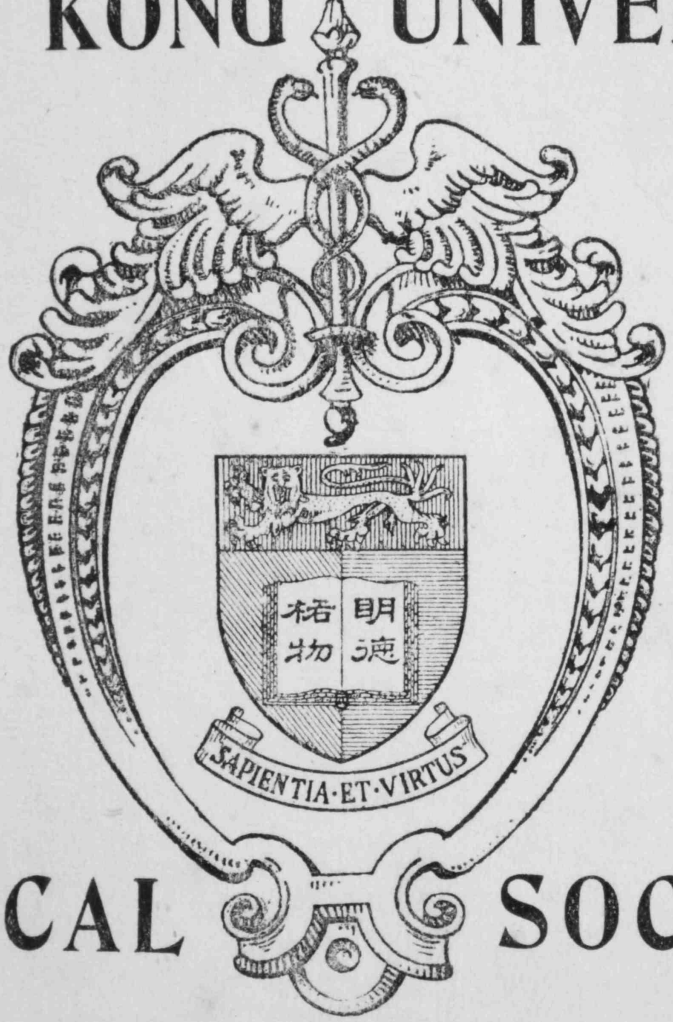
THE CADUCEUS

JOURNAL OF THE HONG KONG UNIVERSITY MEDICAL SOCIETY



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THE CADUCEUS

JOURNAL OF THE HONGKONG UNIVERSITY MEDICAL SOCIETY.

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All medical papers and other scientific contributions intended for the Journal, and all books for review and magazines in exchange, should be addressed to the Editor. "*Caduceus*," Hong Kong University, Hong Kong.

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CLINICAL REPORT OF THE TSAN YUK HOSPITAL AND OF THE MATERNITY BUNGALOW, GOVERNMENT CIVIL HOSPITAL. BEING THE WORK OF THE SCHOOL OF MIDWIFERY OF HONG KONG UNIVERSITY.

May, 1933—April, 1934.

by

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SPECIAL FEATURES OF THE REPORT.

1. Increased admissions to the Maternity Wards. For the year ended:—

30th April.	*Total Admissions.*	*Total Deliveries.*
1927	865	826
1928	1,646	1,576
1929	1,944	1,811
1930	1,778	1,616
1931	1,974	1,841
1932	1,927	1,809
1933	2,018	1,893
1934	2,121	1,985

2. 1934—Mortality rate .25%.

MATERNITY DEPARTMENT.

During the year ended 30th April, 1934 there was a total of 2,121 admissons to the Maternity Wards under our care. Of those 1,985 were delivered, 562 being primiparæ, and 1,423 multiparæ. The total live births numbered 1,855, stillbirths 92, miscarriages 35, and vesicular mole 3. Forty-six children who were born alive, subsequently died in hospital.

The causes of stillbirths were as follows:—

1. Prematurity 21
2. Macerated 44
3. Various causes 27

Under the last heading, as causes of foetal deaths we must include two cases of destructive operations on the fœtus, one case of ruptured uterus, eight cases of placenta prævia, and three cases of prolapse of the cord.

The most important event of the year was the taking over of the Tsan Yuk Hospital by the Government which entailed many changes and some re-organisation. We greatly appreciate the courtesy which has been extended to our Staff by all the Government Officers concerned, and we also should like to place on record our appreciation of the consideration and help which we received from Dr. Tso, Chairman of the Chinese Public Dispensary—in the past.

The University Midwifery School has now the basis of a good clinic which we hope will be capable of much improvement as time goes on. Without the help and support of Dr. Tso this would have been impossible and the University owes much to his friendship.

The following were the principal Obstetrical operations and complications:—

Forceps.

The forceps were applied 65 times giving a frequency of one in 30.5 cases.

The indications were as follows:—

Delayed second stage 52
Prolapsed cord 2
Eclampsia 2
Double uterus 1
Contracted pelvis 2

Placenta prævia	2
Uterine Inertia	2
Fœtal distress	1
Occipito-posterior	1

Breech.

There were 57 cases of breech presentation (14 primiparæ and 43 multiparæ) 36 babies were born alive. The causes of fœtal deaths were as follows:—

Prematurity	5
Macerated	15
Anencephalus	1

Placenta Prœvia.

This condition occurred in 12 patients. All the mothers recovered. The duration of pregnancy was as follows:—

Term	2 cases
37 weeks	1 case
36 weeks	2 cases
33 weeks	2 cases
Below 33 weeks	5 cases

In three of these cases the placenta was situated centrally, in five marginally, and in four laterally. Ten infants were born dead, in two of these cases the infant was macerated. The following treatment was adopted:—

Bipolar Version	7 cases
Internal Version	1 case
Willett's forceps	2 cases
Rupture of membranes	1 case
Extraction of breech	1 case

In one case a slight tear of cervix occurred, which was stitched.

Since our last report, some correspondence appeared in The British Medical Journal on the subject of laceration of the cervix in placenta prævia. We are still of the opinion that it is often extremely easy to tear the cervix in this condition, and even in cases of which the greatest care has been used, lacerations will almost inevitably occur from time to time.

Accidental Hæmorrhage.

There were only two mild cases of accidental hæmorrhage, but in one of them it was necessary to plug the vagina. Both mothers recovered, and both children were born alive.

Prolapse and Presentation of Cord.

This condition occurred 5 times in association with vertex presentation, in one of these the occiput was posterior. Two of the infants were born alive, and all the mothers recovered. In two cases delivery was affected by forceps. In one case, the cord was replaced, in one other case delivery was allowed to occur spontaneously as the cord had ceased to pulsate. In the remaining case the cord prolapsed when the membranes ruptured, the baby was born almost immediately, and before any treatment could be carried out, unfortunately it was not alive.

Cæsarean Section.

There were 6 cases of Cæsarean section, all lower segment operations. All the mothers recovered and 5 of the children were born alive. In the one case of which the fœtus was extracted dead it was found to be anencephalic. In all cases the operation was performed during labour. The indication in 5 cases was pelvic contraction, and in the sixth, Cæsarean section was performed because the uterus had ruptured on a former occasion of pregnancy, and it was feared that the scar would not bear the strain of a long labour. In this case when the abdomen was opened the scar was found to be very thin, and would almost certainly have ruptured.

Eclampsia.

There were 8 cases of eclampsia with one maternal death. All infants were born alive. There has been no change in treatment during the year. The particulars in the fatal case are as follows:—

N.Y. Age 34. Para 1. Admitted on 21-11-33 with the history of having had 2 fits at home, and with marked oedema of legs.

Albumin + +. She had altogether 3 fits before labour began. The baby was born on the day of admission. The patient was morbid for 17 days, H.T. 103° Cough (Lobar pneumonia), difficult respiration, Hæmolytic streptococci recovered from the uterus. Death occurred on the 22nd day. Cause of death:—Sepsis—lobar pneumonia.

Destructive Operation on the Fœtus.

Craniotomy was performed twice, the indication in both cases being contracted pelvis, and dead fœtus.

Morbidity.

Both hospitals were healthy during the year and there were few cases of infectious diseases. The combined morbidity rate of the

two hospitals was 6.15%—a figure which we may consider about an average one.

Mortality.

During the year there were 4 deaths at the Tsan Yuk Hospital out of 1352 deliveries, and one death at the Government Civil Hospital out of 633 deliveries, which gives an average of one in 338 and one in 633 respectively. The combined percentage mortality rate of the two hospitals was about .25%. The causes of the deaths were as follows:—

1. Post-partum hæmorrhage
   Oedema
   Heart disease
2. Ludwig's Angina
   General Septicæmia
3. Post-partum hæmorrhage
4. Puerperal fever
   Lobar Pneumonia
   (Eclampsia)
5. Myocarditis

GYNÆCOLOGICAL REPORT.

During the year there were altogether 333 admissions to the gynæcological wards, 220 operations were performed, and 34 cases received radium treatment for carcinoma. There were 6 deaths.

Hysterectomy.

The subtotal operation was performed 9 times and total hysterectomy once.

Ovariotomy.

Laparotomy was performed on 12 occasions for ovarian and broad ligament cysts. In 2 instances the cyst was the size of a full term pregnancy, in one case the size of 8 months pregnancy, and 3 of the cases were definitely malignant. The largest broad ligament cyst was about the size of 6 months pregnancy.

Extra-uterine Pregnancy.

There were 5 cases of extra-uterine pregnancy, none of which call for special comment.

Prolapse Operation.

There were 6 complete prolapse operations for procidentia.

Mortality.

During the year there were 3 deaths at the Tsan Yuk Hospital and 3 deaths at the Government Civil Hospital.

The causes of deaths were as follows:—

1. Carcinoma of the cervix
   Pulmonary Embolism
2. Malignant ovarian cyst
   Peritonitis
3. Carcinoma of the cervix
4. General peritonitis following the removal of septic broad ligament cyst,
5. Malignant ovarian cyst
6. Typhoid

May 1933 to April 1934.

STATISTICS OF MATERNITY DEPARTMENT.

Table No. I.

