1	40	Brow	Perforation and extraction.	G.	S.B.	6	8½

PERSISTENT OCCIPITO-POSTERIOR POSITION.

In 64 cases the Occiput did not rotate spontaneously. The treatment and results of these cases are shown in the following table.

BOOKED. Mode of Termination.	No. of Cases.	Mother G.	Mother D.	Child G.	Child S.B.	Child D.
Manual Rotation and Forceps	1	—	1	1	—	—
Spontaneous Delivery-Face to Pubes	1	1	—	1	—	—
Forceps-Face to Pubes	3	3	—	3	—	—
Caesarean Section (Flat Pelvis)	1	1	—	1	—	—
EMERGENCY.						
Manual Rotation and Forceps	3	3	—	3	—	—
Spontaneous Delivery-Face to Pubes	45	45	—	38	5	2
Forceps-Face to Pubes	10	10	—	10	—	—

One mother died, (Pre-eclampsia). Maternal Mortality = 1.56%.
5 babies were stillborn and 2 died, a mortality of 10.93%.

SHOULDER PRESENTATIONS.

7 cases.

No Mothers died.

4 Babies were stillborn, a mortality of 57%.

Reg. No.	Age	Gravida	Maturity	Complication	Treatment	Result M.	Result C.	Weight of Child lb.	Weight of Child oz.	Remarks
T.Y.H.										
EMERGENCY										
260	33	6	40	2nd of Twins ...	Internal version...	G.	S.B.	4	6	
1218	26	3	32	Nil	... Internal version...	G.	G.	3	12	
G.C.H.										
EMERGENCY										
75	35	7	42	Nil	... External cephalic version ...	G.	G.	8	6	
281	30	7	31	Nil	... Internal version...	G.	G.	3	0	
356	36	8	36	Nil	... Internal version...	G.	S.B.	6	6	
511	27	4	37	Prolapsed Cord ...	Internal version...	G.	S.B.	3	10	Prolapsed arm
656	25	4	35	Nil	... Internal version...	G.	S.B.	6	8	

PROLAPSE OF CORD.

6 cases.

No Mothers died.

4 Babies were stillborn and 1 died, a mortality of 83.3%.

Reg. No.	Age	Gravida	Maturity	Size of os when diagnosed	Treatment	Result M.	Result C.	Complications	Remarks
T.Y.H.									
EMERGENCY									
260	33	6	40	Full dilatation	Internal version ...	G.	G.	Shoulder presentation	2nd of Twins.
460	42	7	35	3 fingers	Nil	G.	S.B.	Breech	
521	26	1	40	2 fingers	Nil	G.	S.B.	Nil	On admission no pulsation.
764	27	6	37	½ dilated	Nil	G.	D.	Nil	
G.C.H.									
482	32	7	40	Fully dilated	Nil	G.	S.B.	Syphilis	
511	27	4	37	Nearly fully dilated ...	External version ..	G.	S.B.	Transverse presentation.	

*COMPLICATED BREECH DELIVERIES (EXCLUDING BREECH BY VERSION).

Breech delivery with some other Obstetric complication present.

11 cases.

1 Mother died, a mortality of 9%.

6 Babies were stillborn, a mortality of 54.5%.

Reg. No.	Age	Gravida	Maturity	Complications	Treatment	Result M.	Result C.
T.Y.H.							
EMERGENCY							
93	41	8	33	Twins.	Spontaneous delivery.	G.	G.
98	41	8	33				
280	33	2	38	Placenta Praevia.	External version. Leg brought down.	G.	S.B.
389	34	1	40	Bicornuate uterus.	Leg and arms brought down.	G.	G.
896	29	3	34	2nd of Twins.	Spontaneous delivery.	G.	S.B.
975	30	5	39	2nd of Twins.	Spontaneous delivery.	D.	S.B.
1077	21	2	36	2nd of Twins.	Spontaneous delivery.	G.	G.
1078	25	2	37	M. Placenta Praevia. ...	Assisted delivery. Legs and arms brought down.	G.	S.B.
1491	22	1	38	Twins.	Spontaneous delivery. 2nd of twins.	G.	G.
1597	26	3	38	Placenta Praevia.	Legs brought down.	G.	S.B.
G.C.H.							
329	41	11	36	Placenta Praevia	Legs & arms brought down	G.	G.
717	21	1	38	2nd of twins	Legs brought down	G.	S.B.

UNCOMPLICATED BREECH DELIVERIES.

In 30 cases the Breech was delivered spontaneously.
No Mothers died.

8 Babies were stillborn and 3 died, a mortality of 36.6%.
In 30 cases the Breech was assisted.

No Mothers died.

4 Babies were stillborn and 2 died, a mortality of 20%.

Reg. No.	Age	Gravida	Maturity	Method of Delivery.	Result M.	Result C.	Remarks
T.Y.H.							
BOOKED							
1092	32	6	37	Extended head assisted.	G.	S.B.	
400	31	7	31	Spontaneous delivery.	G.	S.B.	Syphilis.
22	32	1	40	Leg brought down.	G.	G.	Left leg extended.
1023	22	1	37	Extended legs brought down.	G.	G.	
1121	27	4	32	Extended legs brought down.	G.	G.	
787	24	4	40	Assisted delivery. Extended legs and arms. ...	G.	G.	Ante-natal version failed.
1070	23	1	37	Extended legs and arms brought down.	G.	G.	
EMERGENCY							
31	25	1	40	Perforation. Arms brought down.	G.	S.B.	Impacted after coming head.
452	24	1	40	Assisted delivery.	G.	G.	Complete.
918	24	2	40	Assisted delivery.	G.	G.	
55	23	2	40	Spontaneous.	G.	G.	Complete.
96	22	2	37	Spontaneous.	G.	G.	Complete.
310	40	8	34	Spontaneous delivery.	G.	G.	Complete.
385	22	1	40	Spontaneous delivery.	G.	G.	Complete.
340	20	1	40	Spontaneous delivery.	G.	S.B.	Extended arms. Syphilis
460	42	7	35	Spontaneous delivery. Complete.	G.	S.B.	Prolapse of cord.
477	25	4	40	Spontaneous delivery.	G.	G.	
519	22	2	36	Spontaneous delivery.	G.	D.	Hydramnios. Syphilis.
535	24	3	40	Spontaneous delivery.	G.	G.	
588	19	1	30	Spontaneous delivery.	G.	S.B.	
681	21	2	40	Spontaneous delivery.	G.	G.	
764	27	6	37	Spontaneous delivery.	G.	G.	Prolapse of cord. D.A.A
841	44	15	40	Spontaneous delivery.	G.	G.	Complete.
970	20	1	37	Spontaneous.	G.	D.	
988	34	4	35	Spontaneous.	G.	G.	
1006	30	7	40	Spontaneous. Complete.	G.	G.	
1230	28	2	40	Complete. Spontaneous delivery.	G.	S.B.	
1297	19	2	29	Complete. Spontaneous delivery...	G.	G.	
1355	23	1	40	Complete. Spontaneous delivery...	G.	S.B.	
1466	28	7	40	Complete. Spontaneous delivery.	G.	G.	
1486	47	5	28	Complete. Spontaneous delivery.	G.	G.	
531	34	7	40	Leg brought down.	G.	G.	Transverse lie. Internal version.
542	35	8	40	Assisted delivery. Leg brought down.	G.	G.	
679	19	1	38	Assisted delivery.	G.	G.	Legs extended. Central Episiotomy.

UNCOMPLICATED BREECH DELIVERIES.—*(Continued).*

Reg. No.	Age	Gravida	Maturity	Method of Delivery	Result M.	Result C.	Remarks
T.Y.H.							
EMERGENCY							
1002	29	1	39	Extended legs brought down.	G.	G.	
1038	25	3	40	Extended legs brought down.	G.	G.	
1136	31	2	40	Extended legs brought down.	G.	G.	
131	17	1	40	Arms and legs brought down.	G.	D.	Extended arms and legs.
372	26	1	40	Assisted delivery. Extended legs and arms. ...	G.	G.	
374	28	1	37	Legs and arms brought down.	G.	G.	
1087	19	1	39	Extended legs and arms brought down.	G.	G.	
1192	33	2	40	Extended legs and arms assisted.	G.	G.	
G.C.H.							
BOOOKED							
220	27	6	35	Extended legs brought down	G.	G.	
718	26	1	38	Spontaneous delivery	G.	D.	Hydramnios. Syphilis.
EMERGENCY							
195	26	1	30	Extended arms brought down	G.	S.B.	
265	35	5	42	Assisted delivery	G.	G.	2nd Twins.
27	26	5	37	Spontaneous delivery	G.	G.	
22	18	1	38	Spontaneous delivery	G.	G.	Complete.
299	24	1	38	Spontaneous delivery	G.	G.	Extended arms.
308	22	1	30	Spontaneous delivery	G.	S.B.	Extended arms. Syphilis.
579	28	5	39	Spontaneous delivery	G.	G.	Complete.
581	28	1	38	Spontaneous delivery	G.	G.	General contracted pelvis.
631	25	1	32	Spontaneous delivery	G.	S.B.	Syphilis.
						G.	Twins.
181	22	1	36	Legs brought down	G.	G.	Twins.
482	26	1	38	Legs brought down	G.	G.	Extended legs.
521	31	1	32	Legs brought down	G.	G.	Extended legs.
645	19	1	40	Extended leg	G.	G.	
656	25	4	34	Bipolar version. Leg brought down	G.	S.B.	Transverse. Prolapsed hand.
723	25	4	40	Assisted delivery. Legs brought down	G.	G.	P.P.H.
592	30	6	29	Extended legs brought down	G.	D.	Hydramnios. Puerperal Pyrexia.

ANTE-NATAL VERSION.

This was attempted in 9 cases. In 5 of these it was successful, and of these 2 were Primipara and 3 were Multipara. In one case there was no anaesthetic given, and in 4 a general anaesthetic was given. No mother died, and there was one stillbirth as the result of perforation owing to rigidity of the cervix.

Of the 4 unsuccessful cases a general anaesthetic was given in all cases, 3 were primipara and 1 multipara.

The deliveries were spontaneous and there was no maternal or foetal mortality.

VERSION (IN LABOUR).

10 cases.

No Mothers died.

5 stillbirths, a mortality of 50%.

Reg. No.	Age	Gravida	Maturity	Indication	Bipolar, External or Podalic	Weight of Child lb.	oz.	Result M.	C.	Remarks
T.Y.H.										
EMERGENCY										
260	33	6	40	Shoulder presentation	Podalic ...	4	6	G.	G.	2nd of Twins.
280	38	2	38	Central Placenta Praevia	External ...	6	4	G.	S.B.	
399	34	1	40	Breech	External ...	7	0	G.	G.	Failed. Bicornuate uterus.
531	34	7	40	Shoulder, Hand presenting	Int. Podalic	7	8	G.	G.	Leg brought down.
1078	25	2	37	Marginal Placenta Praevia	Podalic ...	6	12	G.	S.B.	
1218	26	3	32	Shoulder presentation	Internal ...	3	12	G.	S.B.	
G.C.H.										
75	35	7	42	Shoulder presentation	External ...	8	6	G.	G.	
231	30	7	31	Shoulder c prolapsed arm	Internal ...	3	0	G.	G.	
511	27	4	37	Shoulder presentation	External ...	6	8	G.	S.B.	Prolapse of cord.
656	25	4	34	Shoulder presentation	Bipolar ...	3	10	G.	S.B.	Prolapsed hand.

POST PARTUM HAEMORRHAGE.

51 cases.
5 Mothers died, a mortality of 9.8%.
3 Babies were stillborn, a mortality of 5.8%.

Reg No.	Age	Gravida	Maturity	Relevant Features	Predisposing Cause	Treatment	Result M.	Result C.	Amt. of Bleeding	Remarks
T.Y.H.										
BOOKED										
991	19	1	37	Manual rotation & Forceps...	Atony	General	D.	S.B.	20 ozs.	
1225	37	8	40	Oedema	Uterine atony	General	G.	G.	15 ozs.	
EMERGENCY										
68	40	13	40	Elderly Multipara	Uterine inertia	Pituitrin & Ernutin ...	G.	G.	15 ozs.	
326	21	1	40	Nil	Nil	Bimanual Compression.	G.	G.	20 ozs.	
339	34	1	40	Bicornuate uterus	Retained placenta	Manual removal ...	G.	G.	20 ozs.	
388	20	1	40	Forceps	Uterine inertia	Pit. ergometrine... ...	G.	G.	20 ozs.	
505	36	9	28	———	Rupture of uterus	Plugging, etc.	D.	S.B.	20 ozs.	
685	17	1	40	Nil	Atony	Uterine plugging ...	G.	G.	20 ozs.	
730	22	1	40	Nil	Atony	General	G.	G.	20 ozs.	Puerperal Pyrexia.
748	26	2	40	Nil	Atony	General	G.	G.	20 ozs.	
762	25	1	40	Nil	Laceration of cervix and vagina	General	G.	G.	10 ozs.	Suture.
968	27	1	40	Oedema	Atony	General	G.	G.	20 ozs	
975	40	5	38	Twins	Atony	General		G.	20 ozs.	
1019	21	1	40	Marked oedema of leg ...	Atony	General	G.	G.	20 ozs.	D.A.A.
1078	25	2	37	Placenta Praevia	Retained placenta	General	G.	G.	20 ozs.	Manual Removal.
1113	26	2	34	Toxaemic A.P.H.	Atony	General	G.	S.B.	40 ozs.	
1150	28	2	40	Nil	Atony	General	G.	G.	30 ozs.	
1276	25	1	40	Nil	Laceration cervix	Suture	G.	G.	20 ozs.	D.A.A.
1308	36	6	40	Twins	Atony	General	G.	G.	30 ozs.	Twins.
1285	33	2	38	Normal	Cervical laceration	Suture	G.	G.	30 ozs.	
1350	32	9	40	Previous adherent placenta...	Retained placenta	Manual removal ...	D.	G.	40 ozs.	
1410	20	2	40	Nil	Uterine atony	General	D.	G.	20 ozs.	
1420	28	1	40	Forceps. Pre-eclampsia ...	Atony Cer. laceration... ...	Suture General	D.	G.	30 ozs.	
1433	29	7	40	Nil	Uterine atony	General	G.	G.	20 ozs.	
1450	21	2	40	Nil	Cervical laceration	Suture	G.	G.	15 ozs.	
1426	27	3	40	Nil	Uterine atony	General	G.	G.	20 ozs.	
1458	31	6	40	Nil	Uterine atony	General	G.	G.	20 ozs.	
1491	22	1	38	Twins	Atony	General	G.	G.	25 ozs.	
1517	36	1	40	Nil	Atony		G.	G.	30 ozs.	
1578	25	2	38	Nil	Cervical laceration	Suture	G.	G.	20 ozs.	Puerperal Pyrexia.
1522	18	1	40	Oedema	Cervical laceration	Plugging	G.	G.	20 ozs.	
1523	18	1	40	Oedema	Atony		G.	G.	20 ozs.	
1567	19	1	40	Nil	Cervical laceration	Plugging	G.	G.	25 ozs.	Puerperal Pyrexia.
G.C.H.										
14	18	1	40	B.B.A.	Nil	Uterus plugged ...	G.	G.	20 ozs.	
40	29	5	40	Nil	Nil	Pituitrin & Pitocin ...	G.	G.	20 ozs.	
72	32	8	38	Nil	Nil	Douche-pituitrin... ...	G.	G.	20 ozs.	Retained membranes.
84	19	1	40	Nil	Nil	Uterus & vagina plugged	G.	G.	20 ozs.	Traumatic.
115	28	1	40	Forceps	Uterine inertia	Bimanual compression	G.	G.	20 ozs.	Puerperal Pyrexia.
146	35	4	36	P.O.P.	Nil	Ergot	G.	G.	20 ozs.	Puerperal Pyrexia.
223	18	1	40	Nil	Cervical laceration	Suture	G.	G.	20 ozs.	
256	24	2	42	Nil	Cervical laceration	Suture	G.	G.	20 ozs.	
158	32	3	38	Avitaminosis B1	Atony	General	G.	G.	10 ozs.	
387	26	1	40	Syphilis	Atony	General	G.	G.	20 ozs.	Oedema.
424	27	6	43	Nil	Full bladder	Medical	G.	G.	40 ozs.	
427	21	2	40	Nil	Atony	Medical	G.	G.	20 ozs.	
486	38	5	40	Nil	Atony	General	G.	G.	20 ozs.	
556	33	8	38	Nil	Nil	Pit. & Ergometrin ...	G.	G.	20 ozs.	Gonorrhoea.
641	26	2	29	Nil	Nil	Pit. & Ergometrin ...	G.	G.	20 ozs.	
707	26	1	41	Delayed 3rd stage	Atony	Bi-manual compression	G.	G.	40 ozs.	
729	25	4	40	Nil	Atony	General	G.	G.	15 ozs.	
736	28	3	40	Nil	Atony	General	G.	G.	30 ozs.	

INDUCTION OF LABOUR (SPONTANEOUS DELIVERY).

4 cases.
No maternal or Foetal mortality.