Nature and number of cases treated:—	*T.Y.H.*	*G.C.H.*
Total admissions	1447	674
Total deliveries	1352	633
Multiparæ	926	497
Primiparæ	426	136
Presentation:—		
Vertex normal rotation	1236	601
V. 1	850	401
V. 2	359	185
V. 3	13	7
V. 4	14	8
Vertex face to pubes	17	4
Face	1	—
Breech	51	13
Transverse	4	2
Twins	10	8
Brow	—	1
Abortion and Miscarriage	31	4
Hydatidiform mole	2	1
Hæmorrhages:—		
Placenta Prævia	6	6
Post Partum	39	28
Accidental	1	1

Abnormalities :—		
Prolapse of cord	3	2
„ „ hand	2	2
„ „ leg	1	—
Hydramnios	2	2
Eclampsia	6	2
Hydatidiform mole	2	1
Albuminuria :—		
Slight to moderate	96	34
Considerable	9	7
Operations :—	*T.Y.H.*	*G.C.H.*
Suture of perineal lacerations :		
Incomplete	260	121
Multiparæ	75	44
Primiparæ	185	77
Suture of cervical lacerations	1	9
Forceps	42	23
Destructive Operations on Fœtus	1	1
Bipolar version	7	3
Internal version	2	1
External version	2	—
Manual removal of placenta	14	5
Cæsarean Section	3	3
Laparotomy for Ruptured uterus	—	1
Accidental Complications :—		
Enteric fever	1	—
Large cystocele	—	4
Complete prolapse of pregnant uterus	—	1
Constipation	—	1
Cystocele and rectocele	3	1
Dyspnoea	2	1
Pneumonia	1	—
Cerebro-spinal meningitis	1	—
Enlarged spleen	—	1
Diarrhoea and fever	—	1
Goitre	—	1
Puerperal fever and lobar pneumonia	1	—
Bronchitis	5	—
Anaemia	—	1
Malaria	2	—
Advanced pulmonary tuberculosis	2	—
Puerperal sepsis	1	2
Subserous fibroid	1	—
Condyloma	2	1

Morbidity, B.M.A. Standard:	*T.Y.H.*	*G.C.H.*
Average, one in	16.4	15.8
Percentage	6%	6.3%
Mortality:—		
Total	4	1
Average, one in	338	633
Percentage	.30%	.15%
Left Hospital Against Advice	79	4

Table No. II.—*INFANT STATISTICS.*

	T.Y.H.	*G.C.H.*
Total Births	1352	633
Alive	1253	602
Dead:—	99	31
Premature	18	3
Term	18	9
Macerated	30	14
Children born alive who died in hospital	37	9
Abnormalities:—		
Double hare lip & cleft palate	1	1
Gonorrhoeal ophthalmia	—	3
Hypospadias	1	—
Anencephalus	1	1
Foetal ascites	—	1
Single hare lip & cleft palate	1	—
Congental Talipes	1	—
Complications:—		
Umbilical hernia	1	—
Oedema of scrotum and face	1	—
Marked jaundice	1	1
Facial paralysis	—	1
Double white leg	1	—
Equino-varus of both feet	1	—
Ventral hernia	1	—

Table No. III. *Pelvic Presentation.*

Para	Total	Dead Children	Remarks
T.Y.H.			
Primiparæ.	13	Premature 3	One case placenta prævia.
		Macerated 2	One case prolapse of leg.
Multiparæ.	32	Premature 1	
		Macerated 9	Two cases manual removal of placenta.
G.C.H.			
Primiparæ.	1	Term 1	Cæsarean Section, Anencephalic fœtus.
Multiparæ.	11	Premature 1	Placenta prævia.
		Macerated 4	

Table No. IV. *Placenta Praevia.*

Name	Age	Para	Variety	Period of Pregnancy	Presentation	Result to Mother	Result to Child	Remarks
T.Y.H. Y.M.L. (378)	22	3	Lateral	33 weeks	Breech	Recovery	Dead	Os size of ½ dollar. Bipolar version. Uterus and vagina plugged.
M.C. (380)	24	6	Marginal	33 weeks	Breech	Recovery	Dead	Bipolar version. Pituitrin 1 c.c. given.
L.A.H. (698)	19	1	Marginal	27 weeks	Breech	Recovery	Dead	Antepartum haemorrhage. Os fully dilated. Rapid delivery by the breech.
M.S.K. (862)	23	3	Central	22 weeks	Breech	Recovery	Dead	Bipolar version.
H.T.T. (119)	25	4	Lateral	36 weeks	Vertex	Recovery	Alive	Bleeding from vagina 3 weeks before admission. Os admitted 3 fingers. Membranes bulging. Edge of placenta felt about 1½" up from os. Head presenting and almost fixed. Membranes ruptured artificially. Tight binder applied. Scalp clipped with Willett's forceps and traction applied on forceps too.
K.S.K. (424)	38	10	Central	Term	Breech	Recovery	Dead	History of intermittent bleeding from vagina for 30 days. Os admitted 3 fingers. Placenta sitting on top of os. Pulse 120 per min. (weak). Breast infusion c. normal saline Qii digitalin given before turning. Bipolar version performed. ¼c.c. pituitrin given before foot brought down, ¾c.c. pituitrin and ernutin given aften 3rd stage. Slight bleeding. Uterus flabby. Uterus and vagina plugged.

Table No. IV.—*(Continued).* *Placenta Praevia.*

Name	Age	Para	Variety	Presentation	Period of Pregnancy	Result to Mother	Result to Child	Remarks
G.C.H. P.M. (290)	32	4	Lateral	Vertex	36 weeks	Recovery	Alive	Membranes ruptured. Normal delivery.
C.N. (360)	20	2	Marginal	Transverse	24 weeks	Recovery	Dead	Transverse presentation with prolapsed hand. Internal version. Intrauterine douche for macerated foetus.
W.N.H. (366)	23	2	Marginal	Breech	28½ weeks	Recovery	Dead	2 finger os. Bipolar version. Torn cervix stitched. Macerated foetus.
S.A.L. (525)	41	7	Marginal	Vertex P.O.P.	37 weeks	Recovery	Dead	Willett's forceps applied.
M.J.R. (543)	27	2	Central	Transverse	Term	Recovery	Dead	Os 4 fingers. Transverse presentation with extended spine. Bipolar version. Torn cervix stitched.
C.T. (627)	36	1	Lateral	Breech	28½ weeks	Recovery	Dead	Bipolar version. A.P.H. a foot brought down. Placenta came out at the same time as breech. Camphor gr. 1½.

Table No. V.

Accidental Hæmorrhage.

Name	Age	Para	Period	Variety	Presentation	Result to Mother	Result to Child	Remarks
T.Y.H. L.S. (796)	27	4	Term	Revealed	Vertex 1	Recovery.	Alive	Antepartum Haemorrhage, and tight binder applied. Vagina plugged.
G.C.H. L.S.Y. (209)	41	5	Term	Revealed	Vertex 1	Recovery.	Alive	Slight bleeding before birth of child. Cord round neck, large blood clot occupying ⅓ of placenta.

Table No. VI.

Prolapse and Presentation of the Cord.

Name	Age	Para.	Weight of Child	Presentation	Treatment	Result to Mother	Result to Child	Remarks
T.Y.H.								
C.L.F. (558)	28	4	4 lbs. 12 ozs. 5 lbs. 8 ozs.	P.O.P. Vertex	Forceps applied to first baby.	Recovery	Alive	Prolapse of Cord. Twins. Both infants alive.
C.S. (17)	26	4	7 lbs.	Vertex	Forceps applied P.P.H. H.I. Pit 1 c.c. Ernutin 6 c.c. Uterus plugged.	Recovery	Dead	Prolapsed cord with head presenting. Loop of cord appeared at the vulva. Os fully dilated.
L.L.H. (261)	20	1	6 lbs. 4 ozs.	Vertex	Os fully dilated, head very low. Intended to put on forceps, but baby born before forceps ready.	Recovery	Dead	As the membranes ruptured, the cord was prolapsed. Lots of liquor amnii.
G.C.H.								
W.S. (66)	33	4	3½ lbs.	Vertex	Spontaneous delivery.	Recovery	Dead	Prolapse of cord on admission.
C.Y.H. (589)	39	6	7 lbs. 8 ozs.	Vertex	Normal delivery	Recovery	Alive	Proplapsed cord replaced. Live child. Puerperal sepsis.

Table No. VII.

Application of Forceps.

Indications	Number of Case		Result to Mother				Result to Child				Remarks	
			Recovery		Dead		Recovery		Dead			
	T.Y.H.	G.H.C.	T.Y.H.	G.C.H.	T.Y.H.	G.C.H.	T.Y.H.	G.C.H.	T.Y.H.	G.C.H.	T.Y.H.	G.C.H.
Delayed 2nd Stage.	35	17	35	17	—	—	34	15	1	2	Marked oedema of legs and vulva one case.	One case uterine inertia. One case A.P.H. One case sloughing of anterior 1/3 of cervix.
Prolapsed Cord.	2	—	2	—	—	—	1	—	1	—	One case head presenting loop of cord appeared at vulva, os fully dilated. One case twins.	
Eclampsia.	1	1	1	1	—	—	1	1	—	—		
Double uterus.	—	1	—	1	—	—	—	1	—	—		Complete prolapse of 1st uterus c vaginal wall outside vulva obstructing foetus in 2nd uterus higher up. Head not fixed. Os ¾ duilated.
Contracted pelvis.	—	2	—	2	—	—	—	1	—	1		One case induction with stomach tube. One case 1st degree contracted pelvis. Perineum torn. Puerperal sepsis.
Placenta praevia.	1	1	1	1	—	—	1	—	—	1		
Weak uterine contraction.	1	—	1	—	—	—	—	—	1	—		
Weak uterine pain and slow progress.	1	—	1	—	—	—	—	—	1	—	Uterine Inertia.	
Foetal distress.	—	1	—	1	—	—	—	1	—	—		
Occipito posterior. Chin anterior and to the left.	1	—	1	—	—	—	—	—	1	—		

Table No. VIII.

Number of Pregnancy of Patients in whom Forceps were applied.

PARA.	Number of Forceps Cases.		
	T.Y.H.	*G.C.H.*	*Grand Total.*
1	29	14	43
2	—	3	3
3	2	1	3
4 and over	11	5	16
Total	42	23	65

Table No. IX.

Age of Patients in whom Forceps were applied.

PARA.	Number of Forceps Cases.		
	T.Y.H.	*G.C.H.*	*Grand Total.*
17—25	19	9	28
26—30	12	5	17
31—35	5	5	10
36 and over	6	4	10
Total	42	23	65

Table No. X.

Destructure Operation on the Foetus.

Table No. X.

Destructive Operation on Foetus.