Reg. No.	Age	Gravida	Maturity.	Indication	Int. Spin.	Int. Crist. (Inches)	Ext. Conj.	D.C.	Duration of labour 1st St.	Duration of labour 2nd St.	Weight lb.	Weight oz.	Child Length (Inches)	Circum. of Head	Result M. C.	Result I.D.I.	Method Drug	Method Instrumental	Remarks
G.C.H.																			
BOOKED																			
103	30	1	42†	Post-maturity	9¼	19¾	8	—	7¼ hrs.	3 hrs.	7	2	20	—	G. G.	—	Quinine	—	
EMERGENCY																			
39	21	1	44†	Post-maturity	9	10	8	—	6 hrs.	3 hrs.	7	12	20½	—	G. G.	—	Quinine	—	P.O.P.
42	28	5	44†	Post-maturity	8	9	7½	—	2 hours		6	7	20	—	G. G.	—	Quinine	—	
84	18	1	43	Post-maturity	8¾	10	8	—	24 hours		3	10	18	—	G. G.	—	Quinine	—	Traumatic P.P.H.

FORCEPS (LABOUR NOT INDUCED).

52 cases.

No Mothers died.

11 Babies were stillborn and 3 died, a mortality of 26.9%.

Reg No.	Age	Gravida	Maturity	Indication	Inst. Spin.	Inst. Crist. (Inches)	Ext. Cong.	D.C.	Duration of labour 1st St.	Duration of labour 2nd St.	Weight lb.	Weight oz.	Child Length	Circum. of Head	Result M. C	Remarks
T.Y.H.																
BOOKED																
445	28	1	35	Uterine inertia	9	10	7	—	14 hrs.	3 hrs.	6	—	19	—	G. G.	Post maturity.
991	19	1	37	P.O.P.	9½	10	7½	—	28 hrs.	40 min.	7	2	21	—	G. G.	
1098	28	1	40	P.O.P.	8	9½	7½	—	43 hrs.	3 hrs.	7	3	21	—	G. S.B.	
1015	32	1	38	Foetal distress	9	10	8	—	23 hrs.	15 min.	6	4	19	—	G. G.	
EMERGENCY																
532	25	2	40	Complex presentation...	9	10	6¾	—	14½ hrs.	2½ hrs.	7	—	20	—	G. S.B.	P.O.P.
671	28	1	40	Placenta Praevia ...	9	10	7	—	19 hrs.	3½ hrs.	3	12	18	—	G. G.	
694	39	2	40	Eclampsia	9½	60¼	7	—	38 hrs.	4 hrs.	6	2	19	—	G. G.	Uterine inertia. Asthma.
1082	26	3	40	Face presentation ...	9½	10¾	7¼	—	5 hrs.	1 hr.	6	12	18	—	G. G.	
1507	26	1	40	Eclampsia	9½	10½	7½	—	29 hrs.	3 hrs.	6	4	19	—	G. G.	
1586	32	1	40	Maternal distress ...	8	9½	6½	—	17½ hrs.	1½ hrs.	3	14	17	—	G. S.B.	
1513/35	33	3	40	Delayed 2nd stage ...	9	10	7	—	7 hrs.	2 hrs.	7	8	21½	—	G. S.B.	
1520/35	28	1	40	Delayed 2nd stage ...	9	10	7	—	9½ hrs.	4 hrs.	6	4	20½	—	G. G.	
1535/35	30	1	38	Delayed 2nd stage ...	9½	10½	7½	—	27 hrs.	3 hrs.	4	8	17½	—	G. G.	P.O.P.
1567/35	29	1	39	Delayed 2nd stage ...	9	10	7	—	24 hrs.	4 hrs.	6	4	20	—	G. G.	
71	21	1	40	Delayed 2nd stage ...	9	10	7	—	21½ hrs.	3½ hrs.	6	8	20	—	G. G.	P.O.P.
245	27	1	40	Delayed 2nd stage ...	9	10	7	—	8 hrs.	4 hrs.	6	—	20	—	G. G.	Forceps failed. Perforation.
203	27	1	40	Delayed 2nd stage ...	8	9	7	—	7 hrs.	4 hrs.	7	6	—	—	G. S.B.	Transverse lie of head.
311	28	1	40	Delayed 2nd stage ...	¼	10¼	7½	—	33 hrs.	4 hrs.	6	4	19	—	G. S.A.	Foetal distress.
348	20	1	40	Delayed 2nd stage ...	10	11	7½	—	5½ hrs.	3½ hrs.	6	4	19	—	G. G.	Spontaneous rotation. Foetal distress.
493	22	1	44	Delayed 2nd stage ...	9½	10½	7½	—	13 hrs.	4 hrs.	8	8	22	—	G. G.	Vertex c right hand prolapsed.
626	28	1	40	Delayed 2nd stage ...	9	10¼	7	—	9 hrs.	5 hrs.	6	12	19	—	G. G.	
669	32	1	40	Delayed 2nd stage ...	10	11	8	—	4½ hrs.	4½ hrs.	7	4	20	—	G. G.	
893	28	1	40	Delayed 2nd stage ...	9	10	7	—	19 hrs.	3 hrs.	8	1	20	—	G. G.	
1531	25	1	40	Delayed 2nd stage ...	9½	10¼	7½	—	43½ hrs.	2½ hrs.	7	4	22½	—	G. G.	
388	20	1	40	Uterine inertia	9½	10½	7¾	—	24 hrs.	4 hrs.	7	10	21	—	G. G.	Impacted shoulders. Post maturity.
405	41	9	44	Uterine inertia	10	11	8	—	12 hrs.	2 hrs.	10	12	24	—	G. S.B.	Pre-eclampsia.
721	25	1	38	Uterine inertia	9½	10½	7½	—	30½ hrs.	3 hrs.	7	8	21	—	G. G.	
879	23	1	40	Occipito posterior ...	10	11	7½	—	6 hrs.	3 hrs.	6	8	20	—	G. G.	
393	24	1	40	Occipito posterior ...	9¼	10	7	—	24 hrs.	40 min.	6	4	20	—	G. D.	
783	37	5	40	Occipito posterior ...	10	10¾	7½	—	24½ hrs.	2½ hrs.	7	12	20	—	G. G.	
856	37	6	40	P.O.P.	9	10	7	—	3 hrs.	2 hrs.	6½	—	20	—	G. G.	
880	26	1	40	P.O.P.	9	10	7	—	8 hrs.	2 hrs.	5	—	19	—	G. G.	Eclampsia.
1342	40	1	40	Transverse lie of head...	8½	10½	7½	—	71 hrs.		7	12	22	—	G. G.	
1343	20	1	40	Transverse lie of head...	8½	9½	6½	—	37 hrs.		7	—	19	—	G. S.B.	
1440	24	1	40	Transverse lie of head...	9	9½	7	—	30 hrs.	4 hrs.	7	14	22	—	G. G.	
1420	28	1	40	P.O.P.	8	9½	6	—	12 hrs.	½ hr.	6	10	21	—	G. G.	P.P.H. Pre-eclampsia.
349	36	1	40	Delayed 2nd stage ...	9½	10½	7½	—	32 hrs.	3½ hrs.	5	8	—	—	G. G.	P.P.H. Pyrexia.
541	33	1	40	Foetal distress	9	10	7	—	9 hrs.	3½ hrs.	5	—	19	—	G. G.	
717	28	1	40	Foetal distress	9¼	10¼	7¼	—	14 hrs.	4 hrs.	6	8	19	—	G. G.	
1242	27	1	40	Foetal distress	8	9½	6	—	5 hrs.	4 hrs.	6	12	18	—	G. G.	

FORCEPS (LABOUR NOT INDUCED).—*(Continued).*

Reg. No.	Age	Gravida	Maturity.	Indication	Inst. Spin.	Inst. Crist. (Inches)	Ext. Cong.	D.C.	Duration of labour		Weight lb.	oz.	Child Length	Circum. of Head	Result M. C.	Remarks
G.C.H.																
BOOKED																
687	21	1	41	Delayed 2nd stage ...	9½	10½	7½	—	13 hrs.	4 hrs.	8	4	21	—	G. G.	Toxaemia. Subinvolution.
EMERGENCY																
115	28	1	40	Uterine Inertia	8	9	7	—			6	14	19	—	G. G.	P.P.H. Puerperal Pyrexia.
118	25	1	30	Uterine Inertia	9	10½	7½	—	23 hrs.	4 hrs.	7	14	20	—	G. G.	
142	29	1	42	Delayed 2nd stage ...	9½	10½	8½	—	36 hrs.	2 hrs.	5	14	19	—	G. S.B.	P.O.P.
168	32	3	38	Delayed 2nd stage ...	—	—	—	—	7 hrs.	2 hrs.	6	4	19	—	G. G.	
277	30	1	37	Delayed 2nd stage ...	9½	10½	8	—	11 hrs.	2½ hrs.	6	2	19	—	G. G.	
345	34	2	40	Delayed 2nd stage ...	9½	10½	7½	—	9 hrs.		7	—	20	—	G G	
578	24	3	42	Delayed 2nd stage ...	9	10½	7½	—	14½ hrs.	2½ hrs.	8	12	20	—	G. G.	Anaemia. Subinvolution.
618	23	1	39	Delayed 2nd stage ...	9	10½	8	—	13 hrs.	6 hrs.	7	10	21	—	G. G	
177	30	3	43	P.O.P.	9½	10½	7½	—	12 hrs.	5½ hrs.	9	12	22	—	G. S.B.	
218	32	2	37	P.O.P.	8½	10	7¼	—	7½ hrs.	4½ hrs.	6	18	19	—	G. D.	
717	21	1	38	P.O.P. 1st of twins ...	9	10½	8½	—	2½ hrs.	2 hrs.	3 3	8 4	—	—	D. G. S.B.	Twins. Avitaminosis B.

CAESAREAN SECTION.

7 cases.

No Mothers died.

1 Baby died, a mortality of 14.2%.

Reg. No.	Age	Gravida	Maturity	Indication	Int. Spin.	Int. Crist.	Ext. Conj.	D.C.	Duration of labour 1st St.	2nd St.	Weight lb.	oz.	Child Length Inches	Circum. of Head	Result M. C.	Admitted for Trial Labour	Type of Operation	Remarks
T.Y.H.																		
BOOKED																		
308	37	1	39	Flat Pelvis.	8¾	9¾	7	4½	29 hrs.	—	7	14	20	—	G. G.	Yes	Lower segment.	Occipito posterior
1264	24	2	39	Bad obstetric history.	8¾	9¼	7	4½	7 hrs.	—	8	4	18¼	14¼	G. G.	Yes	Upper segment.	
EMERGENCY																		
750	42	5	31	Central Placenta Praevia.	—	—	—	—	2½ hrs.	—	3	8	17	—	G D.	No	Lower segment.	
1211	28	3	40	Flat Pelvis.	8½	9½	6½	3¾	37 hrs.	--	6	12	20	—	G. G.	No	Lower segment.	Pyrexia Syphilis.
1382	29	2	40	Disproportion.	9¼	10¼	7¼	4¼	16 hrs.	—	9	8	—	—	G. G.	No	Lower segment.	
G.C.H.																		
BOOKED																		
271	35	1	41	Elderly primip... ... Uterine inertia ...	9	10½	7¼	—	58 hours		7	12	—	—	G. G.	No	Lower segment.	
EMERGENCY																		
681	25	1	37	Generally Contracted Pelvis	7½	8½	6¾	4½	37 hours		5	10	—	—	G. G.	No	Lower segment.	

EMBRYOTOMY AND CRANIOTOMY.

7 cases.

No Mothers died.

Reg. No.	Age	Gravida	Maturity	Indication	Previous Treatment	Int. Spin. in.	Int. Christ. inches	Ext. Conj.	D.C.	Duration of labour 1st Stage	Duration of labour 2nd Stage	Weight of Child lb.	Weight of Child oz.	Result to Mother	Type of Operation	Remarks
T.Y.H.																
BOOKED																
871	24	1	40	Rigid cervix uterine inertia	Sedatives	9	10	7	—	50½ hrs.		7	6	G.	Perforation Extraction	Partial Slough cervix.
EMERGENCY																
81	25	1	40	Impacted after coming head	Nil	9¾	10½	7½		11 hrs.	3 hrs.	5	0	G.	Perforation	Extended arms.
205	27	1	40	Failed forceps & Foetal death... ..	Forceps ...	9	10	7	--	33 hrs.	8 hrs.	7	6½	G.	Extraction & Perforation	
568	26	1	40	Obstructed labour. Outlet contracted	Nil	9	9½	7½	--	16 hrs.	1 hr.	5	0	F.	Weight traction. Perforation	Ankylosis left hip.
1049	25	1	40	Impacted brow	Attempt at flexion	10½	11¼	7½	--	53 hrs.	2 hrs.	6	8½	G.	Extraction & Perforation	
1250	38	4	40	Obstructed labour	Sedatives	8	10	1	1	27 hrs.	3 hrs.	7½	0	G.	Extraction & Perforation	Flat pelvis Pre-eclampsia.
G.C.H.																
194	33	1	42	Uterine inertia, foetal death	Nil	--	--	--	—	59 hours		5	7½	G.	Perforation & extraction	H.S. septicaemia.

TRIAL LABOUR FOR SUSPECTED DISPROPORTION.

2 cases.

Reg. No.	Age	Gravida	Maturity	Onset of Labour Spontaneous	Onset of Labour Induced	Method of Delivery	Int. Spin.	Int. Crist.	Ext. Conj.	D.C.	1st St.	2nd St.	Weight lb.	Weight oz.	Length	Circum. of Head	M. C.	Remarks
T.Y.H.																		
BOOKED																		
203	27	1	39	Yes.	—	Lower Segment C.S.	9½	10	6½	4½	29 hrs.	—	7	14	20	--	G. G.	Flat pelvis. Occipito posterior.
1264	24	2	39	Yes.	—	Lower Segment C.S.	9½	10	6½	1½	7 hrs.	—	8	4	18½	14½	G. G.	

PRIMARY UTERINE INERTIA.

(Arbitrary definition being the first stage of labour lasting 48 hours or more).

13 cases—1 Mother died.

Reg. No.	Age	Gravida	Maturity	Position of Foetus	Time of Rupture of Membranes	Other Obstetric Abnormalities	Int. Spin.	Int. Crist. Inches	Ext. Conj.	D.C.	Duration of Labour 1st St.	Duration of Labour 2nd St.	Method of Delivery	Treatment Medical	Treatment Operative	Wright of Child lb.	Wright of Child oz.	Result
T.Y.H.																		
BOOKED																		
871	26	1	40	V1	30 hrs.	Partial Slough of Cervix; General oedema ...	9	10	7	P.N.F.	48 hrs.	2 hrs.	Perforaton & Extracton	Hyoscine	Yes	7	6†	G. S.B
1300	40	3	40	V2	44 hrs.	Nil	9	10½	7	P.N.F.	51 hrs.	2 hrs.	Normal	Sedatives	—	7	4	G. S.B.
EMERGENCY																		
1520	35	1	40	V1	37 hrs.	Nil	9	10	7	—	64 hrs.	4 hrs.	Forceps	Yes	—	6	4	G. G.
1049	30	1	40	Brow	5 hrs.	Nil	10½	11½	7½	—	53 hrs.	2 hrs.	Perforaton Extracton	Nil	Yes	6	8	G. S.B.
1152	19	1	40	V1	8 hrs.	Nil	8½	9	7	—	52 hrs.	3 hrs.	Normal	Sedatives Yes	Nil	6	14	G G.
1842	40	1	40	V1	69 hrs.	Transverse lie of head	9½	10½	7	—	71 hrs.		Forceps	Sedatives	—	7	12	G. G.
1531	25	1	40	V1	14 hrs.	Nil	9½	10½	7½	—	43½ hrs.	2½ hrs.	Forceps	Sedatives	Forceps	7	4	G. G.
1541	32	1	36	V1	3 hrs.	Nil	8½	9½	7½	—	69 hrs.	3 hrs.	Normal	Sedatives	Nil	6	12	G. G.
1586	32	1	40	V1	11 hrs.	Nil					47½ hrs.	1½ hrs.	Forceps	Sedatives	Forceps	8	14	G. S.B.
G.C.H.																		
BOOKED																		
271	35	1	41	V1	—	—	9	10½	7½	—	58 hours		C.S.	Sedatives	C.S.	7	12	G. G.
EMERGENCY																		
586	1	36	38	V4	18 hrs.	P.O.F.	10	11	8	—	48½ hours		Normal	Yes	—	7	2	G. G.
118	25	25	40	V1	36 hrs.	Nil	9	10½	7½	—	66 hrs.	2 hrs.	Forceps	Yes	—	7	14	G. G.
184	38	1	43	V1	24 hrs.	Nil	—	—	—	—	53 hours		Perforation	—	Yes	5	7†	D. S.B.

MANUAL REMOVAL OF PLACENTA.

4 cases.

1 Mother died, a mortality of 25%.

2 Babies were stillborn, a mortality of 50%.

Reg. No.	Age	Gravida	Maturity	Method of Delivery	Length of 3rd Stage	Indication	Morbidity	Result M.	Result C.	Amt. of Bleeding	Remarks
T.Y.H.											
EMERGENCY											
389	34	1	40	Breech.	50 min.	Haemorrhage.	Nil.	G.	G.	20 ozs.	
927	29	5	38	Normal.	3½ hrs.	Haemorrhage.	Nil.	G.	S.B.	20 ozs.	
1078	25	2	37	Breech.	20 min.	Haemorrhage.	Yes.	G.	S.B.	20 ozs.	Bicornuate uterus.
1350	32	9	40	Normal.	2½ hrs.	Haemorrhage.	Nil.	D.	G.	40 ozs.	