Name	Age	Para	Indication.	Operation.	Remarks.
G.C.H. C.Y.Y. (264)	37	4	Brow presentation. 1st. degree contraction. No foetal heart sound heard.	Perforation - extraction-difficult. Manual removal of placenta. Uterus packed.	Induced with stomach tube. Obstructed labour.
T.Y.H. Y.M. (379)	39	7	Os almost fully dilated. Chin anterior and to the left. Head moderately high. Internal conjugate 3″.	Perforation. Head brought out by Winters combined perforator, cephalotribe and craniocast.	Failed forceps. No foetal heart heard. Weight of infant without brain matter- 6¾ lbs.

Table No. XI.

Morbidity, B.M.A. Standard.

	MAY		JUNE		JULY		AUGUST		SEPTEMBER		OCTOBER		NOVEMBER	
	T.Y.H.	G.C.H.	T.Y.H.	G.C.H.	T.Y.H.	G.C.H.	T.Y.H.	G.C.H.	T.Y.H.	G.C.H.	T.Y.H.	G.C.H.	T.Y.H	G.C.H.
Total Deliveries..	91	58	87	51	110	65	102	56	117	49	122	69	120	48
Cases Morbid.....	—	3	3	1	10	2	7	5	4	2	12	3	2	3

	DECEMBER		JANUARY		FEBRUARY		MARCH		APRIL		TOTAL		GRAND TOTAL
	T.Y.H.	G.C.H.	T.Y.H.	G.C.H.	T.Y.H.	G.C.H.	T.Y.H.	G.C.H.	T.Y.H.	G.C.H.	T.Y.H.	G.C.H.	
Total Deliveries..	113	51	154	49	94	37	127	55	115	45	1352	633	1985
Cases Morbid....	7	2	14	5	8	5	6	6	9	3	82	40	122

	T.Y.H.	G.C.H.	GRAND TOTAL
Total Number of Morbid cases	82	40	122
Total Average Morbidity	16.4	15.8	16.3
Total Percentage Morbidity	6%	6.3%	6.1%

Table No. XII.

Comparative Morbidity in Primiparae and Multiparae.

Primiparae	MAY		JUNE		JULY		AUGUST		SEPTEMBER		OCTOBER		NOVEMBER	
	T.Y.H.	G.C.H.	T.Y.H.	G.C.H.	T.Y.H.	G.C.H.	T.Y.H.	G.C.H.	T.Y.H.	G.C.H.	T.Y.H.	G.C.H.	T.Y.H.	G.C.H.
Total Deliveries..	30	13	27	11	25	7	35	10	31	10	2	15	41	9
Cases Morbid.....	—	2	2	—	1	1	5	1	1	—	10	3	2	—

Primiparae	DECEMBER		JANUARY		FEBRUARY		MARCH		APRIL		TOTAL		GRAND TOTAL
	T.Y.H.	G.C.H.	T.Y.H.	G.C.H.	T.Y.H.	G.C.H.	T.Y.H.	G.C.H.	T.Y.H.	G.C.H.	T.Y.H.	G.C.H.	
Total Deliveries..	40	9	46	15	34	12	38	12	27	13	426	136	562
Cases Morbid.....	4	—	6	3	5	2	3	4	6	1	45	17	62

	T.Y.H.	G.C.H.	GRAND TOTAL
Total Average Morbidity .. .. one in ..	9.5	8	9
Total Percentage Morbidity..	10.6%	12.5%	11%

Table No. XII.—*(Continued)*

Comparative Morbidity in Primiparae and Multiparae.

Multiparae	MAY		JUNE		JULY		AUGUST		SEPTEMBER		OCTOBER		NOVEMBER	
	T.Y.H.	G.C.H.	T.Y.H.	G.C.H.	T.Y.H.	G.C.H.	T.Y.H.	G.C.H.	T.Y.H.	G.C.H.	T.Y.H.	G.C.H.	T.Y.H.	G.C.H.
Total Deliveries..	61	45	60	40	85	58	67	46	86	39	70	54	79	39
Cases Morbid.....	—	1	1	1	9	1	2	4	3	2	2	—	—	3

Multiparae	DECEMBER		JANUARY		FEBRUARY		MARCH		APRIL		TOTAL		GRAND TOTAL
	T.Y.H.	G.C.H.	T.Y.H.	G.C.H.	T.Y.H.	G.C.H.	T.Y.H.	G.C.H.	T.Y.H.	G.C.H.	T.Y.H.	G.C.H.	
Total Deliveries..	73	42	108	34	60	25	89	43	88	32	926	497	1423
Cases Morbid.....	3	2	8	2	3	3	3	2	3	2	37	23	60

		T.Y.H.	G.C.H.	GRAND TOTAL
Total Average Morbidity	one in ..	25	21.6	23.7
Total Percentage Morbidity		4%	4.6%	4.2%

Table No. XIII.

Extra-genital causes of Morbidity.

	T.Y.H.	*G.C.H.*
Enteric fever	1	—
Ulceration of urethra	—	1
Constipation	—	1
Cerebro-spinal meningitis	1	—
Enlarged spleen	—	1
Bronchitis	4	—
Anaemia	—	1
Malaria	2	—
Puerperal fever	1	2
Condyloma	1	—
Cracked nipple	1	—

Table No. XIVa.

Caesarean Section.

Name	Age	Para	Nature of Operation	Indication	When Performed	Result to Mother	Result to Child	Remarks	Date
G.C.H. F.F. (303)	35	8	Prophylactic Caesarean Section.	Previous labour ruptured uterus due to face presentation.	During labour.	Alive	Alive	The old tear that was stitched was very thin, and probably would have ruptured if patient was allowed to deliver herself. Normal puerperium.	10-10-33
C.Y.N. (347)	33	1	Low Caesarean Section.	Contraction of pelvis at inlet.	During labour.	Alive	Alive	Old injury to spine in region of 2nd, 3rd, 4th and 5th. Lumbar vertebrae, curvature of spine causing contraction of pelvis. On delivery there was a deep tear of left side of uterus, stitched.	31-10-33
L.K. (519)	24	1	Lower segment Caesarean Section.	Contracted pelvis.	During labour.	Alive	Dead	Leg found prolapsed through ¾ dilated cervix. Remainder tightly packed in pelvic brim, hand could not be passed into uterus. Small tear in post. Vaginal wall. Anencephalic foetus.	12-2-34

Table No. XIVa.— *(Continued)* *Caesarean Section.*

Name.	Age.	Para.	Name of Operation.	Indication.	When performed.	Result to Mother.	Result to Child.	Remarks	Date.
T.Y.H. C.C. (795)	35	5	Lower segment Caesarean section.	Caesarean section. Contracted Pelvis. Previous labours were all forceps cases, with history of very difficult extractions.	During labour.	Recovery	Alive	Extraperitioneal Caesarean Section.	8-9-33
C.T. (21)	33	7	Lower segment Caesarean section.	Previous labours. 4th and 5th forceps (dead babies) 6th craniotomy. Admitted in labour, head mobile, above the brim.	During labour.	Recovery	Alive	Profuse haemorrhage, shock, and almost death of patient on the table. Stimulants H.I. Camphor, digitalin and Adrenalin given. Owing to condition of patient, ligature of tubes not carried out. *Post operative treatment.* 2nd day after operation abdomen distended, turpentives enema given but retained 6 hours later second enema given still retained. H.I. Pit ½ c.c. ½ hourly up to 1 c.c. Bowel still not opened. Next morning castor oil 1½ z given. 3′ hours later. C and S (Calomel gr. ½ half hourly up to gr. iii given and mist Alba zii given ½ hourly after last dose of calomel) Bowels well opened and abdominal distension disappeared. Discharge from the wound. "Staphylococcus Albus."	4-1-34
W.C. (219)	19	1	Lower segment	In labour for 3 days. Head still not fixed. 2nd degree of contracted pelvis.	During labour.	Recovery	Alive	*Post operative treatment.* Patient had bad cough. Mist Expect Strin zss 4 hourly. Pulse not good. Digitalin gr. 1/100 4 hourly. Antiphlogistine applied to chest, front and back. Antistrep serum 20 c.c. Breast infusion of normal saline Ojss. Lochia offensive and purulent. Abdominal wound septic Vaccine given, wound dressed with Dakins solu. *P.V. findings*—uterus retroverted.	17-2-34

Table No. XIVb.

Rupture of Uterus.

Name	Age	Para	Nature of Operation	Indication	When performed	Result Mother	Result Child	Remarks	Date
G.C.H. C.C. (504)	30	4	Laparotomy for Ruptured uterus	Ruptured uterus	During labour	Alive	Dead	Uterus ruptured at lower segment. Child in abdomen, born dead. Membranes intact. Old tear of cervix extending up into abdomen. Very weak labour pains, probably rupture was gradual and resulted in an extension of old tear.	6-2-34

Table No. XV.

Eclampsia.

Name	Admission	Age	Para	Condition on Admission	Urine	Number of Fits			Treatment	Result to Mother	Result to Child	Remarks	Period of Pregnancy
						Before Labour	After Labour	During Labour					
T.Y.H. A.Y.M. (725)	19-8-33	27	1	Oedema of legs.	++	—	—	33	H.I. Morphia gr. 1/6. H.I. Atropine gr. 1/150. Rectal wash out. Castor oil zii by mouth. Rectal ether 1½oz. and Paradehyde 1 dr. Mist Alba ziii by mouth. Morphia gr. 1/6. Rectal and Stomach wash out. Rectal ether 1½ oz. and Paraldehyde 1 dr. Rectal ether repeated (not retained). Rectal wash out. H.I. Hyoesine Hydrobrom gr. 1/100. Rectal ether. Breast Infusion Sod. Bicarb oii. Rectal saline 1½ pts. Rectal wash out. H.I. Morphia gr. 1/6. Rectal wash out. Mag. Sulph. Ozvi per rectum. H.I. Hyoscine Hydrobrom gr. 1/200. Breast infusion. Sod. Bicarb oii. Rectal ether 1 oz. and 1 dr. paraldehyde.	Recovery	Alive	Post partum Eclampsia. 22-8-33. Fits very frequent. Clonic stage was prolonged while comatose was comparatively short.	Term

Table No. XV.—*(Continued)*

Eclampsia.