MATERNAL MORBIDITY.

161 Cases.

All cases with pyrexia and all maternal deaths are included as Morbid.

In the 142 Booked Cases there were 7 cases of Pyrexia. There was no maternal death.

The Morbidity rate for Booked Cases was 4.9%.

In the 2,103 Emergency Cases there were 142 cases of Pyrexia and 12 deaths without a rise in temperature, a total of 154 cases.

The Morbidity rate for Emergency Cases was 7.2%.

The Morbidity rate for the whole Clinic was 7.1%.

The definition of Puerperal Pyrexia as adopted at this Clinic is: "A temperature of 100.4 or over, occurring during the puerperium, while the patient is under observation, not including the first 24 hours."

The details of the Morbid cases are as follows:—

BOOKED CASES.

The causes of Pyrexia were as follows:—

Total number, 7. The causes of Pyrexia were as follows:—

Uterine sepsis, 1. (H. S.)
Urinary infection, 2.
Breasts engorgement, 2.
Wound sepsis (Caesarean Section), 1.
Entero-colitis, 1.

EMERGENCY CASES.

Total number, 154. Of these 12 died without Pyrexia and 6 died with a rise in temperature The causes of Pyrexia were as follows:—

Uterine sepsis, 58. (H. S. 17).
Urinary infection, 8
Perineal infection, 5.
Breast engorgement, 27.
Breast abscess, 1.
Septicaemia, 1.
Wound sepsis (Caesarean Section), 1.
Influenza, 6.
Pelvic cellulitis, 1.
Tonsillitis, 1.
Typhoid, 1.
Dysentery, 1.
Mumps, 1.
Malaria, 1.
Mania, 1.
Ascariasis, 1.
Constipation, 1.
Gastro-enteritis, 1.
Bronchitis, 1.
Avitaminosis B_1, 1.
Venereal disease, 8.
Cracked nipples, 1.
Suppurative nephritis, 1.
Unknown cause, 18.

Monthly distribution of the Pyrexia cases and of the cases infected by Haemolytic Streptococci.

	No.	H.S.		No.	H.S.
January	6	3	July	18	0
February	4	0	August	20	1
March	7	2	September	15	1
April	5	0	October	21	1
May	7	0	November	14	5
June	16	0	December	21	9

The Parity of the cases is as follows:—

Para	1	2	3	4	5	6	7	8	9	over 10
No. of cases.....										
BOOKED.....	3	1	2	1	0	0	0	0	0	0
EMERGENCY	74	31	18	7	7	7	1	0	0	9

MATERNAL DEATHS.

In booked cases, no maternal deaths.

In emergency cases, 18 deaths, a mortality of 8.1 per 1,000.

Mortality for all cases, 7.6 per 1,000.

T.Y.H.
Reg. No.

EMERGENCY

275 *Death from pulmonary embolus.*

Para 1, age 27, maturity 37 weeks. Normal labour, on the third day after delivery there was cough, slight fever, rusty sputum. The following day patient complained of acute pain in the chest asked for the bed-pan and died. P.M. Pulmonary embolus.

290 *Death from pre-eclampsia.*

Para 1, age 26, full term. Patient admitted with generalised oedema, marked albuminuria. B.P. 196 136. Pulmonary oedema also present. Normal delivery 24 hours after admission. Patient died six days after delivery.

533 *Death from rupture of uterus (pendulous abdomen).*

Para 5, age 30, full term. Patient admitted in labour with pendulous abdomen. Membranes ruptured two and a half hours after onset of labour. Three and a half hours later the foetal heart was not heard. Nine hours after labour began the patient complained of sudden abdominal pain parts could easily be felt. On catheterisation blood was withdrawn but no urine. The os was fully dilated and the vertex presenting. Patient died whilst being prepared for operation. P.M. The body of the foetus was outside the uterus but the head was deeply engaged in the pelvis. The foetal body was twisted so that the back was on the left but the occiput on the right, thus the face was on the side of the back. The rupture involved the lower segment of the uterus anteriorly and laterally and posteriorly it was intact.

505 *Death from rupture of the uterus.*

Para 9, age 36, 30 weeks. The duration of labour was two hours and a premature macerated foetus delivered. With rupture of the membranes, one hour after labour started there was some bleeding. About one hour after delivery there was haemorrhage. Although the uterus was contracted there was still oozing. On speculum examination a tear of the anterior lip of the cervix was seen and this extended into the lateral fornix. Despite treatment the patient died seven hours after delivery. P.M. Haematoma in both broad ligaments and in left side extending retro-peritoneally as far as the kidney. A tear, 2" in length, was seen on anterior aspect of cervix extending into lower uterine segment.

650 *Death from puerperal mania.*

Para 2, age 28, full term. Normal delivery. Patient suddenly became violent five days after delivery. Large doses of sedatives were necessary. She died two days later. On the fourth day temperature was 103, pulse 100. Bacteriological examination of the urine and vagina was negative. There was a history of marital disharmony and persecution by the husband's relatives. A clairvoyant predicted death of the patient or her baby on the sixth day. The patient died on the sixth day.

975 *Death from post-partum haemorrhage.*

Para 5, age 30, maturity 39 weeks. Normal twin delivery. The placenta of the first baby was delivered before the second baby. The second placenta was delivered after the second child. Following this there was severe bleeding due to atony of the uterus. Despite the usual restorative measures which included plugging uterus, the patient died twelve hours after delivery.

880 *Death from eclampsia.....*

Para 1, age 26, maturity 40 weeks. Patient admitted in a severe state of eclampsia. Artificial rupture of membranes. Coma developed. Forceps delivery 10 hours after rupture of membranes. The following day the blood-pressure was normal, urinary output within normal limits and the patient conscious. On the third day of the puerperium there was a sudden rise of blood pressure with suppression of urine. The patient died the same evening.

1119 *Death from haemorrhage (ante—and post-partum).*

Para 2, age 26, maturity 34 weeks. Patient admitted in labour and bleeding (concealed and external). There was marked albuminuria. The foetus was dead. After the delivery of the placenta there was severe atonic haemorrhage. There was a history of oedema of the legs for 6 weeks.

1178 *Death from polyneuronitis (Vitamin B1 deficiency).*

Para 6, age 34, maturity 30 weeks. Patient was admitted with heart failure and paralysis of all limbs, reflexes were absent. Speech was slurring. Delivery was normal, the day after admission. Several attacks of acute dyspnoea. W.R.†. Despite massive doses of vitamin B1, the patient died six days after delivery.

1171 *Death from rupture of uterus.*

Para 7, age 34, maturity 40 weeks. On admission the patient had been in labour for 8½ hours. The head was floating and the membranes intact. Five hours later as the abdomen was being palpated, the uterus was actually felt to "go"

T.Y.H.

and foetal parts were appreciated much more readily. On catheterisation blood-stained urine was withdrawn. Laparotomy was immediately performed. The foetus and placenta were in the abdominal cavity. There was a transverse rupture of the lower uterine segment and the bladder was also ruptured transversely. There was some difficulty in effecting haemostasis. Hysterectomy was performed. Patient died one hour after operation.

1180 *Death from eclampsia.*

Para 2, age 21, maturity 33 weeks. Patient was admitted in a severe state of pre-eclampsia. She responded to treatment and had a normal twin delivery 6 days later. Two days after delivery the blood-pressure suddenly rose. There was little or no oedema now but weakness of limbs and pulse irregularity. The following day, fits started and before death the patient had thirty-one fits.

1332 *Death from paralytic ileus following Caesarean section.*

Para 2, age 29, maturity 40 weeks. Patient admitted in obstructed labour. Previous history of renal disease and tuberculous peritonitis necessitating termination of last pregnancy at 20 weeks. Diagonal conjugate 3¾″. Lower segment Caesarean section. Patient died on 4th day from paralytic ileus.

1350 *Death from post-partum haemorrhage.*

Para 9, age 32, maturity 40 weeks. Normal delivery except manual delivery of placenta was necessary for post-partum haemorrhage. There was considerable difficulty in removing the placenta owing to its being adherent in parts. Patient died 3 hours after delivery. Previous history of manual removal of placenta and severe P.P.H. in last two labours.

1420 *Death from post-partum haemorrhage.*

Para 1, age 23, maturity 40 weeks. Admitted in labour with severe degree of pre-eclampsia. Manual rotation and forceps extraction for occipito-posterior presentation. Laceration of cervix which was sutured. Two hours after delivery there was severe atonic uterine bleeding and the patient died.

1557 (a) *Death from intestinal obstruction.*

Para 10, age 42, maturity 27 weeks. Patient was admitted with obvious signs of intestinal obstruction. An omental band was found obstructing the ascending colon, another was found crossing the sigmoid. Uterus expelled its contents on following day. Patient died 5 days later from heart failure.

G.C.H.

194 *Death from puerperal septicaemia.*

Para 1, age 33, maturity 43 weeks. Uterine inertia for 53 hours. Intra-partum foetal death. Perforation and extraction. Rigor day after delivery. Haemolytic streptococci in throat and vaginal mabs and blood culture. Abscesses developed in right forearm and right buttock. Death 16 days after delivery.

178 *Death from typhoid.*

Para 1, age 21, full term. Patient admitted in labour with temperature 102, pulse 130. Normal delivery. Blood culture—B. typhosus. Died on 17th day after delivery.

892 *Death from acute suppurative nephritis and vitamin B1 deficiency.*

Para 6, age 32. Normal delivery. The day following delivery there was weakness of the lower extremities and six days later there was complete paralysis of all limbs and loss of voice. W.R.!. Death on 8th day, the temperature rising to 105. Post mortem: multiple abscesses in kidneys, liver clonarchis present.

SUNDRY CASES NOT INCLUDED IN PREVIOUS TABLES.

101 cases.

Reg. No.	Age	Gravida	Maturity		Result M.	Result C.	Remarks
T.Y.H.							
BOOKED							
409	31	7	31	Syphilis	G.	S.B.	
1023	22	1	37	Avitaminosis B1. Two months post partum	G.	D.	Infantile beri beri.
EMERGENCY							
215	22	1	40	Leprosy	G.	—	Failed forceps & Perforation.
212	38	6	40	Precipitate labour. Fotal skull tracture	G.	G.	
203	27	1	40	Piles. Puerperal Sepsis. Post partum vaginal cicatricial Stenosis	G.	S.B.	
259	26	1	40	Mumps. (Post partum)	G.	G.	
338	36	13	40	Precipitate labour	G.	G.	
339	34	1	40	Bicornuate uterus	G.	G.	
245	24	1	37	Pulmonary embolus following labour	D.	G.	
401	29	3	38	Precipitate labour chronic mastitis (right)	G.	G.	
402	21	1	40	Chronic Endocarditis (mitral regurgitation)	G.	G.	
495	43	12	40	Varicose veins right legs	G.	G.	
513	33	7	30	Precipitate labour	G.	D.	
627	25	3	40	Influenza Sciatica (right)	G.	G.	
717	28	1	40	Asthma	G.	G.	
732	25	1	36	Malaria	G.	G.	
741	26	2	40	Breast abscess opened and drained	G.	G.	
748	30	3	40	Avitaminosis B1	G.	G.	
792	25	1	40	Ovarian cyst obstructing labour	G.	G.	
558	26	1	40	Ankylosis left hip. Vulvo-Ischio-rectal abscess	F.	S.B.	Perforation.
921	23	3	40	Pulmonary tuberculosis	G.	G.	
959	25	3	8	Retroverted gravid uterus and Hyperemesis gravidarum	G.	?	
1081	20	2	33	Acute gastro-enteritis	?	G.	D.A.A.
1086	23	1	40	Puerperal haemorrhage (Retained fragment of placenta)	G.	G.	15 days post partum
1171	37	7	40	Rupture of uterus	D.	S.B.	
1178	34	6	30	Polyneuronitis Vitamin B1 deficiency Syphilis	D.	S.B.	
1461	31	6	40	Dysentery	G.	G	
119	30	5	23	Incomplete abortion	G.		
1525/35	19	1	10	Abortion	G.		
219	25	3	34	Inevitable abortion	G.	-	
298	23	3	27	Complete abortion	G.		
869	30	5	26	Inevitable Abortion	G.	—	
1123	30	3	25	Abortion	G.	-	
1010	21	2	10	Missed abortion	G.	- -	
1143	22	2	26	Complete abortion. Syphilis	G.	- -	
169	32	1	37	Syphilis	G.	D.	
415	22	7	31	Syphilis	G.	S.B.	
419	33	9	29	Syphilis	G.	S.B.	
579	22	2	36	Syphilis	G.	D.	
564	17	1	40	Syphilis	G.	F.	
583	19	1	30	Syphilis	G.	S.B.	
754	24	1	30	Syphilis	G.	S.B.	
776	18	1	35	Syphilis	G.	G.	
1133	27	4	40	Syphilis	G. G.	G. S.B.	Twins.
1241	17	1	40	Syphilis	G.	G.	
1211	28	3	40	Syphilis. Baby syphilitic pemphigus	G.	G.	
141.	24	3	40	Syphilis	G.	G.	
1528	37	4	36	Syphilis	G.	G.	
1512	21	2	10	Syphilis	G.	—	Aborted.
536	30	5	40	Pendulous abdomen and rupture of uterus	D.	—	
693	28	3	36	Pendulous abdomen	G.	G.	Twins.
910	27	2	40	Hypertension. Pendulous abdomen	G.	G.	Puerperal Pyrexia.
1805	39	9	40	Pendulous abdomen. Divarication of recti	G.	G.	

SUNDRY CASES NOT INCLUDED IN PREVIOUS TABLES.—*(Continued).*

Reg. No.	Age	Gravida	Maturity		Result M.	Result C.	Remarks
T.Y.H.							
EMERGENCY							
1547	17	1	38	Intestinal obstruction due to omental band	G.	D.	
1557 (a)	42	10	27	Syphilis	D.	—	D.A.A.
1557 (b)	36	2	36	Syphilis	G.	G.	
1566	22	1	30	Varicose veins	G.	D.	D.A.A.
G.C.H.							
BOOKED							
220	27	6	35	Syphilis	?	G.	D.A.A.
279	38	8	37	Vitamin B.1. deficiency	G.	G.	
480	20	1	37	Puerperal insanity. Puerperal pyrexia	G.	G.	
518	27	1	44	Subinvolution—Breast engorgement	G.	G.	
687	21	1	41	Toxaemia of pregnancy. Subinvolution. Forceps	G.	G.	
686	24	2	37	Varicose veins of legs. Pulmonary tuberculosis	G.	G.	
704	30	4	39	Varicose veins on right leg for 3 months	G.	G.	
EMERGENCY							
66	30	8	39	Phthisis	G.	G.	
91	32	2	40	Ano-perineal fistulae for 6-7 yrs.	G.	G.	
134	33	1	43	Abscesses in right forearm and right buttock	D.	S.B.	
145	31	5	39	Varicose veins in right thigh External Pile	G.	G.	
172	25	1	35	Diarrhoea and vomiting	G.	G.	
177	30	3	43	Forceps. P.O.P. External Piles	G.	S.B.	
194	33	7	40	Cyst of vaginal wall	G.	D.	
178	21	1	40	Typhoid	D.	G.	
238	33	8	?	Carneous Mole	G	—	
211	27	2	39	Pelvic cellulitis Iliac abscess incised	G.	G.	
330	33	11	38	Varicose veins of both legs	G.	G.	
507	27	1	39	Subinvolution	G.	G.	
523	28	4	36	Bacillary Dysentery complicating Pregnancy and Puerperium	G.	G.	
559	23	2	38	Prolapse of anterior lip of cervix	G.	G.	
561	34	7	38	Precipitate labour	G.	G.	
606	25	4	38	Varicose veins of legs	G.	G.	
673	25	3	38	Melaena neonatorum. Subinvolution	G.	G.	
80	26	3	25	Abortion and Syphilis	G.	—	
116	19	2	32	Pregnancy and Syphilis	G.	G.	
353	20	1	40	Old tuberculous hip (Ankylosed)	G.	G.	
317	30	5	40	Influenza	G.	G.	
417	26	2	40	Tonsillitis...	G.	G.	
259	22	1	36	Syphilis	G.	G.	
308	22	1	30	Syphilis	G.	S.B.	
387	26	1	40	Syphilis P.P.H.	G.	G.	
620	40	11	40	Syphilis	G.	G.	
626	26	2	38	Syphilis	G.	G.	
631	25	1	32	Syphilis	G.	S.B.	
168	32	3	38	Avitaminosis B.1. Peripheral Polyneuritis	G.	G.	
458	20	1	36	Avitaminosis B1. Gonorrhoea. Subinvolution	G.	G.	
494	28	6	36	Avitaminosis B1.	G.	G.	
639	37	6	40	Vitamin B1. deficiency	G.	G.	
655	25	2	44	Avitaminosis B1	G.	G.	
685(a)	31	3	42	Vitamin B.1. deficiency	G.	G.	
321	34	3	38	Ascariasis, Anaemia, Ankylostomiasis	G.	G.	
560	24	3	37	Pre-eclampsia. Ankylostomiasis. 2nd Anaemia. Syphilis	G.	G.	
646	27	2	40	Ankylostomiasis	G.	S.B.	

INFANTS REPORT.

	T.Y.H.	G.C.H.	
Babies Born Total	1541	714 =	2255
Babies born alive	1456	673 =	2129
Stillborn	57	27 =	84
Died in Hospital	28	14 =	42
Abortions	8	2 =	10

Premature births/there were 138 premature babies born.

(Birth weight--5 lbs. or under)

Reg No.	Birth Weight lb.	oz.	Cause of Premature labour	Method of Feeding	Weight on discharge lb.	oz.	Result	Sex	Remarks
T.Y.H.									
BOOKED									
400	2	12	Unknown	— —	— — —		S.B.	M.	
608	3	8	Unknown	—	—		S.B.	F.	
1121	4	0	Unknown	Breast	3	10	G.	F.	Breech.
1583	3	8	Unknown	Breast					
EMERGENCY									
1518/35	4	10	Unknown	Breast	4	8	G.	F.	
1535/35	4	8	Unknown	Breast.	4	12	G.	F.	
1540/35	4	8	Unknown	Breast.	4	4	G.	F.	
44	2	12	Unknown	Nil.			D.	M.	
98	4	4	Unknown	Nil.	— —		D.	F.	
52	3	12	Unknown	Breast.	4	6	G.	F.	} Twins.
93	4	0	Unknown	Breast.	4	0	G.	M.	}
111	5	0	Unknown	Breast	5	0	G.	M.	
169	5	0	Hydramnios & Syphilis	—	— —		D.	M.	Lived 4 hours.
185	5	0	Unknown	Breast.	5	4	G.	F.	
260	4	6	Twins	Breast.	4	8	G.	M.	2nd of twins.
289	3	0	Unknown	—	— —		D.	M.	
337	3	0	Unknown	—	— —		S.B.	M.	Macerated.
374	4	14	Unknown	Breast.	5	4	G.	M.	
415	3	4	Syphilis...	—	— —		S.B.	M.	
419	2	2	Syphilis...	—	— —		S.B.	F.	
460	4	0	Syphilis...	—	— —		S.B.	M.	Prolapse of cord.
475	4	0	Unknown	Breast.	4	0	G.	F.	
488	5	0	Unknown	Breast	5	0	G.	M.	
491	4	8	Unknown	Breast	4	0	G.	M.	
506	5	0	Unknown	—	— —		D.	M	
513	3	0	Unknown	—	— —		D.	F.	Spina bifida.
579	5	0	Syphilis...	—	— —		D.	M.	Foetal Ascites.
588	3	0	Antepartum Accidental Haemorrhage...	—	— —		S.B.	F.	Macerated.
541	5	0	Unknown	Breast	— —		G.	F.	
564	3	10	Syphilis...	Breast	3	12	G.	F.	
566	4	0	Unknown	—	— —		S.B.	M.	
583	2	12	Syphilis...	—	— —		S.B.	F.	
597	3	0	Unknown	—	— —		D.	M.	
633	4	12	Unknown	Breast	4	8	F.	F.	Forceps.
657	5	0	Unknown	Breast	5	0	G.	M.	
671	5	12	Placenta Praevia	Breast	3	12	G.	M.	
675	5	0	Placenta Praevia	— —	— —		D.	M	
707	5	0	Unknown	Breast	5	4	F.	F.	

INFANTS REPORT.—*(Continued 1).*

Reg. No.	Birth Weight lb.	oz.	Cause of Premature labour	Method of Feeding	Weight on discharge lb.	oz.	Result	Sex	Remarks
T.Y.H.									
EMERGENCY									
754	3	0	Syphilis...	——	—	—	S.B.	M.	
764	2	12	Prolapse of cord	——	—	—	D.	M.	
750	3	8	Placenta Praevia		—	—	D.	F.	
887	5	0	A.P.H.	Nil.	—	—	D.	F.	
847	5	0	Unknown	Breast	5	0	G.	M	
776	4	0	Syphilis...	Breast	1	12	G.	M.	
971	4	8	Unknown	Nil.	—	—	S.B.	F.	
789	4	4	Heart disease	Breast	4	8	G.	F.	
895	5	0	Twins	Breast	5	0	G.	M.	
895	2	0		- -	—	—	S.B.	M.	
108	4	0	Placenta Praevia	——	—	—	S.B.	M.	
915	4	10	Vitamin B1 deficiency	Breast	1	12	G.	F.	
917	2	8	Unknown	Nil.	—	—	S.B.	M.	
945	4	4	Unknown	Breast	3	10	G.	M.	
952	3	8	Unknown	Breast	3	0	G.	F.	
880	5	0	Eclampsia	Breast	-	-	G.	F.	
983	3	4	Unknown	Nil.	—	—	D.	F.	
1000	3	15	Pyrexia	Breast	3	12	G.	M.	
1004	2	4	Unknown	Nil.	—	—	S.B.	F.	
1089	5	0	Unknown	Breast	1	8	G.	M.	
1101	3	2	Unknown	Breast	3	8	G.	M.	
1105	4	8	Unknown	Nil.	—	—	S.B.	M.	
1113	4	0	Unknown	Breast	3	4	G.	F.	D.A.A.
1021	4	4	Placenta Praevia	Breast	-	-	D.	M.	
1077	4	12	Twins	Breast	4	4	G.	F	2nd of twins
1083	5	0	Unknown	Breast	4	8	G.	M.	
1123	4	8	Twins	Breast	3	12	G.	M.	D.A.A.
1113	4	4		Breast	3	12	G.	M.	
1154	4	0	Unknown	Breast	3	12	G.	F.	D.A.A.
1157	4	14	Unknown	Breast	5	0	G.	F.	D.A.A.
1178	4	8	Syphilis...	Nil.	—	—	S.B.	F.	
1180	3	12	Pre-eclampsia Twins	Breast	-	-	G.	F.	
1180	4	12			-	-	G.	F.	
1188	4	12	Syphilis...	Nil.	—	—	S.B.	F.	2nd of twins.
1218	3	12	Unknown	Nil.	-	-	S.B.	F.	
1282	4	8	Unknown	Breast	4	6	G.	F.	D.A.A.
1292	4	14	Unknown	Breast	4	14	G.	F.	
1297	3	0	Unknown	——	—	—	S.B.	M.	
1308	4	0	Unknown	——	—	—	D.	M.	D.A.A.
1317	4	6	Unknown	Breast	4	2	G.	M.	
1352	4	4	Unknown	Breast	4	1	G.	F.	
1355	4	12	Breech	Breast	4	8	G.	F.	
1486	4	8	Nil	Nil.	—	—	S.B.	M.	
1491	4	8	Pre-eclampsia	Breast	4	4	G.	F.	1st of twins.
1498	4	4	Nil	Breast	3	6	G.	M.	
1525	4	0	Unknown	——	4	8	G.	M.	
1528	4	12	Syphilis...	Breast	—	—	D.	F	
1586	3	6	Syphilis...	Breast	3	12	G.	M.	
1586	3	14	Unknown	——	—	—	S.B.	M.	
1597	3	2	Placenta Praevia	——	—	—	S.B.	F.	

INFANTS REPORT.—*(Continued 2).*

Reg. No.	Birth Weight lb.	oz.	Cause of Premature labour	Method of Feeding	Weight on discharge lb.	oz.	Result	Sex	Remarks
G.C.H.									
BOOKED									
17	2	14	Unknown	Glucose water c C. Milk	—		D.	F.	
309	4	2	Pre-eclamptic	Breast	3	12	G.	F.	
551	5	0	Twins	Breast	5	4	G.	F.	1st of twins.
613	3	10	Twins	Breast	—		G.	F.	
613	2	10		Breast	2	9	G.	F.	
EMERGENCY									
	2	8					S.B.	M.	
547	1	12	Unknown	—	—		S.B.	M.	Twins.
38	3	4	Unknown	Breast c glucose water	3	2	G.	M.	B.B.A.
131	4	8		Breast	4	6	G.	M.	
195	4	0	Breast	—	—		S.B.	M.	
217	4	4	Breech	—	—		S.B.	M.	
224	4	0	Heart disease	—	—		S.B.	F.	
231	3	0	Unknown	Breast	3	0	G.	F.	
258	4	0	Unknown	Breast	3	14	G	M.	
265	4	4	Unknown	Breast	4	0	G.	M.	
265	4	5	... ...	Breast	4	4	G.	M.	Twins.
250	4	12	Syphilis...	Artificial	4	6	G.	M.	
276	4	2	Unknown	—	—		S.B.	F	
288	4	12	Unknown	Breast	4	12	G.	M.	
292	3	8	Unknown	Breast	3	8	G.	M.	
301	4	6	Unknown	—	—		S.B.	F.	Hydrocephalus.
307	5	0	Unknown	Breast	5	0	G.	F.	
308	3	12	Syphilis...	—	—		S.B.	F.	
321	3	4	Unknown	Breast	3	6	G.	M.	
328	4	14	Unknown	Breast	4	12	G.	F.	
332	4	0	Syphilis...	—	—		S.B.	F.	
339	4	14	Unknown	Breast	4	10	G.	M.	
351	4	14	Unknown	Breast	4	4	G.	F.	
352	3	8	Unknown	Breast	3	0	G.	M.	
366	4	6	Syphilis...	—	—		S.B.	M.	Jaundiced.
367	4	12	Syphilis...	—	—		D.	M.	
393	4	10	Unknown	Breast	4	12	G.	F.	
413	4	10	Unknown	Breast	5	0	G.	F.	
431	3	8	Unknown	—	—		D.	F.	
477	3	8	Unknown	—	—		D.	M.	
465	3	6	Placenta praevia	—	—		D.	M.	
521	4	4	Unknown	Breast	4	2	G.	M.	
528	4	8	Unknown	—	—		S.B.	M.	
535	4	14	Unknown	Breast	4	13	G.	M.	
550	3	14	Syphilis...	Breast	3	14	G.	M.	
565	5	0	Unknown	Breast	5	0	G.	F.	
567	4	12	Unknown	Breast	4	12	G.	M.	
592	3	6	Breech	—	—		D.	F.	
604	5	0	Unknown	Breast	5	8	G.	F.	
610	3	0	Unknown	Breast	3	2	G.	M.	
620	4	10	Syphilis...	Breast	5	0	G.	F.	
640	4	2	Syphilis...	—	—		S.B.	F.	Macerated.
641	4	1	Unknown	—	—		S.B.	M.	
717	3	8	Unknown	—	—		D.	M.	
717	3	4	Unknown	—	—		S.B.	M.	Twins.

TWINS (AND TRIPLETS IF ANY).

There were 20 cases of Twins and none of Triplets.

2 Mothers died, a mortality of 10%.

6 Babies were stillborn and 2 died, a mortality of 40%.

Reg. No.	Age.	Gravida	Maturity	Position 1st	Position 2nd	Sex 1st	Sex 2nd	Weight 1st lb.	Weight 1st oz.	Weight 2nd lb.	Weight 2nd oz.	Type	M.	Result 1st	Result 2nd	Remarks
T.Y.H.																
BOOKED																
1058	25	2	34	V3	V1	M.	M.	5	10	5	8	Binovular	G.	G.	G.	D.A.A.
EMERGENCY																
1582/35	25	3	40	V2	V2	M.	M.	5	8	5	4	Binovular	G.	G.	G.	
93	41	8	34	B1	B2	F.	M.	4	4	4		Binovular	G.	G.	G.	
260	38	6	40	P.O.P.	Shoulder	M.	M.	5	12	4	6	Uniovular	G.	G.	G.	Internal version for 2nd.
608	23	3	36	V1	V2	M.	M.	5	4	5		Binovular	G.	G	G.	Hydramnios Pendulous abdomen.
895	20	3	34	V1	B1	M.	M.	5	0	2	0	Binovular	G.	G.	S.B.	
975	30	5	39	V1	B1	F.	M.	5	4	—		Binovular	D.	G.	S.B.	P.P.H.
982	25	1	39	V2	V1	F.	F.	5	2	5	6	Binovular	G.	G.	G.	D.A.A.
1077	21	2	36	V1	B2	F.	F.	5	4	4	12	Binovular	G.	G.	G.	
1123	26	1	31	V1	V1	M.	M.	4	8	4	4	Binovular	G.	G.	G.	
1180	24	2	33	V1	V2	F.	F.	3	12	4	12	Binovular	D.	G.	G.	
1183	27	4	40	V1	V1	F.	F.	5	8	4	12	Binovular	G.	G.	S.B.	
1303	36	6	40	V1	V3	F.	M.	5	4	6	4	Binovular	G.	G.	G.	P.P.H.
1491	22	1	38	V1	B1	F.	F.	4	8	6	8	Binovular	G.	G.	G.	P.P.H.
G.C.H.																
BOOKED																
551	37	8	39	V2	V1	F.	F.	5	0	5	8	Binovular	G.	G.	G.	Delayed 2nd stage.
618	21	1	36	V2	V2	F.	F.	5	10	2	10	Uniovular	G.	D.	G.	
EMERGENCY																
547	32	2	31	V1	B1	M.	M.	8	3	1	12	Binovular	G.	S.B.	S.B.	
131	22	1	36	B1	B1	M.	M.	5	8	4	8	Binovular	G.	G.	G.	
285	35	5	42	V1	B2	M.	M.	4	4	4	5	Binovular	G.	G.	G.	
717	21	1	38	F.O.P.	B1	M.	M.	3	8	3	4	Binovular	G.	D.	S.B.	Avitaminosis B1.

STILL BIRTHS.

84 cases.

The still birth rate = 3.4%.

Reg. No.	Sex.	Method of delivery	Maternal Complication	Cause of Death (P.M. if done)	Remarks
T.Y.H.					
BOOKED					
371	M.	Perforation Extraction ...	Rigid cervix	Macerated. Prematurity ...	
400	M.	Breech	Syphilis	Cerebral haemorrhage	
603	F.	Normal	Nil	Prematurity	
1092	M.	Breech	Nil	Cerebral haemorrhage	
1098	M.	Forceps	Nil	Cerebral haemorrhage	P.O.P.
1300	M.	Normal	Uterine inertia	Asphyxia	Short cord round neck 3 times.
1559	M.	Normal		Asphyxia	Difficulty with shoulders.
EMERGENCY					
1513/35	M.	Forceps	Delayed 2nd stage...	Asphyxia	Foetal distress.
31	F.	Breech (Perforation) ...	Obstructed delivery	Perforation	Impacted after coming head
78	F.	Normal	Puerperal Sepsis	Macerated foetus	
208	M.	Perforation	Puerperal Sepsis	Prematurity	Failed forceps.
308	M.	P.O.P.	Nil	Cerebral haemorrhage	Syphilis.
311	F.	Forceps	Uterine inertia	Asphyxia	Transverse lie of head.
280	F.	Breech	Central Placenta Praevia	Macerated	Oedema of legs.
340	M.	Breech	Nil	Asphyxia	Syphilis.
360	M.	Normal	Nil	Prematurity	Impacted shoulders.
397	M.	Normal	Nil	Perforation	Macerated. W.R.--
406	M.	Forceps	Post-maturity	Macerated. Prematurity ...	Uterine inertia.
415	M.	Normal	Syphilis	Prematurity	
419	F.	Normal	Syphilis	Prematurity	Prolapse of cord.
460	M.	Breech	Nil	Intracranial haemorrhage ...	
500	M.	Normal	Nil	Shock	
516	F.	Normal	Nil	Generalised oedema. Ascites ...	
521	M.	Normal	Nil	Prolapse of Cord	
582	M.	Forceps	Nil	Cerebral haemorrhage	
593	F.	Normal	Antepartum Accidental Haemorrhage ...	Prematurity	Macerated foetus.
595	M.	Normal	Rupture of uterus	Asphyxia & prematurity ...	
583	F.	Breech	Syphilis	Prematurity	
746	M.	Face presentation	Nil	Prematurity	
754	M.	Normal	Syphilis	Prematurity	
812	M.	Normal	Macerated	Prematurity	
558	M.	Perforation	Obstructed labour	Perforation	
849	M.	Normal	Nil	Macerated	
871	F.	Normal	Nil	Prematurity	
895	M.	Normal	Nil	Prematurity	2nd of twins.
906	M.	Normal	Placenta Praevia	Prematurity Asphyxia	
925	F.	P.O.P.	Nil	Prematurity	
927	M.	Normal	Nil	Prematurity	
975	M.	Breech	P.F.H.	Asphyxia	2nd of twins.
1004	F.	Normal	Nil	Prematurity	
1105	M.	Normal	Nil	Prematurity	
1049	F.	Perforation	Brow	Perforation	
1078	M.	Breech	Placenta Praevia	Asphyxia. Prematurity ...	

STILL BIRTHS.—*(Continued)*.