Name	Admission	Age	Para.	Condition on Admission	Urine	Number of Fits			Treatment	Result to Mother	Result to Child	Remarks	Period of Pregnancy
						Before Labour	After Labour	During Labour					
T.Y.H. Y.K. (934)	8-10-33	24	1	Oedema of legs.	†††	—	—	6	H.I. Morphia gr. 1/6. Atropine gr. 1/150. Rectal wash out with Sod. Bicarb. Mist Alba oziv per rectum. Mist Alba ziv by mouth. H.I. Morphia gr. 1/6. H.I. Atropine gr. 1/150. H.I. Hyoscine gr. 1/200. Rectal wash out. Rectal ether 1 oz. Paraldehyde 1dr. Stomach wash out.	Recovery	Dead	1st fit started 20 mins. after delivery.	37 weeks
N.Y. (1126)	21-11-33	24	1	Oedema of legs.	††	3	—	—	Rectal and stomach wash out. Morphia gr. ¼. Mist Alba ziii by mouth. but not retained. Mist Alba repeated.	Dead	Alive	Had 2 fits at home. 1 fit before labour. Infant born with blue asphyxia. Artificial respiration performed. Died on 23-11-33 due to cerebral haemorrhage.	Term.
T.C. (1118)	27-11-33	24	1	Oedema of legs.	†††	6	1	—	Morphia gr. ¼. Atropin gr. 1/200. Rectal and stomach wash out. Castor oil zii by mouth. Digitalin gr. 1/100. Breast infusion. Sod. Bicard 2Q. Ether 1 oz. and 2 dr. paraldehyde per rectum.	Recovery	Alive	Os ½ dilated. Infant born. Mist Alba ziii by mouth.	Term.
W.S. (1174)	5-12-33	19	1	Slight oedema of legs.	†	2	—	—	H.I. Camphor 1½ gr. H.I. Digitalin. Sod. Bicarb QI. Submammary infusion.	Recovery	Alive	Low forceps applied. Protuse bleeding from vagina after delivery Placenta expressed spontaneously. H.I. Ernutin ½ c.c. Bleeding gradually stopped.	Term.

Table No. XV.—*(Continued)* *Eclampsia.*

Name	Admission	Age	Para	Condition on Admission	Urine	Number of Fits			Treatment	Result to Mother	Result to Child	Remarks	Period of Pregnancy
						Before Labour	During Labour	After Labour					
T.Y.H.													
L.C.P. (463)	17-4-34	20	1	Oedema of legs	†††	2	—	3	Enema and rectal wash out e.g. Sod. Bicarb Mist Alba P.R. ziv and P.O. ziii. H.I. Morphia gr. 1/6. Atropine gr. 1/150. ether 1 oz., paraldehyde 2 dr. PR. Pulv. Jalap Co. grs. 60. Mist Alba ziii by mouth. Enema, Calomel ½ gr. ½ hourly up to gr. 5 followed by Salt and enema. After delivery. H.I. Morphia gr. 1/6. Atropine. gr. 1/150. Hyoscine Hydrobrom gr. 1/200. Mist Alba ziv by mouth.	Dead	Alive	Under general anaesthesia stomach tube inserted (at the time of insertion os was taking up and admitted 2 fingers.) Membranes ruptured during tube insertion. Tight binder applied. Baby born without any difficulty.	Term.
L.Y. (61)	1-6-33	32	1	—	†	1	—	1	Rectal wash out after 2nd fit. Mist B.B. zv given with stomach tube. Mist B.B. ozv given per rectum.	Recovery	Alive	Forceps delivery.	Term.
W.S. (88)	17-6-33	32	5	In a comatose condition	†††	1	—	—	Mist Bromidia zp. H.I. Morphia gr. ½. H.I. Hyoscine gr. 1/200. Mist B.B. 4 oz.	Dead	Alive	Had 1 fit before admission.	Term.

Table No. XVI. *Operative Cases showing Morbidity.*

Name of Operation	Number		No. of Morbidity		Percentage		Average		Remarks
	T.Y.H.	G.C.H	T.Y.H.	G.C.H.	T.Y.H.	G.C.H.	T.Y.H.	G.C.H.	
Forceps.	42	23	11	6	26.2%	26.1%	1 in 3.8	1 in 3.8	T.Y.H. 10 cases delayed 2nd stage. 1 case weak uterine contraction. G.C.H. 1 case complete prolapse of 1st uterus. (Case of double uterus). 1 case placenta praevia. 1 case admission 3 days in labour with head on perineum. Pulse 160. Temp. 100°.
Perineal lacerations.	260	121	30	11	11.5%	9%	1 in 8.6	1 in 11	—
Cervical lacerations.	1	9	—	3	—	33.3%	—	1 in 3	—
Manual removal of placenta.	11	5	—	2	—	40%	—	1 in 2.5	1 case baby born 31 hours before admission, placenta retained for 2 hours admitted with placenta outside vulva in decomposed condition. 1 case extraction difficult, uterus packed.
Induction of labour.	1	2	—	1	—	50%	—	1 in 2	1st degree contraction.
Bipolar version.	7	3	—	2	—	66.6%	—	1 in 1.5	Both cases placenta praevia.
Internal version.	2	1	—	1	—	100%	—	1 in 1	Placenta praevia transverse presentation with prolapsed hand.
Eclampsia.	6	2	1	—	16.6%	—	1 in 6	—	—
Destructive operation on foetus.	1	1	—	1	—	100%	—	1 in 1	1st degree contraction, forceps failed, perforation.

Table No. XVII.

Duration of Stay in Hospital of Morbid Cases.

	T.Y.H.		G.C.H.
Less than 10 days	43 cases including	— death	31 cases.
10 to 19 days	32 ,,	—	8 ,,
20 to 29 days	3 ,,	1	1 ,,
Over 29 days	4 ,,	—	—
Total :—	82	1	40

Table No. XVIII.

Duration of Temperature.

	T.Y.H.		G.C.H.
Less than 5 days	70 cases including	— death	38 cases.
5 to 9 days	7 ,,	—	2 ,,
10 to 19 days	4 ,,	1	—
Over 19 days	1 ,,	—	—
Total :—	82	1	40

Table No. XIX.

Highest Temperature Charted.

	T.Y.H.		G.C.H.
100° to 100.9°	23 cases including	— death	9 cases.
101° to 101.9°	23 ,,	—	18 ,,
102° to 102.9°	19 ,,	—	7 ,,
103° to 103.9°	10 ,,	1	3 ,,
104° and over	7 ,,	—	3 ,,
Total :—	82	1	40

Table No. XX. *Mortality.*

Name	Age	Para	Admitted	Delivered	Died	Cause of Death	Remarks
T.Y.H. C.H. (402)	36	4	14-5-33	15-5-33	15-5-33	Haemorrhage. Oedema of legs. Cardiac Disease.	Been ill for 14 days before admission. Haemorrhage started in dark blood clots. Respiration too fast and shallow. Pulse weak and rapid. Pit. 1 c.c. and Ergotin given. Inj. of Camphor and digitalin gr. 1/100. Uterus douched and packed with gauze. Breast saline 1½ pints. digitalin gr. 1/100 4 hourly. Pulse did not improve. Pt. died at 5.30 p.m.
M.L. (520)	18	1	23-6-33	23-6-33	25-6-33	Ludwigs Angina Septicaemia.	Swelling of neck for 2 days. Swelling hard and very tender. Difficulty in swallowing food. 25-6-33. Sore throat, tongue swollen, trismus very marked. Swelling incised in 8.30 a.m. Patient died at 8.45 a.m.
L.F. (612)	28	7	19-7-33	19-7-33	19-7-33	Post partum haemorrhage. Cardiac failure.	Transverse presentation. Bipolar version when os was ½ dilated. ¼ c.c. pituitrin given when leg was out. P.P.H. Uterus and vagina plugged. 1 c.c. pituitrin and 1 c.c. Ernutin given. 2 pints sub-mammary saline. Camphor 1½ gr. given twice. Digitalin gr. 1/100. Morphia gr. ⅙. Strychnine gr. 1/64 History of P.P.H. in all previous pregnancies. Patient died at 6.30 p.m.
N.Y. (1126)	24	1	21-11-33	21-11-33	13-12-33	Puerperal Fever Labar Pneumonia.	Eclampsia. 7-12-33. Patient developed oedema and weakness of lower limbs. Urine smoky colour numerous granular casts and pus cells few blood cells. 8-12-33. Complains of cough. Rales heard over base of right lung. 10-12-33. Respiration much distressed. Injection digitalin 6 hourly. 11-12-33 Injection of digitalin 4 hourly. Patient died at 12.30 a.m. 13-12-33.

Table No. XX.—*(Continued)* *Mortality.*

Name	Age	Para	Admitted	Delivered	Died	Cause of Death	Remarks
G.C.H. A.T. (389)	30	1	26-11-33	26-11-33	1-12-33	Myocarditis.	Albuminuria. Normal puerperium. Died on the 6th day of myocarditis heart failure.

Table No. XXI.

Induction of Labour with Stomach Tube.

	T.Y.H.	*G.C.H.*
Total number of cases	1	2
Number of cases successful	1	1

Table No. XXII.

Duration of Stay in Hospital.

	T.Y.H.	*G.C.H.*
Total number	1352	633
Less than 3 days	3	1
From 3 to 5 days	9	—
6 to 8 days	1183	581
9 or more days	157	51

Table No. I.

May 1933 to April 1934.

STATISTICS OF GYNÆCOLOGICAL DEPARTMENT.

	T.Y.H.	*G.C.H.*
Number of admissions	202	131
Number of operations	120	100

Table No. II.

Nature and Number of Operations.

	T.Y.H.	*G.C.H.*
Vulva :—		
Hypertrophy of clitoris	—	1
Excision of clitoris	1	—
Bartholin cyst	1	2
Lipoma of left labium majorum	—	1
Perineum :—		
Perinæorrhaphy	1	1
Colpo-perinæorrhaphy	—	1
Urethra :—		
Ulceration of urethra	1	—
Caruncle, excision of	1	—
Vagina :—		
Vesico-vaginal fistula	—	4
Anterior colporrhaphy	1	—
Senile vaginitis	—	1

Uterus :—		
Curettage, insertion of radium	1	—
Curettage, insertion of pessary	4	1
Dilatation and curettage	54	30
Prolapse	11	10
Ventral-suspension (abdominal)	4	4
Ventral-suspension (vaginal)	3	—
Hysterectomy (subtotal)	3	6
Hysterectomy (total)	—	1
Abortion (curettage for)	3	—
Myomectomy	8	4
Cervix :—		
Erosion of cervix	—	2
Amputation	1	—
	T.Y.H.	*G.C.H.*
Polypus, removal of	2	5
Fixed retroversion & polypus	—	1
Tubes and Ovaries :—		
Ovariotomy	4	8
Salpingectomy	2	1
Salpingostomy	1	5
Luteal cyst	—	2
Ovarian dermoid	—	1
Extra uterine gestation	5	—
Broad ligament cyst	1	3
Hæmorrhagic ovarian cyst	1	—
Par-ovarian cyst	—	1
Miscellaneous :—		
Exploratory Laparotomy	2	3
Retroverted gravid uterus with ascites	1	—
Breast abscess	2	—
Vesicular Mole, curettage for	1	—
Retroperitoneal malignant tumour	—	1
Total :—	120	100

Table No. III.