Reg. No.	Sex.	Method of delivery	Maternal Complication	Cause of Death (P.M. if done)	Remarks
T.Y.H.					
EMERGENCY					
1119	F.	Normal	A.P.H. & P.P.H.	Prematurity & Asphyxia	
1146	M.	Normal	Nil	Unknown	D.A.A.
1171	F.	Laparotomy	Rupture of uterus	Asphyxia	
1173	F.	Normal	Nil	Unknown	
1178	F.	Normal	Syphilis Vitamin B1 deficiency	Prematurity	
1183	F.	Normal	Syphilis	Prematurity	2nd of twins.
1218	F.	Breech	Shoulder presentation	Prematurity	
1250	M.	Perforation	Obstructed labour	Perforation	
1297	M.	Breech	Nil	Prematurity	
1342	F.	Forceps	Uterine inertia & transverse lie of head	Intracranial haemorrhage	
1343	M.	Forceps	Eclampsia	Intracranial haemorrhage	
1486	M.	Breech	Nil	Prematurity	
1525	M.	Normal	Nil	Prematurity	
1586	M.	Forceps	Uterine inertia	Prematurity	
1607	F.	Breech	Placenta Praevia	Prematurity	
G.C.H.					
BOOKED					
190	M.	Normal	Nil	Prematurity	Macerated foetus.
EMERGENCY					
547	M.	Normal	Nil	Prematurity	Twins.
547	M.				
7	M.	Normal	Nil	Prematurity	
142	M.	Forceps P.O.P.	Uterine inertia	Asphyxia	
134	F.	Perforation	Uterine inertia	Asphyxia	Intra-partum death.
177	F.	Forceps P.O.P.	Uterine inertia	Cerebral haemorrhage	
195	M.	Breech	Nil	Prematurity	
217	M.	Normal	Heart disease	Prematurity	
224	F.	Normal	Syphilis	Macerated. Prematurity	
276	F.	Normal	Nil	Prematurity	
301	F.	Normal	Nil	Prematurity	Hydrocephalus.
308	F.	Normal	Nil	Prematurity. Syphilis	
325	F.	Breech	Syphilis	Macerated	
332	F.	Normal	Syphilis	Macerated	
356	M.	Normal	Nil	Intracranial haemorrhage	
366	M.	Breech	Syphilis	Prematurity	
422	M.	Normal	Syphilis	Prolapse of cord. Asphyxia	
511	M.	Transverse to Breech	External Version	Macerated foetus. Asphyxia	
528	M.	Normal	Nil	Asphyxia	
585	M.	Normal	Nil	Macerated	
631	F.	Breech	Syphilis	Prematurity	Macerated.
586	M.	Breech	Nil	Prematurity	Transverse. Prolapsed hand.
696	M.	Normal	Nil	Unknown	D.A.A.
730	M.	Normal	Nil	Prematurity	Macerated.
717	M.	Breech	Avitaminosis B1	Prematurity	2nd of twins.
646	F.	Normal	Ankylostomiasis	Asphyxia	

INFANT DEATHS.

42 cases.

Reg. No.	Sex	Birth Weight lb.	oz.	Method of delivery	Maternal Complication	Cause of Death (P.M. if done)	Age	Method of Feeding	Remarks
T.Y.H.									
EMERGENCY									
1533/55	F.	6	0	Normal	Nil	Unknown	5 days	Breast	Syphilis.
44	M.	2	12	Normal	Nil	Prematurity	1½ hrs.	—	
52	F.	3	12	Normal	Nil	Prematurity	1½ hrs.	—	
89	M.	6	14	Normal	Nil	Unknown	2½ days	Breast	
100	M.	6	10	Breech	Nil	Asphyxia	1 hrs.	—	
169	M.	5	0	Normal	Hydramnios & Syphilis	Prematurity	4 hrs.	—	
289	M.	3	0	Normal	Nil	Prematurity	3 hrs	—	
367	M.	5	12	Normal	Nil	Unknown	46½ hrs.	Breast	
398	M.	6	4	Forceps	Nil	Cerebral haemorrhage	35 min.	—	Uterine inertia.
506	M.	5	0	Normal	Nil	Prematurity	7 hrs.	—	
518	F.	3	0	Precipitate labour	Nil	Prematurity	7 hrs.	—	
519	M.	5	0	Breech	Syphilis	Ascites Prematurity	14 hrs.	—	Hydramnios.
597	M.	3	0	Normal	Nil	Prematurity	1 hr.	—	
675	M.	5	0	Normal	Placenta Praevia	Prematurity	3 hrs.	—	
764	M.	2	12	Breech	Prolapse of cord	Prematurity	3 hrs.	—	
750	F.	3	8	Caesarean Section	Placenta Praevia	Prematurity	16 hrs.	—	
857	F.	5	0	Normal	A.P.H.	Prematurity	6 hrs.	Nil	
860	F.	6	8	Normal	Nil	Atelectasis	12 hrs.	Nil	
937	F.	6	13	Normal	Nil	Atelectasis	3 days	Breast	
961	M.	6	8	Normal	Oedema	Atelectasis	8 hrs.	Nil	
988	F.	3	4	Breech	Nil	Prematurity	12 hrs.	Nil	
1021	M.	4	4	Willett's forceps	Placenta Praevia	Septic Scalp	8 days	Breast	
940	F.	6	0	Normal	Vitamin B1 deficiency	Cerebral haemorrhage	1 hr.	Nil	
1303	M.	4	0	Normal	Nil	Prematurity	8 hrs.	Nil	
1452	M.	6	6	Normal	Eclampsia	Unknown	4 days	Artificial	
1476	F.	6	0	Normal	Nil	Intracranial haemorrhage	37 hrs.	—	
1547	F.	6	6	Normal	Nil	Icterus gravis	7 days	Breast	Syphilis
1566	F.	3	6	Normal	Syphilis	Prematurity	4 days	Breast	
G.C.H.									
BOOKED									
17	F.	2	14	Normal	Nil	Prematurity	1 day	Glucose	
220	M.	5	6	Breech	Nil	Cerebral haemorrhage	4 hrs.	—	
618	F.	3	10	Twins	Nil	Prematurity	8 hrs.	Breast	1st of twins.
EMERGENCY									
20	F.	6	0	Normal	Oedema of legs	Cerebral haemorrhage	1 day	Breast	
175 (b)	M.	6	12	Normal	Nil	Pneumonia	3 days	—	
194	M.	8	0	Normal	Cyst of vaginal wall	Cerebral haemorrhage	27 hrs.	—	
218	F.	6	10	Forceps	Nil	Asphyxia	2½ hrs.	—	
367	M.	4	12	Normal	Syphilis	Asphyxia	2 hrs.	—	
421	F.	3	8	Normal	Nil	Prematurity	3 hrs.	—	
477	M.	3	8	Normal	Nil	Prematurity	1 day	—	
485	M.	3	6	Normal	Placenta praevia	Prematurity	4 hrs.	—	
502	F.	3	6	Breech	Hydramnios	Prematurity. Asphyxia	3½ hrs.	—	
599	M.	6	2	Normal	Nil	Asphyxia	30 min.	—	
717	M.	3	8	Forceps P.O.P.	Avitaminosis B1	Prematurity	6 hrs.	—	1st of twins

FOETAL ABNORMALITIES.

14 cases.

Reg. No.	
T.Y.H.	
EMERGENCY	
166	Bilateral Harelip ... ...
841	Harelip & cleft palate ... ...
513	Spina bifida ... ...
599	Foetal Ascites ... ...
505	Hare-lip and cleft palate ... ...
648	Harelip ... ...
675	Harelip & cleft palate ... ...
856	Fracture of frontal bone (Forceps (P.O.P.) ...
961	Cleft palate & Talipes equino varus (rt.) ...
G.C.H.	
195	Hydrocephalus ... ...
210	Cleft palate and hare lip ... ...
301	Hydrocephalus ... ...
656	Monster ... ...
666	Syndactylism ... ...

OPHTHALMIA.

2 cases.

Reg. No.	Number of days Treated	Remarks
T.Y.H.		
559	7 days...	Nil
776	10 days...	Gonococcal.

GYNAECOLOGICAL DEPARTMENT.

Table No. I.

	T.Y.H.	*G.C.H.*
Number of admissions	232	140
Number of operations	143	92

Table No. II.

Nature and Number of Operations.

Vulva :—	*T.Y.H.*	*G.C.H.*
Laceration, repair of	1	—
Lipoma of labium major, removal of	1	—
Bartholin abscess, incision of	1	—
Perineum :—		
Perineorrhaphy	2	1
Urethra :—		
Urethral Stenosis, dilatation of	—	1
Urethro-vaginal fistula, repair of	—	1
Vagina :—		
Cicatricial Stenosis, dilatation & excision	1	—
Vesico-vaginal fistula, repair of	—	5
Removal of vaginal septum (Double uterus and vagina)	—	1
Colpo-perineorrhaphy for prolapse	5	9
Uterus :—		
Curettage	23	14
(Of these for incomplete abortion there were	14	7
Hysterectomy (Subtotal)	1	4
Hysterectomy (Total)	3	2
Wertheim's Hysterectomy	2	2
Abdominal Hysterotomy	—	2
Myomectomy	1	2
Removal of fibroid polypi	—	3
Cervix :—		
Amputation of	1	—
Polypus, removal of	7	3
Cauterization (Electro) for erosion	19	15
Dilatation and Tubal insufflation	43	6
Tubes & Ovaries :—		
Ovariotomy	13	8
Salpingectomy	2	2
Salpingostomy	2	—
Salpingo-oophorectomy	4	2
Extra-uterine gestation	6	4

Miscellaneous :—	*T.Y.H.*	*G.C.H.*
Exploratory Laparotomy	2	3
Pelvic haematocele	1	—
Abscess of pelvis	1	2
Breast abscess	1	—
	—	—
Total :—	143	92
	—	—

Table No. III.

Nature and Number of Cases Treated Without Operations:

	T.Y.H.	*G.C.H.*
Refused operation	9	2
Constipation	—	2
Dyspepsia	1	—
Normal pregnancy	3	2
Hyperemesis gravidarum	1	1
Toxaemia of pregnancy—Hypostatic pneumonia	1	—
Pregnancy with ankylostomiasis	—	1
Ankylostomiasis	—	1
Hydatidiform mole	1	—
Carneous mole	1	—
Amenorrhoea	1	—
Dysmenorrhoea	—	1
Fibromyoma	1	—
Carcinoma of body of uterus	—	1
Carcinoma of cervix	4	5
Secondary carcinoma of vagina from carcinoma of cervix	1	—
Malignant ovarian tumour	—	1
Ovarian cyst	3	—
Neglected Post-abortum haemorrhage	1	—
Incomplete abortion	4	2
Threatened abortion	13	3
Inevitable abortion	15	3
Retained product of conception	1	—
Post abortive sepsis	1	—
Extra-uterine gestation	1	—
Endocervicitis	3	5
Prolapse	2	2
Prolapse and Primary cancer of thyroid	—	1
Rigid hymen	1	—
Imperforate hymen	1	—
Mitral Stenosis	1	—
Retroversion	4	2
Retroverted gravid uterus	1	—
Salpingo-oophoritis	7	8
Sterility	1	—
Cerebro-spinal syphilis	—	1

	T.Y.H.	*G.C.H.*
Syphilis	1	—
Malignant peritonitis	1	—
Pelvic peritonitis	1	—
Accidental tear of vaginal wall	1	—
Complete Hypospadias	—	1
Polyneuritis	—	1
Premature delivery, Avitaminosis B_1	—	1
For Observation	1	1
Total :—	89	48
Mortality	7	1

T.Y.H.
1. Multilocular pseudomucinous cyst—Heart failure.
2. Sarcoma in fibroid of cervical stump following sub-total hysterectomy.
3. Post-operative intestinal strangulation.
4. Neglected Post-abortum haemorrhage.
5. Incomplete abortion—Secondary anaemia.
6. Secondary anaemia—Heart failure.
7. Toxaemia of pregnancy—Hypostatic pneumonia.

G.C.H.
1. Carcinoma of cervix (Death following Wertheim's operation).

CLASSIFICATION OF DISEASES.

	T.Y.H.	*G.C.H.*
Valva :—		
Laceration of	1	—
Lipoma of right labium majora	1	—
Bartholin abscess	1	—
Hymen :—		
Imperforate hymen	1	—
Rigid hymen	2	—
Perineum :—		
Laceration	2	1
Vagina :—		
Traumatic laceration	1	—
Cicatricial Stenosis	1	1
Metastatic deposits from carcinoma of cervix	1	—
Vesico-vaginal fistula	—	5
Urethra :—		
Urethro-vaginal fistula	—	1
Urethral Stenosis	—	1

	T.Y.H.	*G.C.H.*
Uterus :—		
Congenital—Double uterus and vagina	—	1
Acquired—Displacements—		
Retro-displacement (Retroversion)	10	1
Utero-vaginal prolapse	7	12
Retroverted gravid uterus	1	—
Disorders of pregnancy—		
Incomplete abortion	17	7
Threatened abortion	13	4
Inevitable abortion	18	5
Carneous Mole	1	—
Hydatidiform Mole	1	1
Retained products of conception	1	—
Disorders of Menstruation—		
Amenorrhoea (Secondary)	1	—
Menorrhagia	—	1
Dysmenorrhoea	9	1
Subinvolution	1	1
Metropathia Haemorrhagica	2	—
Endometrial hyperplasia	1	—
Endometritis	—	2
Tuberculous endometritis	1	—
Endometrial polyp	1	—
Neoplasms—		
Fibroid polyp	5	3
Uterine fibroid	2	7
Endometrioma of uterus	—	2
Sarcoma	1	—
Adeno-carcinoma	2	2
Cervix :—		
Erosion of cervix	18	18
Hypertrophied	1	—
Polyp	5	4
Carcinoma	7	6
Tubes & Ovaries :—		
Inflammation—		
Acute Salpingo-oophoritis	2	—
Chronic Salpingo-oophoritis	11	12
Tubo ovarian abscess	1	—
Pelvic abscess	1	3
Extra-uterine pregnancy	9	4
Pelvic haematocele	1	—

	T.Y.H.	*G.C.H.*
Neoplasms—		
Follicular cyst	2	—
Ovarian pseudomucinous cystadenoma	15	7
Papilliferous cyst	1	—
Dermoid cyst	1	1
Fibroma	1	—
Broad ligament cyst	3	1
Carcinoma of ovary	—	1
Sterility	32	7
Miscellaneous :—		
Malignant peritonitis	1	—
Pelvic peritonitis	1	1
Neglected Post-partum haemorrhage	1	2
Breast abscess	1	—
Fibro-adenoma of breast	1	—
Heart failure (Mitral Stenosis)	1	—
Toxaemia of pregnancy-Hypostatic pneumonia	1	
Normal pregnancy	3	3
Syphilis	1	—
Cerebro-spinal syphilis	—	1
Hyperemesis gravidarum	1	1
Dyspepsia	1	—
Constipation	—	2
Post abortum sepsis	1	—
Complete Hypospadias	—	1
Ankylostomiasis	—	1
Abdominal tumour	—	1
Polyneuritis (Avitaminosis B_1)	—	2
Diverticulitis	—	1
For observation	1	1

THE RÔLE OF THE SUBEPITHELIAL LYMPHATIC GLANDS.

KENELM H. DIGBY.

(Read before a combined meeting of the Hong Kong University Medical Society and the Chinese Medical Association.)

Distribution of Lymphoid Tissue.

Lymphoid tissue occurs in three different ways:—

(1) Associated with *blood vessels* in the spleen, in red bone marrow and in haemolymph glands.

(2) In the *interstitial lymphatic* glands (1) which occur in crevices or intervals in the body. They receive lymph from large areas of the body. Efferent vessels carry the lymph eventually into the veins. The axillary, bronchial and cervical lymphatic glands are examples.

(3) In the *subepithelial lymphatic* glands (1) which occur in certain parts of the body each immediately beneath an overlying epithelium. They receive no afferent lymphatics; but efferent vessels run from them to neighbouring interstitial lymphatic glands. The tonsils, Peyer's patches and vermiform appendix are instances of subepithelial lymphatic glands.

In the first group—associated with blood vessels—there is evidence that the lymphoid tissue receives and destroys toxins and bacteria which have entered the blood stream. These are destroyed and an excess of antibodies produced.

In the second group—the interstitial lymphatic glands—the lymphoid tissue deals similarly with any toxins and bacteria in the entering lymph. McMaster and Hudack (2) have produced evidence that agglutinins are actually manufactured in the *interstitial lymph gland.*

The action of the third group—*subepithelial lymphatic glands*—is considered in the ensuing paragraphs.

Structure of Lymphoid Tissue.

Lymphoid tissue is composed of units known as *lymph follicles.* There are two principal tyes of cells:—

(*a*) A small one with darkly staining nucleus and very little cytoplasm,

(*b*) A large cell having a large pale nucleus and abundant cytoplasm.

These may be termed small and large adenocytes* respectively. The small adenocytes probably pass into the blood to form the small lymphocytes there. The large adenocyte is usually identified as belonging to the reticulo-endothelial system. Some of the large adenocytes possibly

pass on to form large lymphocytes in the blood. The large adenocytes commonly are grouped in the centre of a lymph follicle, being surrounded by a zone of small adenocytes. Giant cells—giant adenocytes—also occur in lymphoid tissue in some situations in certain animals.

Ingestion of Bacteria. Transepithelial Migration and Phagotaxis.

In subepithelial lymphatic glands, such as tonsils, Peyers patches, and appendix, the lymph follicles are situated beneath a delicate epithelium on the free surface of which bacteria are both numerous and active. Many of the adenocytes work their way between the overlying epithelial cells to the surface. (Figs. 1 & 2). This was described by Stohr (3) and Heidenham (4). A few break loose altogether and are swept along in the alimentary contents to be lost to the body. A much larger number however attack and engulf bacteria in contact with the epithelium. Then laden with micro-organisms these adenocytes work their way back through the epithelium once more and on to the centres of the lymph follicles with their prey. (Figs. 2 & 3). It is inside the larger adenocytes that the final stages of digestion of the bacteria takes place and that high antibacterial powers are developed. (Figs. 3, 4 & 5).

Foot-note.—*From the Greek word meaning "gland" which has often been used in connection with collections of lymphoid tissue. By using adenocyte to signify the cells in lymphatic glands, one avoids any confusion with lymphocytes in the blood-stream or elsewhere.