Nature and Number of Cases Treated Without Operation.

	T.Y.H.	*G.C.H.*
Refused operation	9	3
No operation indicated	6	—
Pregnancy with vomiting	2	1

Pregnancy with hæmorrhage	1	—
Pregnancy with procidentia	1	—
Pregnancy with gastritis	1	—
Normal pregnancy	—	1
Acute mastitis	1	1
Vulvitis	1	—
Subinvolution	—	1
Carcinoma of cervix (radium treatment)	21	13
Threatened abortion	1	1
Incomplete abortion	3	1
Incomplete miscarriage	—	2
Parametritis	1	—
Adeno-carcinoma	1	—
Puerperal Sepsis	1	1
Retroversion	5	2
Salpingitis	—	3
Retroversion (pessary)	1	—
Gonorrhœa	4	—
Cystitis	1	1
Tampon treatment	17	—
Ulceration of labia	1	—
Leucorrhœa	1	—
Dysmenorrhœa and constipation	1	—
Menorrhagia	1	—
Total :—	82	31
Mortality	3	3

Table No. IV.

Hysterectomy.

No.	Name	Age	Date	Disease	Operation	Result	Remarks
	T.Y.H.						
115	Y.Y.K.	28	20-7-33	Uterine tumour.	Subtotal hysterectomy	Recovery.	Tumour size of 4 months pregnancy Adhesions of the uterus anchoring it to the pelvis. One ovary resected, the other removed.
167	L.C.	45	21-9-33	Fibro-myoma.	Subtotal hysterectomy.	Recovery.	Uterus enlarged to size of six months pregnancy. Left cystic ovary was removed.
258	L.S.	45	5-4-34	Fibroid.	Subtotal hysterectomy	Recovery.	Uterus enlarged and adherent to rectum. Adhesions in Douglas pouch due to chocolate cyst. Oozing from Douglas pouch, gauze inserted, the end drawn out through vagina.
	G.C.H.						
214	L.T.	21	11-7-33	Vesico - vaginal and recto-vaginal fistula.	Subtotal hysterectomy. Ovaries not removed.	Recovery.	Recto-vaginal fistula 1½″ from vaginal outlet. Fistula size of 20 cent piece Opening was very much scarred. Another opening into the bladder admitting 2 fingers easily.
243	W.B.H.	25	19-9-33	Fibroid of uterus.	Subtotal hysterectomy.	Recovery	Uterus enlarged to size of foetal head.
264	K.N.	39	14-11-33	Haematometra and Haematosalpinx.	Subtotal hysterectomy.	Recovery	Uterus enlarged to size of 4 months pregnancy, and partly leaking. Free blood in peritoneal cavity. The right tube size of hen's egg distended probably with blood.

Table No. IV.—*(Continued)*

Hysterectomy.

No.	Name	Age	Date	Disease	Operation	Result	Remarks
241	**G.C.H.** L.Y.	35	29-8-33	Vesico-vaginal fistula.	Subtotal hysterectomy. Ovaries left behind.	Recovery.	Prolapsed bladder, mucous membrane seen through vagina found to be trigone of bladder and the left ureteric orifice could be seen. Cervix stenosed. Patient menstruating through bladder.
288	T.C.	46	20-2-34	Cyst-adenoma.	Total hysterectomy.	Recovery.	Irregular lumps protruding from cervical canal size of pigeon's egg. Uterus retroverted and enlarged size of 2 fists and elongated. Vaginal attachments were freed from below. Mackenrodts ligaments clamped and divided. Abdomen was then opened. broad ligaments divided left ovary removed.
295	K.S.	52	20-2-34	Ovarian tumour.	Subtotal hysterectomy for myoma.	Recovery	Omentum was adherent to tumour at several places.
20	L.Y.	36	17-4-34	Fibroid. Typhoid.	Subtotal hysterectomy.	Dead.	Lump size of 7 months pregnancy. 19-4-34. Temperature rose 21-4-34. Slight rales at bases of lungs 23-4-34. Pus cells found in urine, B. Coli found in smear. Temperature keeping up till 24-4-34 when patient became delirious at 9 a.m. and died at 12.50 p.m.

Table No. V.

Ovariotomy.

No.	Name	Age	Date	Disease	Operation	Result	Remarks
91	**T.Y.H.** H.M.	36	15-6-33	Right broad ligament cyst.	Cyst was removed with its capsule.	Recovery.	Cystic swelling was tapped. Right ovary contained a small chocolate cyst. Left overy was hard in consistency. Fluid removed 180 ozs. Weight of sac and pedicle = 1 lb.
107	L.H.	27	17-7-33	Multiple ovarian cysts.	Ovariotomy.	Recovery	Uterus retroverted resting on the cysts. Omentum adherent to fundus and completely roofing over the cyst. Adhesions separated, both ovaries size of hen's eggs removed. 4 other serious cysts removed. others ruptured. Tubes also removed.
131	S.K.M.	35	3-8-33	Right tubal abortion. Left haemorrhagic ovarian cyst.	Right pregnant tube resected with partial excision of right ovary. Left ovary and tube completely removed.	Recovery.	Free dark blood in peritoneal cavity. Blood clot size of fist lying in Douglas' Pouch. Haemorrhage coming from the right tubal abortion. Blood clot evacuated. Left ovary was enlarged to size of a hen's egg, darkish in colour.
226	W.Y.W.	33	8-2-34	Right ovarian syst.	Cyst removed. Left ovary cystic resected. Base left behind. Uterus suspended.	Recovery	Small cyst size of a tennis ball found on the right side.

Table No. V.—*(Continued 1)*

Ovariotomy.

No.	Name	Age	Date	Disease	Operation	Result	Remarks
237	**T.Y.H.** Y.S.	43	8-3-34	Left ovarian cyst. (malignant)	Ovariotomy.	Dead	Peritoneum adherent to tumour size of a melon. Adhesions freed, tumour found to be semi-solid. Uterus and both vaginal fornices were fixed. Peritoneum thickened extendng upwards to liver. Tumour removed. Radium inserted to fundus of uterus and vagina.
243	H.L	36	15-3-34	Left ovarian cyst.	Ovariotomy.	Recovery.	Cyst size of ping pong ball resected. Right ovary cystic partly excised. Right tube also resected. Uterus suspended.
208	**G.C.H.** T.C.H.	32	13-6-33	Suppurative Dermoid cyst.	Ovariotomy.	Recovery.	Tumour size of football adherent to transverse colon on top. Tumour evidently growing from the pelvis lifting broad ligament on right side. Trying to shell the tumour, cyst was ruptured into, foul offensive purulent fluid with hair discharged into the wound about 2 to 3 pints. Sac was shelled out, raw surface packed with gauze and sewn up and drained with rubber tubing.

Table No. V.—*(Continued 2)*

Ovariotomy.

No.	Name	Age	Date	Disease	Operation	Result	Remarks
220	**G.C.H.** K.H.	47	11-7-33	Malignant ovarian tumour.	Ovariotomy.	Recovery.	Large tumour size of full term pregnancy growing from root of mesentery displacing intestines to the right. Transverse colon, was also lifted up. No secondary deposit in liver. Straw colour free fluid. Condition was inoperable. No attempt made to remove the tumour.
223	C.Y.	47	18-7-33	Broad ligament myoma.	Cyst shelled out. Excess of sac excised.	Recovery.	Broad ligament cyst size of a foetal head. Both ovaries identified.
224	Y.A.C.	35	25-7-33	Broad ligament cyst.	Cyst shelled out. the bed obliterated.	Recovery.	Uterus displaced to the right by a cystic tumour size of foetal head, lying between layers of left broad ligament.
242	L.W.	36	5-9-33	Ovarian dermoid	Ovariotomy.	Recovery.	Cyst size of full term pregnancy adherent to all peritoneal surfaces especially in front. Unsuccessful in freeing adhesions. Pedicle clamped and excised, tumour delivered by lower pole. Whole of omentum, transverse colon sigmoid adherent to cyst wall. Adhesions separated, and ligatured and cyst removed. Weight = 6 lbs.

Table No. V.—*(Continued 3)*

Ovariotomy.

No.	Name	Age	Date	Disease	Operation	Result	Remarks
252	**G.C.H.** Y.Y.	28	25-9-33	Ovarian cyst.	Ovariotomy.	Recovery.	Cyst size of 8 months pregnancy. Pedicle clamped. Cyst removed. Unilocular cyst containing clear fluid. Weight of cyst = 9½ lbs.
259	I.C.T.	33	31-10-33	Ovarian cyst.	Ovariotomy.	Recovery.	Slight amount of free fluid in abdomen. Little adhesion above. Cyst tapped, pedicle clamped, ligatured, sewn over, cut. Uterus suspended. Amount of fluid in cyst = 18 pints.
286	L.M.	32	30-1-34	Retroverted uterus deflected to the left side.	Left ovariotomy. Removal of par ovarian cyst right side. Ventral suspension.	Recovery.	Parovarian cyst size of a tennis ball. Left ovarian cyst size of a billiard ball shelled out.
294	B.S.	69	27-2-34	Malignant ovarian cyst.	Ovariotomy.	Dead.	Large quantity of free fluid escaped. Papillomatous mass on each side surrounding uterus. The left one size of cricket ball adherent to rectum. Both tumours shelled out as far as possible. Radium inserted. Patient died at 10 p.m. on 17-3-34.

Table No. V—*(Continued 4)*

Ovariotomy.