To quote from a discussion on the reticulo-endothelial system (5) "It is reasonable to assume that the tissue which originally destroys a foreign substance is also the origin of antibodies to it; the very process of destruction may be that in the course of which antibody formation occurs." The conveyance of the bacteria through the epithelium into the lymph follicle is known as phagotaxis (1). The intracellular digestion of the bacteria may be termed physiological phagocytosis. Why physiological? Because it is continuously occurring in healthy animals. The whole process can be called the *Ingestion* of *Bacteria* by the *Subepithelial Lymphatic Glands*. (10).

Many of the small adenocytes which have undertaken the transepithelial migration and have survived their encounter with bacteria pass to adjacent lymph glands and so on into the blood stream to become the small lymphocytes of the blood. Thus the subepithelial lymph glands are not only like munition factories in that they probably produce a great excess of opsonins, bacteriolysins, agglutinins and other anti-

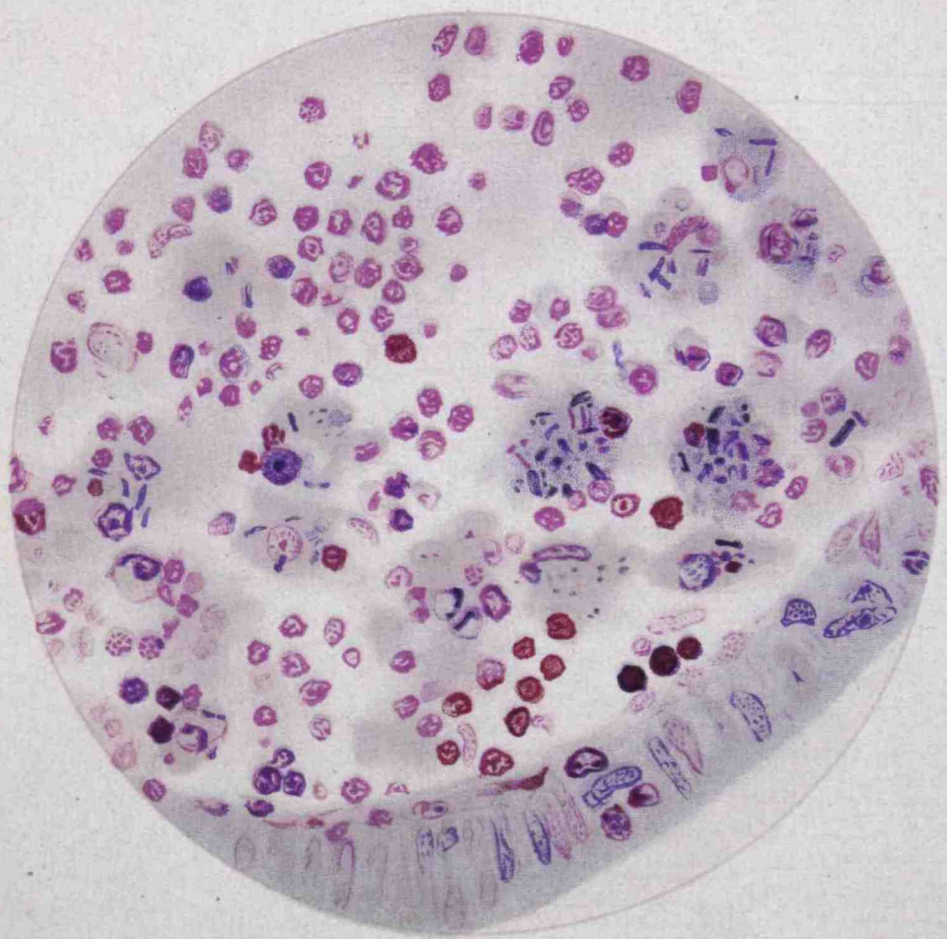

FIGURE 1.

Painting by Dr. Lien Tsoong Kya of a section of a healthy rabbit's appendix stained with Gram-safranin. (1/12 inch oil immersion objective and No. 12 eyepiece—magnification 1200).

This shows:—

1. Transepithelial migration of adenocytes.
2. Physiological phagocytosis in a superficial lymph follicle.

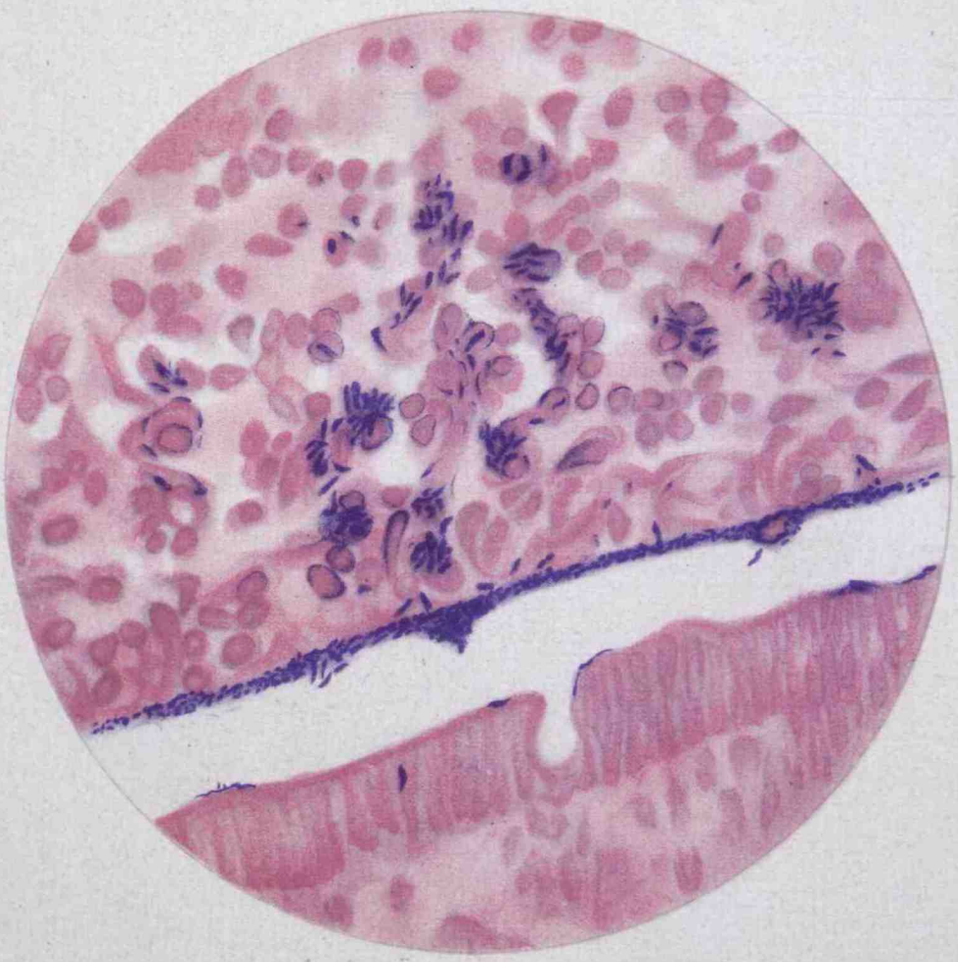

FIGURE 2.

Painting by Dr. Lien Tsoong Kya of a section of a rabbit's appendix stained with Gram-eosin. No nuclear stain has been used.

The deep part of a calyx crypt is shown with phagotaxis through the epithelium covering a superficial lymph follicle.

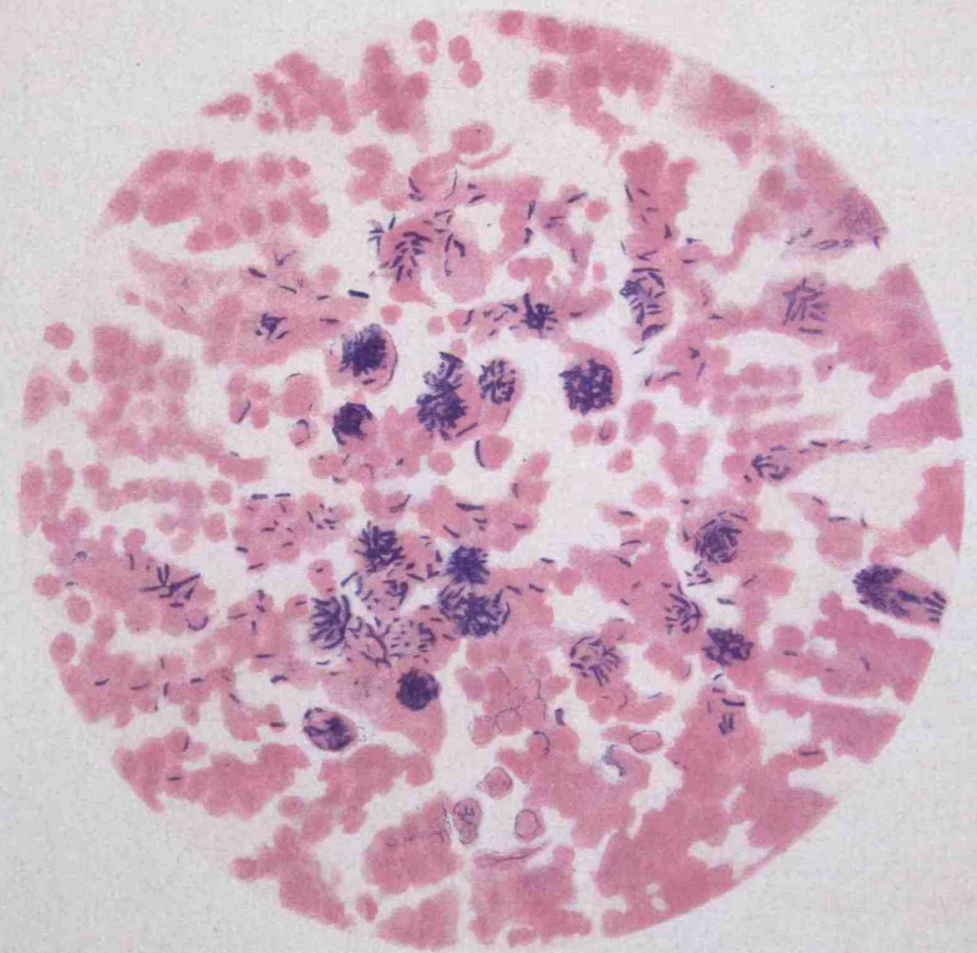

FIGURE 3.

Painting by Dr. Lien Tsoong Kya of a section of a healthy rabbit's appendix. (Gram-eosin stain—the nuclei are uncoloured).

The centre of a superficial lymph follicle is seen with numerous intracellular bacilli in the process of being destroyed.

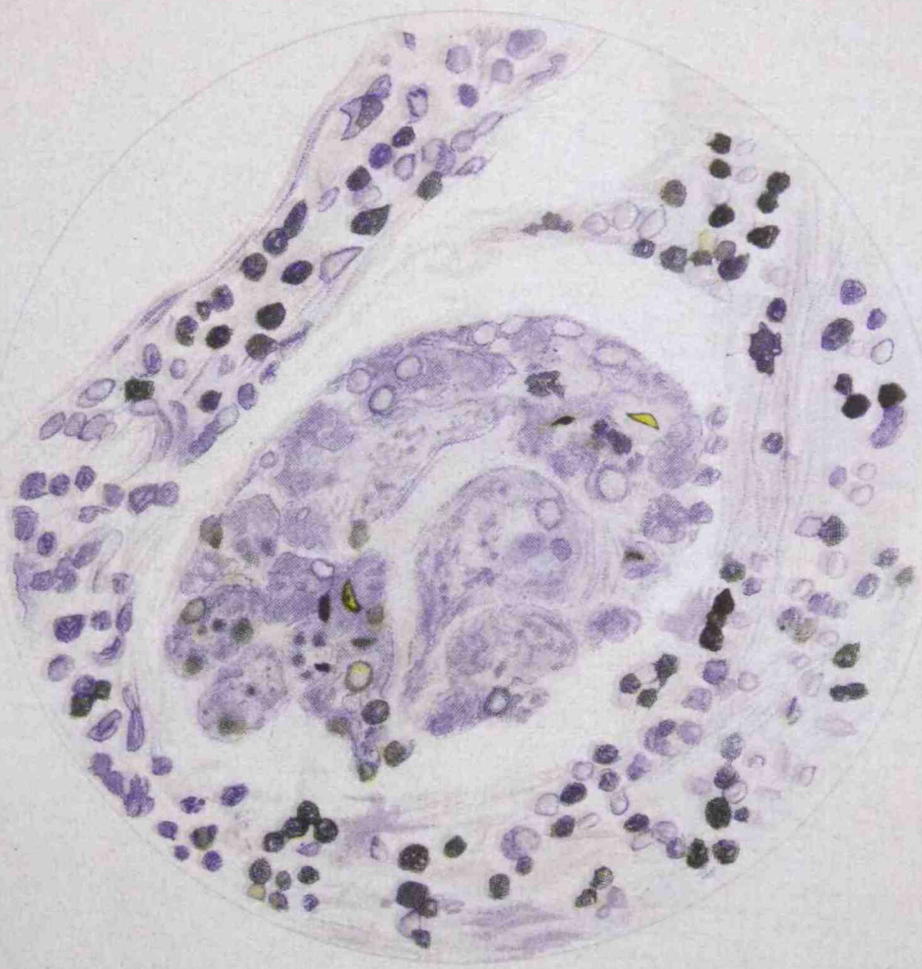

FIGURE 4.

Painting by Dr. Lien Tsoong Kya of a section of a healthy rabbit's appendix.

A mass of cells in one of the deeper lymph follicles. The cell outlines are not easily distinguished. Giant cells may be seen enclosing smaller cells. Stained bacilli are not seen in this view but débris and a few yellow refractile crystals are visible.

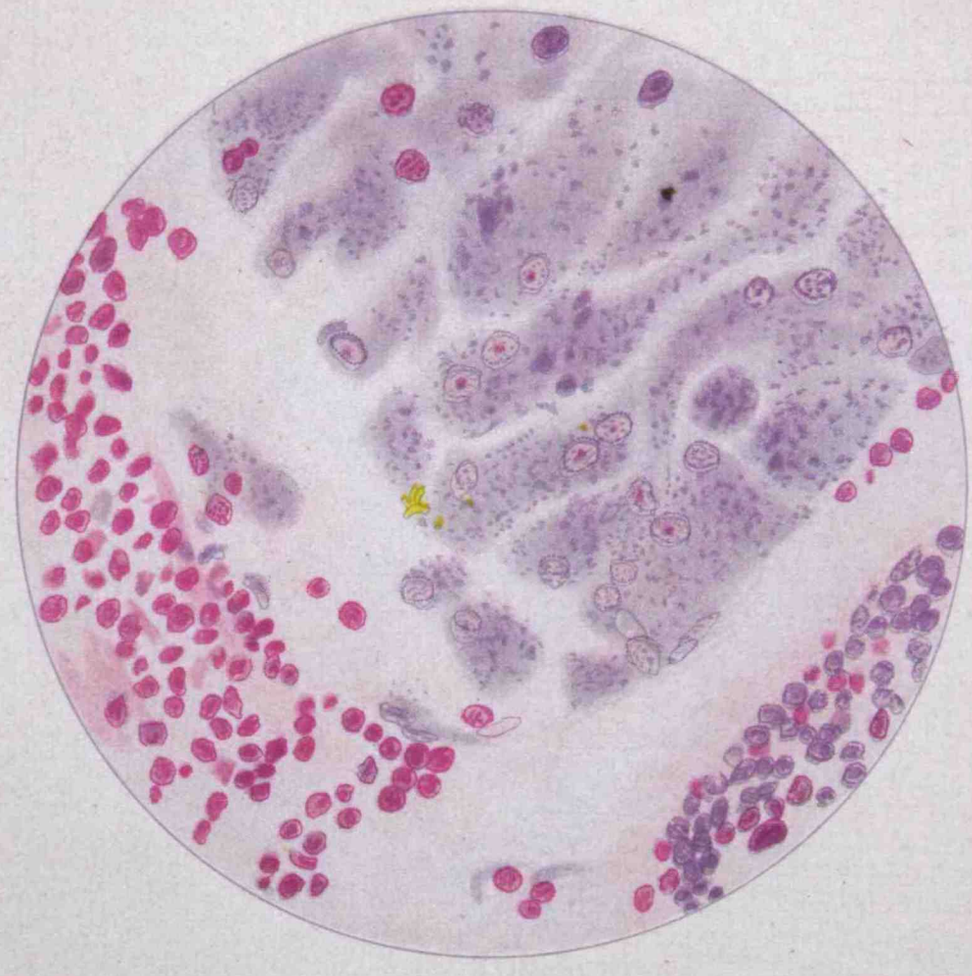

FIGURE 5.

Painting by Dr. Lien Tsoong Kya of a section of a healthy rabbit's appendix.

Part of a mass of giant adenocytes crammed with débris in one of the deeper lymph follicles.

Stained bacilli are no longer clearly recognised but the enormous adenocytes with large nuclei form a pale area contrasting with the more deeply stained lymphoid tissue around.

The cytoplasm of the giant cells includes the débris of bacteria. A clump of yellow refractile crystals and two or three nuclei of small adenocytes which have been engulfed by the giant cells are to be seen.

bacterial weapons, but they also seem analogous to police training schools or military academies in supplying the blood with cells specially trained, experienced and selectively tested in fighting the bacteria adjacent to, and therefore most likely to invade, the body.