No.	Name	Age	Date	Disease	Operation	Result	Remarks
2	**G.C.H.** C.H.	21	20-3-34	Ovarian tumour.	Ovariotomy.	Recovery.	Right ovarian cyst size of small football excised. Left cystic ovary size of hen's egg partially excised. Uterus suspended.
10	W.S.	26	27-3-34	Ovarian cyst.	Ovariotomy.	Recovery.	Broad ligament cyst on left side size of a cricket ball was ruptured while attempting to shell it out, sac excised. Ovarian tumour of right side size of a ping pong ball also excised.
23	L.C.	27	24-4-34	Broad ligament cyst.	Ovariotomy.	Recovery.	Cyst size of 6 months pregnancy shelled out. Capsule resected and sewn over. Uterus adherent to Douglas' Pouch and deflected to the left.
24	H.Y.	56	24-4-34	Ovarian cyst. (Malignant).	Ovariotomy.	Recovery.	Omentum adherent all over the anterior and lateral surfaces of the tumour. Sigmoid and transverse colon also adherent. Most malignant part of the tumour was resected. Purse string put round edges of tumour sewn round to abdominal wall. Gauze packing in cavity and peritoneal cavity drained by rubbing tubing.

Table No. VI. *Operations on Uterus Tubes and Ovaries.*

No.	Name	Age	Date	Disease	Operation	Result	Remarks
86	**T.Y.H.** W.Y.	29	8-6-33	Retroverted uterus and hydro-salpinx.	Rt. salpingectomy. Resected of right ovary. Ventral suspension.	Recovery.	Uterus and tubes were bound down by adhesion. Left tube freed. Right tube enlarged and contained fluid. Right chocolate ovarian cyst.
148	L.W.C.	22	29-8-33	Fixed retroversion. Thickened tubes.	Laparotomy. Left salpingostomy. Suspension of uterus.	Recovery.	Uterus adherent to Douglas' Pouch. Omentum attached to anterior surface. Ovaries small and cystic. Cystic areas punctured and sewn over. Right tube closed.
138	C.K.	32	17-8-33	Retroversion left tube thickened.	Right cystic ovary partially excised. Right tube thickened but patent. Left tube patent. Uterus suspended.	Recovery.	Uterus adherent to pouch of Douglas. adhesion separated.
147	F.Y.M.	23	29-8-33	Retroverted uterus not easily replaced.	Laparotomy. Uterus suspended.	Recovery.	Uterus adherent to Douglas' Pouch. Adhesions freed. Fimbriated end found patent after freeing adhesions.
188	T.S.C.	26	9-11-33	Uterus retroverted and not freely movable.	Ventral suspension and partial resection of right ovary.	Recovery.	2-11-33. Blowing of the tubes. Right tube quite patent. Left tube sound not so clear also patent.
211	C.Y.	26	3-1-34	Retroversion.	Adhesions freed. Uterus suspended.	Recovery.	Uterus retroverted and drawn to the right.
251	N.K.S.	23	27-3-34	Fixed retroversion.	Right ovary cystic. Right tube and ovary removed.	Recovery.	Uterus retroverted and drawn to the left by a swelling size of ping pong ball. Thickening in posterior fornix. Cervix eroded.

Table No. VI.—*(Continued 1)* *Operations on Uterus Tubes and Ovaries.*

No.	Name	Age	Date	Disease	Operation	Result	Remarks
204	G.C.H. H.L.	32	30-5-33	Retroversion.	Left cystic ovary dissected, uterus ventrally suspended.	Recovery.	Uterus adherent to Douglas' Pouch by bands of adhesions. Adhesions separated.
246	H.K.H.	41	12-9-33	Fixed retroversion.	Salpingostomy. Uterus suspended.	Recovery.	Uterus adherent to Douglas' Pouch, adhesions separated. Right tube closed and anchored in Pouch of Douglas, tube separated. Right cystic ovary was ruptured and sewn over.
254	F.Y.L.	26	17-10-33	Fixed retroversion.	Uterus ventrally suspended.	Recovery.	Light adhesions cleared
270	A.S.H.	22	28-11-33	Movable retroversion.	Uterus ventrally suspended.	Recovery.	Patient habitually constipated and subjected to flatulence.
272	M.W.C.	23	5-12-33	Fixed retroverted uterus.	Excision of right cystic ovary. Ventral suspension.	Recovery.	Ruptured luteal cyst in right ovary.
277	T.S.C.	28	18-12-33	Retroverted uterus and Salpingitis.	Right hydrosalpinx excised, uterus suspended, partially fixed.	Recovery.	Uterus adherent to Douglas'Pouch. Hydrosalpinx size of a goose egg.
281	L.C.H.	32	9-1-34	Fixed retroverted uterus.	Uterus suspended.	Recovery.	Left and right Fallopian tubes found blocked.
282	T.M.	40	16-1-34	Fixed Retroversion and Salpingitis.	Left tubes and ovarian cyst dissected. Uterus suspended.	Recovery	Left cystic ovary size of a pigeon's egg was ruptured.

Table No. VI.—*(Continued 2)* *Operation on Uterus, Tubes and Ovaries.*

No.	Name	Age	Date	Disease	Operation	Result	Remarks
284	**G.C.H.** C.W.H.	25	16-1-34	Subacute salpingitis.	Right tube with ovary desected.	Recovery	Adhesion at the back of cervix. Adhesion separated. Left tube found patent.
12	L.T.	31	6-3-34	Chronic Salpingitis.	Excision of left tube. Partial excision of left ovary. Right tube also excised. Right ovary cut into and a small cyst shelled out. Uterus suspended.	Recovery	Uterus adherent to Pouch of Douglas. Both tubes thickened size of a thumb, and both ovaries cystic.
15	C.S.	30	10-4-34	Chronic Salpingitis.	Omentum separated bleeding points ligatured. Right corpus luteal cyst excised. Right tube found blocked. Left tube and ovary matted down unable to separate. Uterus suspended.	Recovery	Uterus adherent to Pouch of Douglas and roofed over by adherent omentum.

Table No. VII.

Extrauterine Pregnancy.

No.	Name	Age	Date	Disease	Operation	Result	Remarks
78	T.Y.H. Y.M.	30	1-6-33	Extra-uterine pregnancy.	Laparotomy for extra-uterine pregnancy.	Recovery.	Douglas' Pouch filled with a large blood clot. Pregnancy situated in outer ⅓ of the right tube. Right tube removed together with ovary. Uterus suspended.
112	W.M.C.	22	13-7-33	Extra-uterine pregnancy.	Laparotomy. Excision of right tube partial excision of ovary. Uterus suspended.	Recovery.	Bluish lump size of American orange on right fornix. Lump was pregnant right tube.
122	C.H.	27	27-7-33	Left ovarian pregnancy. Ruptured extra-uterine pregnancy of left ovary.	Midline incision blood clot shelled out. Uterus not suspended.	Recovery	Free blood in peritoneal cavity, bleeding from ruptured extra-uterine pregnancy. Haemorrhage from bed stopped by square stitches.
162	L.S.M.	31	21-9-33	Left sided extra-uterine pregnancy.	Ruptured during operation was removed.	Recovery	Extra-uterine pregnancy size of a sheep's heart.
255	W.H.	35	29-3-34	Extra-uterine pregnancy.	Laparotomy. Broad ligament on right side was cut. clot turned out. Ovarian cyst removed.	Recovery.	The tube ruptured into the broad ligament resulting in formation of a tumour extending up to level of umbilicus. On left side small ovarian tumour size of a tennis ball.

Table No. VIII. *Prolapse.*

No.	Name	Date	Age	Disease	Operation	Result
79	**T.Y.H.** T.M.	1-6-33	65	Complete prolapse and ulceration of cervix.	Prolapse operation. Anterior-Colporrhaphy; Shortening of ligaments; Amputation of cervix; Perinaeorrhaphy.	Recovery.
134	C.F.	5-8-33	40	Procidentia.	Complete prolapse operation with ventral vaginal suspension.	Recovery.
139	W.W.	17-8-33	24	Cystocele and retroversion.	Modified prolapse operation performed. Shortening of Mackenrodt's ligaments and perinaeorrhaphy.	Recovery.
168	L.A.C.	28-9-33	44	Complete prolapse of uterus.	Complete prolapse operation. Anterior-Colporrhaphy; Amputation of cervix; Shortening of Mackenrodt's ligaments; Vaginal suspension and perinaeorrhaphy.	Recovery.
169	I.T.	28-9-33	54	2nd degree prolapse.	Prolapse operation. Amputation of cervix; Anterior-Colporrhaphy; Ventral suspension. Abdomen opened from above for purpose of freeing adhesions.	Recovery.
184	C.Y.M.	26-10-33	45	2nd degree prolapsed	Amputation of cervix. Radium therapy.	Recovery.
192	L.H.	9-11-33	38	Complete prolapse.	Cervix dilated, cuff reflected, bladder pushed up, peritoneum opened, fundus pushed through opening, both tubes separated. Peritoneum stitched to back of fundus of uterus. Cornua of uterus stitched to tissues most adherent to pelvic arch. Amputation of cervix, repair of anterior-vaginal wall. Perinaeorrhaphy.	Recovery.
235	H.K.	8-3-34	25	Procidentia uterus retroverted and prolapsed. Cervix hypertrophied.	Complete prolapse operation and Colpo-perinaeorrhaphy.	Recovery.
218	F.C.	25-1-34	45	Prolapse of uterus 2nd degree.	Anterior Colphorrhaphy. Vaginal suspension of uterus. Perinaeorrhaphy. Schroeder's amputation of cervix.	Recovery.

Table No. VIII.—*(Continued)* *Prolapse.*

No.	Name	Date	Age	Disease	Operation	Result
249	**T.Y.H.** C.Y.	22-3-34	58	Complete prolapse. Uterus retroverted and prolapsed.	Amputation of cervix. Colporrhaphy. Vaginal suspension.	Recovery.
250	N.W.F.	29-3-34	24	Prolapse 2nd degree.	Amputation of cervix. Schroder's operation. Anterior Colporrhaphy. Vaginal suspension.	Recovery.
199	**G.C.H.** K.S.	15-5-33	34	Prolapse. 2nd degree.	Complete prolapse operation. Colpo-perinaeorrhaphy.	Recovery.
210	K.A.N.	13-6-33	25	Complete prolapse.	Vaginal suspension, Shortening of Mac's ligaments. U.S. ligaments, Amputation of cervix, Anterior Colporrhaphy and perinaeorrhaphy.	Recovery.
219	L.M.N.	4-7-33	40	Complete prolapse.	Vaginal suspension, Shortening of Mac's ligaments, Amputation of cervix.	Recovery.
235	C.S.	10-8-33	26	Procidentia congenitalis.	Complete prolapse operation performed. Vaginal ventral suspension. No perinaeorrhaphy performed.	Recovery.
236	L.S.	21-8-33	38	Complete prolapse.	Complete prolapse operation. Vaginal ventral suspension and perinaeorrhaphy.	Recovery.
256	K.M.	17-10-33	22	2nd degree prolapse Retroverted uterus.	Complete prolapse operation and Colpo-perinaeorrhaphy.	Recovery.
257	P.A.P.	24-10-33	63	Complete prolapse.	Complete prolapse operation. Vaginal suspension and Colpo-perinaeorrhaphy.	Recovery.
260	T.Y.F.	24-10-33	63	Complete prolapse.	Complete prolapse operation and Colpo-perinaeorrhaphy.	Recovery.
268	W.K.M.	28-11-33	48	Complete prolapse.	Interposition operation done. Colpo-perinaeorrhaphy.	Recovery.
11	C.Y.	3-4-34	44	Complete prolapse.	Plastic operation for complete prolapse with vaginal ventral suspension and perinaeorrhaphy.	Recovery.

Table No. IX.

Miscellaneous Operations.

No.	Name	Age	Date	Disease	Operation	Result	Remarks
64	**T.Y.H.** L.N.W.	55	11-5-33	Fibro-myoma.	Myomectomy.	Recovery.	Tumour size of 5 months pregnancy. On opening abdomen found tumour lying in cervical canal. Tumour removed per vagina. Cervix divided anteriorly and posteriorly. Tumour removed piecemeal. Weight—½ lb.
73	N.Y.	32	18-5-33	Tumour of uterus.	Vaginal myomectomy.	Recovery.	Tumour size of a sheep's heart removed from vagina.
129	K.K.	45	3-8-33	Pedunculated submucous fibroid.	Myomectomy.	Recovery.	Tumour twisted off. Uterus curetted and packed.
152	C.L.	53	7-9-33	Fibro-myoma.	Myomectomy.	Recovery.	Tumour size of a pigeon's egg was twisted off from cervix. Uterus curetted.
194	L.C.L.	40	13-11-33	Tumour of uterus.	Myomectomy.	Recovery.	Left ovary enlarged to size of a cricket ball. Cyst contained collection of blood clots. Ovary removed. Uterus suspended.
199	H.M.	42	23-11-33	Pedunculated submucous fibroid.	Myomectomy.	Recovery.	Tumour size of a tennis ball twisted off. bed of tumour clipped off with Schultz's spoon forceps. Uterus curetted and plugged with gauze.
203	C.M.	40	30-11-33	Tumour at side of uterus.	Myomectomy and resection of ovaries. Ventral suspension.	Recovery.	Myoma size of golf ball removed. Both ovaries resected. Owing to that the uterine cavity was encroached upon it was deemed advisable to crush and tie both tubes.

Table No. IX.—*(Continued 1)* *Miscellaneous Operations.*

No.	Name	Age	Date	Disease	Operation	Result	Remarks
219	**T.Y.H.** C.Y.K.	36	18-1-34	Cervical polypus.	Removal of polypus.	Recovery.	Uterus vertical in position. Apparently some adhesions in the pelvis.
223	N.Y.	24	25-1-34	Retroverted gravid uterus with Ascites.	Laparotomy.	Recovery.	Large swelling in Douglas' Pouch and big rectocele. Abdomen size of 7 months pregnancy. Upper surface of bladder thickened considerably. Bladder causing retroversion of uterus. Dark coloured fluid 160 oz. withdrawn.
236	L.Y.T.	48	8-3-34	Pedunculated submucous fibroid.	Myomectomy.	Recovery.	Tumour removed. Stalk of pedicle curetted.
266	K.M.	29	26-4-34	Broad ligament cyst.	Laparotomy.	Recovery.	Small broad ligament cyst size of a mandarin orange. Right ovary enlarged and had a small cavity containing pus.
268	F.T.	40	26-4-34	Myoma.	Myomectomy and left oophorectomy	Recovery.	Entire uterus found to be myomatous. Many adhesions due to a chocolate cyst. Owing to difficulty of with drawing the uterus, myoma was cut out and cervix left. Left ovary was removed.
194	**G.C.H.** L.A.Q.	49	8-5-33	Pedunculated submucous fibroid.	Tumour clamped with Schultz's spoon forceps and twisted off its pedicle. Uterus curetted.	Recovery.	Fibroid presenting at external os, size of a walnut.
205	W.Y.H.	55	30-5-33	Vesico-vaginal fistula.	Laparotomy for repair of vesico-vaginal fistula.	Recovery.	Bladder pushed down on uterus.

Table No. IX.—*(Continued 2)* *Miscellaneous Operations.*

No.	Name	Age	Date	Disease	Operation	Result	Remarks
	G.C.H.						
216	L.Y.	33	27-6-33	Fixed retroversion.	Uterus, thickened and occluded, tubes adherent to Douglas' Pouch. Both tubes found closed, not opened. Uterus suspended. Partial excision of left chocolate ovarian cyst.	Recovery.	
217	L.S.Y.	40	27-6-33	Submucous fibroid.	Fibroid clamped with Schultz's spoon forceps and twisted off.	Recovery.	Fibroid attached to inner surface of anterior lip of cervix.
222	L.A.C.	37	18-7-33	Uterine fibroid.	Uterus sheath and peritoneum oedematous, and firmly adherent to anterior wall of cervix. Impossible to separate adhesions due to pus condition.	Death.	Uterus cut into accidentally and flaky pus issued from side. Patient died on 28-7-33 at 5.30 a.m. Provisional Pathological Diagnosis at Autopsy:—"Generalised peritonitis following septic broad ligament cyst also right basal pneumonia."
226	C.C.	24	25-7-33	Fixed retroversion.	Laparotomy. Uterus suspended.	Recovery.	Uterus bound down to Douglas' Pouch by adhesion. Right tube thickened resected. Right ovary partially resected. Left tube also thickened, patent.
227	C.P.	28	25-7-33	Submucous fibroid.	Fibroid removed piecemeal with Schultz's spoon forceps.	Recovery.	Pedunculated submucous fibroid in uterine cavity. Patient suffered from pelvic cellulitis had swinging temperature for about three weeks.

Table No. IX.—*(Continued 3)* *Miscellaneous Operations.*

No.	Name	Age	Date	Disease	Operation	Result	Remarks
238	**G.C.H.** F.W.M.	38	21-8-33	Fixed retroversion cervical polypus.	Cervix incised as tumour was too big to be enucleated.	Recovery.	Tumour size of an egg, half in the cervix and half in the uterus.
251	W.S.	51	15-10-33	Retroperitoneal malignant tumour.	Incision made in midline, malignant growth found. Abdomen closed.	Recovery.	Abdominal wound healed nicely, but tumour still about same size, general condition better.
290	K.M.C.	40	6-2-34	Double hydrosalpinx.	Laparotomy.	Recovery.	Both tubes removed. Right ovary removed. Myoma from front and another at the back of uterus shelled out.
19	C.A.T.	35	17-4-34	Vesico-vaginal fistula and pruritus vulvae. Operation	Plastic operation.	Recovery.	

Table No. X.

Mortality.

No.	Name	Age	Admitted	Dead	Diagnosis	Treatment	Remarks
234	**T.Y.H.** K.K.M.	49	28-2-24	13-3-34	Carcinoma of cervix. Pulmonary embolism and cardiac failure.	Radium inserted 1-3-34 12-3-34. Patient had bleeding from vagina. Hot vaginal douche and vaginal plugged 13-3-34. Had dyspnoea and pain over epigastrium. Heart sound weak and slow. Cardiac stimulants given. Oxygen inhalation given in the afternoon.	Vagina shortened. Os admitted one finger. Uterus fixed. Dyspnoea more marked in the afternoon 13-3-34. At 6.45 p.m. severe pain over front of chest. Face marked cyanosis. Died at 7.20 p.m.
237	Y.S.	43	8-3-34	16-3-34	Malignant ovarian cyst. Peritonitis and cardiac failure.	Ovariotomy and radium inserted. Had severe vomiting after operation. Turpentine enema given, and castor oil given in the following morning. Radium removed owing to bad condition of patient. 11-3-34 Calomel and salt given. 12-3-34 Mag sulphate ½ hrly. Vomiting stopped. 13-3-34 Patient developed trismus. 15-3-34 Vomiting recurred Pulse weak. Cardiac stimulants given.	Swelling size of 6 months pregnancy. Vomitted matter black colour. Patient died at 7.30 a.m. 16-3-34.
267	C.K.	28	23-4-34	2-5-34	Carcinoma of the cervix.	Radium therapy.	Cervix occupied by a growth size of a dollar. No involvement of vaginal wall. Very marked anaemia. General condition of patient exceedingly poor.

Table No. X.—*(Continued)*

Mortality.

No.	Name	Age	Date	Dead	Diagnosis	Treatment	Remarks
222	**G.C.H.** L.A.C.	37	10-7-33	28-7-33	Uterus fibroid. Provisional Pathological Diagnosis at the Autopsy:—Generalised peritonitis following septic broad-ligament cyst, also right basal pneumonia.	Incision made over tumour. Rectus sheath peritoneum oedematous and firmly adherent to anterior wall of uterus. Uterus was cut into accidentally, flaky pus issued from the side. Impossible to separate adhesions due to pus condition.	Cervix conical. Uterus enlarged to size of 5 months pregnancy. More or less fixed. Tenderness over tumour.
294	B.S.	69	16-2-34	17-3-34	Malignant ovarian cysts. Papillary carcinoma of ovary.	Laparotomy and ovariotomy. Radium inserted.	Tumour size of full term pregnancy. Papillomatous mass on each side surrounding uterus. One on left size of cricket ball adherent to rectum. Tumour shelled out as far as possible.
20	L.Y.	36	16-4-34	24-4-34	Fibroid. Typhoid	Subtotal hysterectomy.	Tumour size of 7 months pregnancy. Temperature keeping up after operation and became delirious on 24-4-34 and died at 12.50 p.m.

HUMAN GENETICS AND ITS RELATION TO MEDICAL PROBLEMS.

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INTRODUCTION.

Modern clinical medicine has often been aptly likened to a vast complicated jigsaw puzzle, an unfinished task at which scientists through the centuries have been patiently working, some contributing to the picture additions of immediate and obvious worth, and others un-ostentatiously passing on and leaving to time and later workers the task of discovering the value of their labours. Men of all ages and every branch of pure science have contributed knowingly or unknowingly to the growth of this picture, the forms and details of which have become more and more complicated as each generation added its tested and tried contribution.