Occasionally the enclosed bacteria may threaten the life of an adenocyte which will then in its turn be phagocyted by a giant adenocyte. Large masses of such cells loaded with debris are to be found in some animals. (Figs. 4 & 5). The masses may even be large enough to be visible to the naked eye. At other times an adenocyte may have surrounded resistant but not quickly virulent bacteria such as tubercle bacilli. With these inside them they may reach the neighbouring interstitial lymph gland with their indigestible meal. It is possible that adenocytes may enter the blood stream with bacteria inside them and convey them to those other antibacterial workshops the spleen and the bone marrow.

Evidence for this Ingestion of Bacteria.

The presence of "enormous numbers of intestinal bacteria which in their various shapes and sizes were identical with those always present in the intestinal contents," in the lymph follicles of the rabbit's appendix and sacculus rotundus was discovered independently in 1885 by Ribbert in Germany (6) and by Bizozzero in Italy (7) Ribbert carefully excluded the possibilities of post-mortem invasion or of mechanical carriage of organisms by the sectioning knife. Bizzozero took control sections which showed that the passage of bacteria through the epithelium did not take place to any extent except where it overlay lymph follicles. In 1890 Ruffer, working under Pasteur and Metchnikoff in Paris (8), confirmed and amplified their observations and extended them to the Peyer's patches and tonsils of dogs and guinea pigs. In the dog's tonsils he particularly noted the presence of inert particles such as carbon as well as bacteria in the lymph follicles. In 1890 Pirera experimentally showed the ingestion of inert particles and of bacteria in human tonsils (9).

In 1911 the writer of this note proposed on theoretical grounds that the subepithelial lymphatic glands had an immunising function (10) (11) and in 1912 began to look for bacteria in lymph follicles in Professor Lorrain Smith's laboratory in Manchester, being then unaware of the previous observations recorded above. The presence of bacteria was easily demonstrated and reported in 1913 (12). In 1919 with Professor (now Sir) Arthur Keith's kind help and encouragement a book was published (1) and the whole subject has been elaborated in later papers (13).

In 1935 Dr. Gordon Thompson of the Lester Institute of Medical Research in Shanghai observed bacteria in the lymph follicles of appendices of rabbits on a special deficiency diet. (At present unpublished).

The demonstration in rabbits is facilitated by the fact that the principal intestinal bacteria are Gram-positive bacilli. The appendices must be removed immediately on killing the animals, fixed in alcohol and stained with Gram-eosin. The nuclei are better left unstained, and the Gram stain must be used delicately, as the bacteria undergoing phagocytosis decolour rather readily.

The specimens illustrated in the present paper were exhibited at the Pathological Society of Great Britain and Ireland in July 1936. I am indebted to my colleague, Professor L. J. Davis, for help and advice with the microscopical preparations and to Dr. T. K. Lien for the paintings.

Auto-Immunising Stations.

The subepithelial lymphatic glands are thus the auto-immunising stations of the body. (10). They develope and maintain a high degree of immunity towards the bacteria of the alimentary canal. They provide early defensive measures against pathogenic bacteria which come in contact with the weaker coverings of the body.

Immunity Acquired during Health.

When a fresh infection attacks a community for the first time the ravages are widespread and serious. Subsequent attacks of the same infectious disease are far less serious for the community has become less susceptible. This rise in the general level of immunity may be due to the fact that many of the most susceptible had succumbed to the disease, that others moderately susceptible had contracted the disease but recovered with resulting immunity. But there is another factor: most of those who have escaped an illness have yet been exposed to contact with the virus and have developed some degree of immunity. Thus in a school where diphtheria is endemic, Dudley has shown that the percentage of immune children increases with each year at the school apart from attacks of the disease (14). And it is of significance that this healthily acquired immunity was not manifested in a small group of boys from whom nasopharyngeal and faucial tonsils had been completely removed. (15). Moreover it has been shown by Collins (16) that disease or removal of the tonsils increases the liability to bronchitis, pneumonia, rheumatism, heart diseases, ear diseases and cervical adenitis; and that the incidence rate for measles, mumps, whooping cough and chicken pox is increased significantly in children

whose tonsils have been removed. Even if the virulence or the dose of the bacteria be overwhelming the subepithelial lymphatic glands bear the brunt of the attack and permit the building up of some protection for the general system. The increased number of small lymphocytes in the blood in infectious diseases where the subepithelial lymphatic glands are specially involved supports this point.

It should be remembered that the subepithelial lymphatic glands are largest and most active in the young when immunity against many infectious diseases is being acquired. (10).

Oral Vaccines.

The administration of bacterial cultures by the mouth in appropriate doses will produce immunity against the bacteria. This has been shown to be the case for typhoid and paratyphoid fevers, (17, 18 & 19), for Malta fever, and for bacillary dysentery (20) and possibly for tuberculosis. Immunity in animals against pneumococci can be attained by oral vaccines as shown by the protection afforded against multiple lethal doses of pneumococci injected intraperitoneally. (21). It has been shown in man that agglutinins appear in the blood after oral vaccination in the case of bacillus typhosus, and bacillus paratyphosus by Hoffstadt and Thompson (22) and others quoted by Thomson, Thomson, and Thompson (23), and in the case of Pfeiffer's bacillus influenzae and streptococci. (23).

Further literature concerning the protective action of oral vaccines in coryza, typhoid, cholera and bacillary dysentery can be cited. (34). But the author has only seen abstracts of these and has not yet been able to study the full papers.

We know that bacilli are rendered harmless and taken up whole by phagotaxis into the subepithelial lymphatic glands and little if at all elsewhere. It is hardly credible that their mere presence in the lumen of the alimentary canal could produce immunity, and the conclusion is that oral vaccines produce their good effect by virtue of the subepithelial lymphatic glands.

Such is a very brief survey of the beneficent activities of the subepithelial lymphatic glands.

Occurrence in Man.

In man the subepithelial lymphatic glands include faucial, nasopharyngeal and lingual tonsils, the solitary follicles of the lesser curvature of the stomach, of the duodenum, jejunum, ileum and large intestine, the Peyer's patches and the vermiform appendix. Other collections of subepithelial

lymphoid tissue occur; for instance beneath the conjunctiva of the upper eyelid, and beneath the ciliated epithelium of the trachea and bronchi. It will be seen that subepithelial lymphoid tissue is found not beneath the skin with its tough corium but (i) where the epithelium is delicate and absorbing, (ii) where fresh strains of bacteria first arrive (iii) where bacteria are most active and numerous.

Development of the Subepithelial Lymphatic Glands.

The biological development of these subepithelial lymphatic glands is full of interest. Wandering phagocytic cells occur in the tissues of invertebrates. These attack and engulf invading bacteria, but no definite subepithelial groups have been described. In the lowest vertebrates—fishes—the wandering amoebocytes are numerous in the loose tissues beneath the delicate respiratory epithelium of the gills but permanent encapsulated collections beneath the epithelium do not occur. Nor, save in rudimentary form, have they been described in amphibians or snakes.

This is hardly surprising. Should a subepithelial lymph collection continually ingest bacteria from the alimentary canal in a cold blooded animal, a great fall in external temperature, resulting in a considerable drop in body temperature, would adversely affect the complex lymphoid tissue more than it would the lowly organised bacteria. So that even mild bacteria ingested might begin to overcome the adenocytes.

But in the warm blooded birds and mammals the evolution of the subepithelial lymphatic glands is successful. We can fairly suppose that where pathogenic bacteria lie close to delicate absorbing epithelia defensive phagocytic cells would collect being attracted chemiotactically, and serving to resist any chance invasion. Where a uniform temperature favourable to the phagocytes was maintained, any genetic variation whereby lymphocytes became permanently stationed under the epithelium, constantly passing to and fro between the epithelial cells, sampling the intestinal contents and thus raising the general resistance—any such genetic variation would tend to persist on account of the great benefit to the individual possessing it. And further variations in the direction of providing local backwaters or blind recesses where (i) the bacteria could incubate; (ii) adenocytes could reach the surface without danger of being swept away; and (iii) where the surface of contact between lymphoid tissue and epithelium could be enormously increased—would tend to be encouraged by natural selection. Thus one envisages the evolution of the tubular appendix in some animals, of

the recessed Peyer's patches in ruminants, of the tubular tonsil which occurs in many animals, of the clefts of the nasopharyngeal tonsil, of the crypts of the faucial tonsils in man and of the calyx recesses in the wall of the rabbits appendix.

Clearing Mechanism.

It may be noted that mechanisms exist for the periodical emptying of these stagnant areas. Thus the musculature of the appendix can cause its evacuation, whilst the contents of the tonsillar crypts tend to be squeezed out during certain movements of the throat. During swallowing the surrounding muscles push the tonsil between the pillars into the throat but when the tonsillo-pharyngeus contracts at the same time as the surrounding muscles, the tonsil cannot escape the pressure, and the contents of the crypts are squeezed out. Moreover when organisms of great virulence are growing in these blind areas very many more adenocytes pass out through the epithelium than ever return, and fighting with the bacteria may gradually destroy them.

"Cut Out" Mechanism.

It is probable that excessively pathogenic organisms destroy or repel the adenocytes before phagocytosis can take place, and no lymphoid ingestion by phagotaxis occurs. In that way the subepithelial lymph glands are at times automatically thrown out of action when called upon to bear so great a strain that they would inevitably be overcome.

Comparative Anatomy.

The following account of the comparative anatomy of the subepithelial lymphatic glands is based on the works and observations of Huntington (24), Berry (25), Hett (26), Hett and Butterfield (27), Cave (28), Alagna (29), Owen (30), and the present writer (1) and on the excellent collection of specimens in the Hunterian Museum of the Royal College of Surgeons of England.

It has already been stated that the subepithelial lymphatic glands are found in a highly developed state only in the warm-blooded birds and mammals—the apices of evolution up to the present. The best known example of subepithelial lymphatic glands in birds is presented by the paired avian caeca at the junction of small and large intestine. They are almost universally present. These avian caeca have walls thickened in part or throughout by a layer of subepithelial lymphoid tissue. Nasopharyngeal lymphoid tissue has also been described in birds.

Throughout the class of mammals with hardly any exceptions subepithelial lymphoid tissue is distributed as solitary lyemph follicles scattered along nearly the whole

length of the alimentary canal and large collections occur at the fauces, along the ileum and in the caecum. The naso-pharyngeal tonsil exists in many mammals. In the sheep there is a tonsil on the posterior part of the septum.

The faucial tonsils sometimes as in man present themselves as a protuberant mass pitted with deep crypts. In other mammals, as the rabbit, a blind tonsillar tube lined by subepithelial lymphoid tissue runs in a forward direction.

Similarly at the caecum the subepithelial lymphatic tissue presents itself at the apex of the caecum sometimes as a tube lined by lymphoid tissue the vermiform appendix, at other times, dog, cat, lion, lynx, as an inwardly projecting caecal tonsil. Nearly all mammals contain subepithelial lymphoid tissue in the caecum in some form or other.

A vermiform appendix is present in a larger number of mammals than is generally known. It occurs in :—

Monotremes —echidna tachyglossus (anteater)

Marsupials —phascolarctus cinereus (koala)
phascolymis wombat (wombat)
phascolymis mitchelli (wombat)
trichosurus vulpinus (phalanger)

Ungulates —babirusa (boar)
dicotyles torquatus (peccary)
tapirus americanus (tapir)

Rodents —avricola amphibius (water vole)
castor fiber (beaver)
carpuncho capybara (a very large rodent)
cercolabes
erythizon dorsatus (Canadian percupine)
jerboa sagitta (jerboa)
lepus cuniculus (rabbit)
lepus timidus (hare)
mypotamus (South African water rat)

Carnivores —felis pardalis (puma)
herpestes griseus (ichneumon)
viverra civetta (civet)
chiromys madagascarensis (aye-aye monkey)
lemur catta (ringtailed) } lemurs
lemur anjuanensis
lemur macaco
nycticebus javanicus } lorises
nycticebus tardigradus (slow)

Lower Anthropoids—*New World Monkeys.*

ateles (spider monkey)
hapale pencillata (marmoset)
lagothrix

Old World Monkeys.

cercopithecus pencillatus
cercopithecus tantallus

Higher Anthropoids—

anthropithecus troglodytes (chimpanzee)
hylobates leniscus } (gibbons)
hylobates lar }
gorilla savagei (gorilla)
simia satyrus (orang-outan)

Hominidae —homo sapiens (man).

Peyer's patches are of widespread occurrence. In the pig they are said to be replaced by a strip of subepithelial lymphoid tissue several feet long.

In rodents, such as the rabbit, the hare, the doormouse and chichilla laniger, two further subepithelial lymphatic glands occur at the junction of the small and large intestines —that is on either side of the ileocacecal valve. The ileal gland, sometimes occupying a pouching of the ileum known as the saccus endolymphaticus lies in the long axis of the ileum; the caecal gland lies in the adjacent caecal wall with the axis at right angles to the axis of the ileal gland. This latter is in a pouched part of the caecum in the hare which even, it is said, becomes a second appendix in lagomys pusillus. (I have however been unable to trace this animal). The ileo-caecal patches also appear in the sheep and the giraffe, and the ileal one lies in a slight pouch of the mucous membrane.

Lymphoid tissue in the caecum may be

1. terminal, as a vermiform appendix,
   or as an inwardly projecting caecal tonsil.
2. basal, as one of the ileo-caecal patches.
3. lateral, as in rats and mice.

Embryology.

Just as lymph follicles appear late in the evolutionary scale of animals, so also they appear late in the development of the individual. In the tonsillar region lymph follicles begin to appear just before birth; in the caecal region they are first seen directly after birth and develope rapidly with the growth of alimentary bacteria.

Advantageous Balance.

So far this paper has dealt only with the beneficent activities of the subepithelial lymphatic glands. They act at times, however, in maleficent and deleterious ways. Yet there can be no doubt whatever that their benefits outweigh their disadvantages or they could not have developed and persisted in the two highest classes of vertebrates.

Pathology.

It is not surprising in view of the dangerous nature of their important and beneficial work, that the subepithelial lymphatic glands should sometimes fail and even occasionally in their failure themselves become a threat to the bodily welfare. It is interesting to consider how this may come about.

Subepithelial Lymph Gland Disease.

The subepithelial lymphatic glands may give trouble in the following ways:—

(1) *Excessive increase in size.*

(2) *Acute inflammation.*

(3) *Recurrent inflammation.*

(4) *Chronic inflammation.*

(5) *Malfunction from cicatrisation following inflammation.*

(6) *Portals of entry of disease.*

(7) *Chronic foci of infection.*

Excessive Increase in Size.

Over activity leads to hyperplasia—the whole gland enlarges. In some cases the enlargement may be so great as to set up mechanical troubles: (a) when the nasopharyngeal tonsil is involved in children the nasopharynx may become obstructed, mouth breathing replaces nose breathing and the development of the nasal cavities and of the upper dental arch is retarded: (b) when the lowest Peyer's patch in infants enlarges it projects into the lumen of the ileum and may be gripped by a peristaltic wave and an intussusception be initiated: (c) when the subconjunctival lymph follicles enlarge in trachoma the cornea is irritated and the results may be serious. (It is however by no means clear that all the granules of trachoma represent preexisting subepithelial lymph follicles.

Acute Inflammation.

Unfortunately it sometimes happens that the ingested bacteria overpower the adenocytes in one or more lymph follicles. The local blood vessels dilate and polymorphonuclear leucocytes join in the fray. An acute lymphadenitis is probably the starting point of acute tonsillitis, gastric and duodenal ulcer typhoid, and paratyphoid fevers, tuberculous enteritis, acute appendicitis and some forms of dysentery. When the infecting

organisms are pyogenic the lymph follicle becomes a small abscess. Owing to the superficial situation of the lymph follicle the abscess bursts into the lumen and a small ulcer forms. Often in appendicitis where many follicles are affected numerous small abscesses or ulcers may be seen.

These ulcers of the subepithelial lymph follicles probably heal quickly in most instances, but not always. Where the ulcer is exposed to the gastric juice for instance the hydrochloric acid may rapidly eat through the whole thickness of the wall and a perforation occur. Or an artery may be breached and severe haemorrhage follow. Sometimes the extreme virulence of the infecting organism may produce similar disasters. Perforation will occur in appendicitis and both haemorrhage and perforation in typhoid fever. Occasionally the submucous abscess may burrow deeply and burst the other way from the lumen causing a localised peritoneal abscess in the case of the appendix, a peritonsillar abscess or quinsy with the faucial tonsil, an acute retropharyngeal abscess with the nasopharyngeal tonsil and deep submaxillary cellulitis (Ludwig's angina) with the lingual tonsil. The greater frequency of the first of these—quinsy—may be associated with the deep crypts with their liability to become blocked.

Recurrent Inflammation.

In some of the subepithelial lymphatic glands one acute attack seems to predispose to further attacks. This may be due to minute damage to the follicles or overlying epithelium. But often it is the sequel of cicatricial contraction resulting from ulceration in the first attack. This cicatricial contraction may cause stenosis of the tubular appendix or of a tonsillar crypt leading to stagnation or distension or excessive decomposition distally or to inspissation and calcification of contents with formation of concretions.

Chronic Inflammation.

Sometimes the nature of the infecting organism leads to chronicity. Thus dysenteric and tuberculous ulcers tend to persist. Tuberculous disease of Peyer's patches is much more common than tuberculosis of tonsils and appendix. As has been recalled previously the sluggish tubercle bacilli are often transmitted to a neighbouring interstitial lymph gland before they overcome the adenocytes. Thus tuberculous disease of the upper deep cervical glands of the mesenteric glands and of the ileo-colic glands are more frequent than tuberculosis of the tonsils, Peyer's patches and vermiform appendix respectively.

Actinomycosis is an occasional cause of chronic inflammation of the vermiform appendix.

Trachoma has been referred to as an hypertrophy. It is however partly a chronic inflammation also.

The acid gastric juice will sometimes cause chronicity in a simple ulcer from subepithelial lymphadenitis of the lesser curvature and of the first part of the duodenum.

Pyogenic organisms by themselves do not usually cause *chronic* inflammation of subepithelial lymphoid tissue.

The so called "chronic appendicitis" is far more often malfunction due to scar tissue than chronic inflammation.

Malfunction from Cicatrisation following Inflammation.

The tonsils frequently show fibrosis even though inflammatory attacks have been infrequent. The vermiform appendix by the time the age of sixty has been reached shows cicatrisation, stenosis or adhesions in over 50% of people, generally in the absence of a history of clinically recognisable attacks of appendicitis. (32). The physiological immunising action of the subepithelial lymphatic glands frequently passes on to slight pathological processes which leave cicatrices behind. Apart from predisposing to further and maybe more serious inflammatory attacks, the scars may interfere mechanically with the movements of the organ itself or of a neighgouring organ. Thus it is probable that a srticture of the appendix or adhesions thereto may hinder the evacuation of its contents and cause muscular tension in the wall distal to the obstruction; this may lead to local pain and tenderness or may reflexly lead to a spasmodic hold-up of some part of the alimentary tract and so to gastric or ileal or caecal or colonic stasis. Or a concretion in the appendix distal to the obstruction may be a source of trouble. Or the appendix adherent at some point may act as an anchor interfering with the free movement of the caecum. In these and other ways old scarring may lead to malfunction causing symptoms.

The difficulty is to tell when we are dealing with such a case. It is well know that many other conditions will cause pain and tenderness in the right iliac fossa. And scarring of the appendix obvious to the naked eye is so common in adults (25% in all cases, but rare in children)—according to Birmingham, quoted by Shennan. (32). Yet it is the exception for such scarring to cause symptoms. We cannot *for certain* say that an appendix is malfunctioning to the point of producing symptoms, even when X-ray examination shows a filling defect or immobility to palpation or delayed evacuation. Nor even directly after removal when we actually see stenosis or adhesions of the appendix

can we be sure that it was the source of the trouble for we know that these lesions are frequently present with no accompanying symptoms. It is a truism that all other possible causes should be excluded as far as possible before removing an appendix for chronic right lower abdominal pain, if one is to keep at the minimum number those disturbing cases where symptoms persist after removal of the organ.

As in this paper gastric and duodenal ulcer are ascribed initially to inflammation of subepithelial lymph nodes, the late results of scarring of these ulcers namely pyloric obstruction and hour-glass stomach should be included under the heading of malfunction from cicatrisation.

Portals of Entry.

Clinical experience leads one to think that infection not infrequently enters the body via the subepithelial lymphatic glands, even though these may have shown no signs of disease, or perhaps only a slight transitory lesion. Reference has already been made to tuberculosis of the upper deep cervical and of the mesenteric glands. Other forms of tuberculous disease have a similar source. Acute suppuration of the upper deep cervical glands occasionally follows acute tonsillitis. Rheumatic endocarditis may be preceded by tonsillitis; and so on.

A recognition of all this however does not *necessarily* justify removal of the subepithelial lymphatic gland concerned. If a burglar breaks into a house through a certain doorway, one does not take the door off its hinges. And though this analogy must not be taken too seriously, yet it may well be that an illness due to an invasion via one of the subepithelial lymphatic glands is far less serious than an invasion through some less well protected mucous membranes, and that the brunt of the attack has been borne by such gland and that some degree of immunity—even if insufficient to abort the trouble—has been conferred before the general invasion of the body.

Chronic Foci of Infection.

A very attractive and popular theory has been that one or other of the subepithelial lymph glands may act as a chronic focus of disease from which periodical invasions of the general system take place. The outpost, it is supposed, has been captured and now serves to shelter forces of the enemy which use it as a base for further raids at long intervals. The cul-de-sac arrangement of many of the subepithelial lymphatic glands, the liability to acute inflammatory attacks often leading to contracting scars which tend to block the blind tubes leaving stagnation or even buried abscesses

would appear to predispose to the formation of such chronic foci.

Retention abscesses or cysts however are not very common and when present do not appear to have acted as chronic foci. Moreover, as long as lymphoid tissue remains, such foci would be surrounded by excellent antibacterial defences. Nevertheless the view has proved very appealing. The faucial tonsils have been removed for acute rheumatic endocarditis, for chronic fibro- and osteo-arthritis, for chronic nephritis and for a host of other chronic lesions characterised by intermittent exacerbations. Clearly the infection is remaining in the system and the question becomes "Is it sheltering on the one hand in the vegetations on the cardiac valves or in the structures of the damaged joints or in the tissues of the diseased kidneys respectively; or on the other hand does it lurk in the recesses of some subepithelial lymph gland?" Does amelioration of such chronic diseases follow removal of some subepithelial lymph gland—as a possible chronic focus—more often than in cases not so treated? Only the most carefully collected statistics can supply staisfactory answers to these questions. Kaiser has produced valuable evidence on these lines (33).

Significance of the Auto-Immunising Theory.

It must be admitted that the theory of the immunising function of the subepithelial lymphatic glands has not been absorbed nor perhaps even accepted by the medical profession. From a philosophical point of view, the theory clears up the baffling problems of the etiology of appendicitis and tonsillitis. It dissipates the perplexing anomaly of these organs being so frequently the site of serious and even fatal illness and yet developing and persisting in the highest types of animals.

Effects of Acceptance of the Theory.

One of the effects of a general acceptance of the views expressed in this paper would be the impetus given to oral vaccination. That a high degree of immunity to many diseases can be acquired by oral administration of dead bacteria is certain. The employment of the natural organs for acquiring immunity—namely the subepithelial lymphatic glands—has however not been comprehensively studied. To bring the circumpharyngeal ring of lymphoid tissue into full play vaccines might have to be run into the nose and brought back into the mouth and then gargled before being finally swallowed. To utilise the extensive lymphoid tissue in the intestines the destructive action of the acid gastric juice must be combatted, whether by coating with keratin or by administering the vaccine with bile which neutralises the acidity in, and ensures the rapid emptying of, the stomach.

In surgical work oral vaccination of the particular bacteria cultivated from the patients own suppurating wounds should be tried. Preoperative oral vaccination by stock pyogenic cultures might be a useful routine procedure. Oral vaccination by dead tubercle baccili as prophylaxis or treatment of tuberculosis disease requires further trial.

On the negative side the conceptions here advocated will have a slightly conservative tendency with regard to the removal of tonsils and appendix.

But this statement must not be misunderstood. Because the tonsils, for instance, have an important function it does not follow that when diseased they may not be removed. The very nature of their work exposes them peculiarly to disease. Moreover the abundance of other subepithelial lymphoid tissue left may take over the work of one diseased part removed. A still closer and more critical study of what constitutes disease justifying removal is indeed demanded. Certainly not the presence of bacteria in the crypts of the tonsils nor even in the lymph follicles. Not the presence of lymphocytes in the crypts. And not merely scarring or fibrosis.

There can be no two sides with regard to the necessity for removing an acutely inflamed appendix. Chronic appendicular malfunction, is however a more difficult problem.

Further critical study of the symptoms of an appendix malfunctioning so as to need excision is required. And while, when the abdomen has been opened on such a diagnosis it is wiser to remove an appendix which looks normal to the naked eye from the outside, yet if the abdomen has been opened for some other condition, the appendix appears normal and there have been no symptoms referable to that organ then it should not be removed. In other words it is not sound surgery to remove an apparently healthy appendix merely because the abdomen is open and there are a few minutes to spare. It can no longer be said that the appendix is useless, might become inflamed one day and is therefore better out in everybody.

Summary.

1. This paper groups together tonsils, adenoids, Peyer's patches, solitary follicles, vermiform appendix and similar structures into one system—the subepithelial lymphatic glands.
2. It asserts that they all share a precise and definite function—namely that of automatically immunising the body during health against adjacent bacteria.

3. It states that the mechanism of this immunisation in health is by phagotaxis and physiological phagocytosis on the part of the lymph corpuscles here called adenocytes.

4. It also claims that susceptible persons exposed to an epidemic tend to acquire immunity physiologically—altogether apart from contracting the disease; and that this power of healthy immunisation is impaired by damage or removal of subepithelial lymph glands.

5. It declares that the subepithelial lymph glands are most widely distributed and most complexly developed in the two highest classes of vertebrates namely birds and mammals, the warm-blooded animals.

6. It points out that the immunising nature of their work peculiarly exposes the subepithelial lymphatic glands to certain diseases: a. excessive enlargement acute, recurrent and chronic inflammation, malfunction from cicatrisation, acting as portals of entry of disease and, possibly, acting as chronic foci of disease.

7. Some of the effects of acceptance of these views are discussed.

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MUSEUM CATALOGUE

SUPPLEMENT TO THE

MUSEUM CATALOGUE

DEPARTMENT OF PATHOLOGY.

The University, Hong Kong.

(The original Catalogue appeared in *The Caduceus,* 1935, Vol. 14, No. 2. It is hoped by the publication of supplements to keep the Catalogue up to date, and, when conditions warrant it, a completely revised Catalogue will be published.—*Ed. The Caduceus.*)

New Additions.

1. CIRCULATORY SYSTEM.

11. PERICARDIUM.

11.32.9. Chronic pericarditis, milk spots.
11.32.10. Acute pericarditis.

13. ENDOCARDIUM.

13.3.16. Chronic endocarditis with button-hole stenosed mitral valve.
13.3.17 Acute endocarditis.
13.3.18. Sub-acute bacterial endocarditis.

14. HEART AS A WHOLE.

14.51.1. Primary anaemia—thrush breast.
14.62.3. Fatty degeneration.
14.8.1. Rupture, root of aorta, anterior surface.
14.8.2. Rupture, root of aorta, posterior surface.

15. ARTERIES.

15.37.14. Aortic aneurysm with laminated clot.
15.37.15. Syphilitic aortitis.
15.37.16. Aorta—atheromatous ulcers.

2. RESPIRATORY SYSTEM.

22. LARYNX.

22.35.2. Diphtheria—false membrane.

24. LUNGS.

24.36.18. Chronic caseous tuberculosis and pleurisy.
24.36.19. Miliary tuberculosis.
24.36.20. Miliary tuberculosis and caseous bronchial glands.
24.78.4. Sarcoma—secondary.
24.78.5. Sarcoma—secondary.

34. PLEURA.

25.32.4. Chronic thickened pleura.

3. DIGESTIVE SYSTEM.

31. TEETH.

31.74.4. Odontome.

34. STOMACH.

34.65.7. Perforated pyloric ulcer.
34.79.5. Carcinoma, pyloric portion.

35. INTESTINES.

35.18.1. Pancreatic rest in jejunum.
35.31.1. Acute ulcerative colitis.
35.35.4. Ulceration in typhoid fever.
35.46.6. Amoebiasis, colon.
35.79.4. Adenocarcinoma of colon with intussusception.
35.79.5. Carcinoma rectum, infiltrative growth.
35.84.3. Intussusception with adenocarcinoma of colon.

36. PERITONEUM AND MESENTERY.

36.36.2. Tuberculosis omentum.
36.73.2. Myxoma of mesentery.
36.78.2. Retroperitoneal myxo-fibrosarcoma.

37. LIVER.

37.22.5. Chronic venous congestion.
37.39.10. Multilobular cirrhosis.
37.39.11. Multilobular cirrhosis (early).
37.39.12. Multilobular cirrhosis.
37.46.1. Rabbit liver, coccidiosis.
37.46.2. Malaria.
37.47.3. Clonorchis sinensis in bile ducts.
37.47.4. Clonorchis sinensis in bile ducts.
37.64.3. Intrahepatic stone formation.
37.78.5. Sarcoma—secondary.
37.79.12. Carcinoma.
37.79.13. Primary carcinoma.
37.79.14. Primary carcinoma, hepatoma.
37.79.15. Secondary carcinoma.

38. GALL BLADDER AND BILE DUCTS.

38.56.1. Thickened gall bladder, beri-beri.

39. PANCREAS.

39.79.1. Adenocarcinoma, head of pancreas.

4. HAEMOPOIETIC AND DUCTLESS GLANDS.

41. BONE MARROW.

41.51.4. Primary anaemia.

42. LYMPH NODES.

42.36.6. Tuberculous mesenteric glands.
42.48.1. Lymphatic gland, filariasis.

43. SPLEEN.

43.46.2. Malaria.
43.35.1. Typhoid spleen.

45. THYROID.

45.52.3. Exophthalmic goitre.
45.52.4. Exophthalmic goitre.
45.57.1. Simple goitre.
45.57.2. Simple goitre.
45.74.3. Adenoma.
45.79.3. Adenocarcinoma.

47. ADRENAL.

47.22.2. Congestion of medulla.
47.23.1. Haemorrhage in medulla.

5. UROGENITAL SYSTEM.

51. KIDNEYS.

51.22.3. Chronic venous congestion.
51.22.4. Chronic venous congestion.
51.32.4. Diffuse suppurative nephritis, pyaemia.
51.32.5. Diffuse suppurative nephritis, pyaemia.
51.64.7. Single calculus in upper pole.
51.64.8. Calculus in pelvis with hydronephrosis.
51.67.4. Pyonephrosis.
51.79.8. Hypernephroma.
51.79.9. Hypernephroma.

54-9. MALE GENITALIA.

56.79.7. Epithelioma of penis.
56.79.8. Epithelioma of penis.
56.79.9. Epithelioma of penis.
58.37.2. Gumma of testis.
58.0.2. Testis, teratoma.

6. FEMALE GENITALIA.

62. VAGINA.

62.74.1. Cervical polyp.

63. UTERUS.

63.73.9. Fibroid.
63.73.10. Fibroma.
63.73.11. Fibromyoma.
63.73.12. Fibromyoma.
63.73.13. Fibroid.
63.73.14. Fibroid.
63.74.1. Adenomyoma.
63.78.1. Fibromyoma undergoing sarcomatous change.
63.78.2. Sarcoma.
63.79.4. Adenocarcinoma, body of uterus.
63.79.5. Carcinoma, cervix.
63.79.6. Carcinoma, body of uterus.
63.79.7. Carcinoma, cervix.
63.79.8. Adenocarcinoma, cervix.
63.79.9. Endocervical carcinoma.

64. FALLOPIAN TUBES.

64.77.1. Fimbrial cyst.

65. OVARY AND PAROVARIUM.

65.73.1. Fibroma of ovary.
65.77.5. Lutein cyst.
65.77.6. Pseudomucinous grape-like cystadenoma.
65.77.7. Parovarian cyst.
65.79.5. Papillary cystadenocarcinoma.
65.79.6. Papillary cystadenocarcinoma.
65.0.4. Teratoma of ovary.
65.0.5. Teratoma of ovary.

67. BREAST.

67.74.8. Fibroadenoma.
67.78.7. Spindle-celled sarcoma.
67.79.19. Adenocarcinoma.
67.79.20. Scirrhus carcinoma.
67.79.21. Carcinoma.
67.79.22. Colloid carcinoma.

68. GENERATIVE SYSTEM IN PREGNANCY.

68.62.1. Hydatidiform mole.

7. NERVOUS SYSTEM.

71. MEMBRANES.

71.79.1. Hypernephroma, secondary.

76. NERVES.

76.78.1. Sarcoma.

78. EYE.

78.79.1. Carcinoma.

8. MUSCULO-CUTANEOUS SYSTEM.

81. SKIN.

81.35.2. Skin, leprosy.
81.74.2. Papilloma.

85. FASCIA.

85.73.6. Lipoma.
85.73.7. Lipoma.
85.73.8. Fibro-lipoma of back.
85.73.9. Fibroma of abdominal wall.
85.78.5. Fibrosarcoma of thigh.
85.78.6. Myxosarcoma of buttock.

9. OSSEOUS AND ARTICULAR SYSTEM.

91. BONES OF CRANIUM.

91.37.4. Syphilitic periostitis.
91.67.1. Hydrocephalus.
91.78.1. Sarcoma, secondary.
91.79.1. Hypernephroma, secondary.
91.82.2. Healed fracture, frontal bone.

94. STERNUM.

94.78.1. Sarcoma, secondary.

96. BONES OF UPPER EXTREMITY.

96.73.1. Cancellous osteoma, fourth metacarpus.

98. BONES OF LOWER EXTREMITY.

98.61.1. Fibula, atrophy—sarcoma of leg.
98.78.2. Sarcoma of upper end of tibia.
98.78.3. Sarcoma, secondary—upper end of femur.
98.78.4. Periosteal sarcoma of lower end of femur.
98.78.5. Periosteal sarcoma of lower end of femur.
98.82.3. Fibula, healed fracture.
98.88.1. Foot, congenital abnormality due to pressure by amniotic bands.

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