The nucleus of the picture was formed when those first few nebulous pieces were placed in position centuries ago by folk-lore; to this indifferent nucleus, the ancient thinkers of Greece added their quota, partly by direct observation and partly by philosophy, and thus was developed a conception of human medicine as indeterminate in size and form as it was futile in its applications. For hundreds of years the puzzle lay untouched on the table of time, no one daring to add to, or subtract from, what had now become accepted, by hardly any other qualifications than hoary age, as a completed and perfected masterpiece of art; a masterpiece so revered that criticism was almost unheard of, and if it did exist, it amounted to sacrilage and was even punishable by death itself.

But as time passed, workers in another part of the table began, by animal experiments and a few, but accurate observations on the human cadaver, to piece together what proved to be the first real and satisfactory part of the future picture. Gradually the pieces took shape, but the ever present critics refused to believe that it would fit on to the existing nucleus, and in this contention they were right. Harvey's work did not fit in with the existing knowledge, but not because his work was wrong, but because the previous work was faulty both in design and orientation. The old setting had to be scrapped, and once the work of Harvey was firmly established as its centre piece, the building of the picture of clinical medicine moved on apace. The whole structure seemed to be surrounded with growing points. The sciences of physics and chemistry were both youthful and vigorous, and from time to time it needed only the advent of a new-comer with fresh vision to see how and where existing separate combinations of knowledge of these subjects could be correctly applied with advantage to the main picture. Thus began to develop modern physiology; the further discoveries of electrical

reactions led to the addition of nervous, to that of chemical regulation of bodily function. Knowledge of biochemistry and improved methods of chemical analysis threw new light on the parts played by ductless glands and revolutionised diagnostic methods, and the X-ray tube provided yet another instrument of penetrating observation for both diagnosis and research. Meanwhile the physics of light had resulted in the introduction of the microscope, and with that it was but a matter of time until histology, normal and morbid, and bacteriology, became established and important sciences. Bacteriology plus chemistry opened the way via asepsis and antisepsis to the remarkable achievements of modern surgery. So amazing have been these achievements made possible through bacteriological advances, that it is no wonder that the greater part of modern medical endeavour is directed against environmental forces, or that the picture of modern medicine is so well developed on the bacteriological side.

From this brief and incomplete resumé, one can readily see how the picture of clinical medicine has by now grown so large that individual workers on one part of the structure, are wholly unable to keep pace and cope with developments in other parts of the field. Only those who have witnessed the tremendous additions of the last two generations, or who are able to take the time and trouble to view the position from vantage points removed from anyone region of rapid growth, can appreciate the immensity and beauty of the developing picture. When a general survey is made from such a position, two facts seem to be worthy of note. They are (*a*) that growth in many parts of the field has become so rapid that the growing points are now far removed from the centre of the picture, and (*b*) that the study of environmental factors has proved so valuable that the whole picture tends to run the danger of being overbalanced or unbalanced by its weight. To overcome the disadvantages arising from the former, it is the duty of those interested to continually widen the base of the applications of new discoveries to the central structure, thus making such applications more easy and obvious; the method of correcting the latter tendency is much more difficult and the discussion of this problem will occupy much of our time and consideration in the following chapters.

To return for a few moments to the opening simile of this chapter, in the early part of the latter half of last century, an obscure monk was working in the middle of Europe trying to piece together the facts of inheritance of certain characters in plants. The work was brilliantly conceived, faithfully executed and remained peacefully unknown until the beginning of this century when it was rediscovered, and its value recognised. Thus was rediscovered the forgotten birth of science's last-born—Genetics, a subject which has developed so rapidly in the last 30 years that it is now ready to be applied to the picture of clinical medicine. There are not wanting, even in these enlightened days, those who fail to

see how and where this newest of the sciences can be fitted with any advantage, to the human picture. It will be our object to try and explore the gulf which seems to separate the two sciences of human medicine and human genetics, to suggest methods of bridging it, and to indicate how genetics may be able to restore the balance of clinical study by bringing the forces of the body of inborn origin into equal prominence in the mind of the clinician with those of the environment.

CHAPTER I.

THE PRESENT POSITION.

The reasons why the study of human genetics is so undeveloped are manifold but may be roughly divided into two groups depending on (*a*) the ignorance of people in general of the facts of heredity and (*b*) the inability of the members of the medical profession in particular to comprehend the value of these facts. The most important reason in the former group is the lamentable persistance of the old and decrepit anthropocentric idea that man has been specially endowed with powers that lift him out of the sphere of action of laws which govern plants and lower animals. The most efficient way of dispelling the erroneous ideas of both types is by firmly implanting correct ideas in the minds of the members of our own profession first of all, and with that end in view the following arguments are elaborated. In reviewing therefore the relation of genetics to medical problems, it is proposed to do so from the viewpoint of the general practitioner and not of the specialist or geneticist, because it is the former who will have to be convinced of the importance of this relation and who will have to make use of its application.

At the present there are a few far sighted medical men who are convinced of the importance of the relation of genetics to medicine; there are some who are mildly sceptical of its value, but who are open to conviction; but by far and away the vast majority of medical men have no conscious thought on the matter at all. One writes 'conscious' purposely because every general practitioner is in reality constantly applying genetical knowledge in his daily practice whether he realises it or not. As soon as an epidemic breaks out in his district, he is not surprised to find family 'A' in trouble again. Through the years of experience as their family doctor he has learnt that they are of low resistance stock, and readily become easy victims of every epidemic in the neighbourhood. Lucky family 'B' next door never seems to be worried by outbreaks of this kind at all. He knows, immediately he gets an urgent message from family 'C' exactly how much reliance to put on its urgency, for experience has taught him that worrying is a characteristic trait of members of that family. He gives a guarded prognosis when Mr. D. develops pneumonia because he has already had experience of the poor fight other members of this family have put up against the

pneumococcus. He is always on the lookout for tuberculosis in family 'E' because all the children seem to have the features so often associated with those prone to develop the disease, and if he has kept up with modern trends, he is constantly applying his observations on diathesis in his differential diagnoses. He may even go so far as to advise against cousin marriage when asked for his opinion, although the scientific basis for his advice is most probably a closed book to him; and he will almost certainly know at least one family many members of which have suffered from the effects of a high systolic blood pressure and who have all died at about the same age as a result, direct or indirect, of this family character. He will also be able to quote instances of parents concerning whom both the lay and medical opinion is that they should have no more progeny because each successive child as it develops, turns out to be feeble minded. In fact any general practitioner would be able to so supplement this list as to produce one whose size would be a revelation and a very potent reminder of the great number of conditions which "run in families".

Since most of this knowledge has been handed down to us, we might even go so far as to suggest that our professional forbears used genetical knowledge, empirical no doubt, much more than we do in our enlightened scientific age, and to much greater advantage. The taking of a detailed family history still forms an important part of the routine examination to which every medical student learns to subject his patient, but the use to which this knowledge is put in these days is relatively small. The taking of a family history is more or less a vestigial part of the routine, a part whose value tends to be dwarfed unfortunately, by that of the more obvious, and so called scientific, aids of the laboratory and the microscope.

But its very persistance as a part of the modern routine of case taking proves how valued an instrument it must have been in the hands of the older physicians, and it but needs careful modern scientific treatment to make the family data now locked away in hospital records, play its part in the hands of the future physician, a part in its own sphere every bit as comparable to that played by the test tube.

The view here put forward is that empirically genetics has played, in the hands of the old, observing physician, a very important part in the art of healing, but that modern advances in other directions have so overshadowed its value, that the younger scientific physician is in danger of losing the ability of utilising its help; nor is it difficult to see and understand how this state of affairs has come about. We have already noted in the introductory chapter that as each new science developed, its base was broadened and its application to clinical medicine made more easy and obvious by interested workers. It is only after such treatment that intricate and scientific discoveries can become valuable clinical aids.

In these enlightened days the clinical value of empirical beliefs must necessarily fade on the introduction of established scientific facts. But the strict application of statistics to accurately observed experimental and natural data, has caused genetics to develop from the stage of scattered empirical beliefs to that of an exact science and the time is now ripe for medicine to accept the help that applied genetics has to offer.

There are many reasons why this application may be difficult, and by far the most important of these is the insufficient genetical instruction given during the period of medical training. It is all very well a few enthusiasts proving to their own satisfaction the value of this new science to the physician, but if the physician has not had the training to record the required data in a usable manner or to interpret and appreciate the value of the results when they have been obtained, genetics will never attain the status of an applied science.

At present the already overloaded curriculum gives time for but a few lectures on Mendelism, given either in the botany or zoology course, or in some favoured cases in both, but it is impossible to get far in the time available, and if the crammed, newly qualified practitioner has any knowledge left of genetics at all, it begins and ends with a vague notion of 'the 3:1 ratio', and 'dominants and recessives', indissolubly but inexplicably linked with dim memories of 'talls' and 'dwarfs.' (Should any excuse be necessary for this statement it will be found in the writer's own personal experience, and the more one investigates, the more common one finds that experience to be. The statement is not made to cast reflections on the medical practitioner, but to draw attention to the shortcomings of medical education; nor is the statement concerning the 3 : 1 ratio meant to be taken too literally; rather is it meant to symbolise what is considered to be the inadequate genetical knowledge possessed by medical men.)

With an inadequate foundation of genetical knowledge is it any wonder that the physician readily gives up any ideas of getting any help from this branch of science, for in his practice he never meets the 3 : 1 ratio, and the talls and dwarfs merge indistinguishably into one another? In order to try and prove that it is not the inadequacy of the science, but our ignorance of its applicability that is to blame for this state of affairs, it is proposed here first of all to give a brief and elementary description of the main facts of genetics and then to try and show why human data do not seem to fit in with the simple genetical laws with which each newly qualified practitioner is acquainted.

The question before the medical jury is; "Has genetics yet attained the status of an applied medical science and if it has, has such application any immediate or great value?" Here follows an attempt to state the case for the affirmative.

CHAPTER II.

GENETICAL RATIOS REVEALED BY THE LAW OF SEGREGATION.

In the last chapter two main assertions were made. They were, (*a*) genetics has reached the stage where it may profitably be applied to the science of clinical medicine and (*b*) the main factor in preventing this successful application is that the genetical knowledge of the average medical man is such as to make it impossible for him to realise its value or to successfully apply the knowledge he possesses. To be rewarded by a favourable verdict from the hypothetical medical jury on this case, one must first of all establish to the jury's satisfaction the facts of modern genetical science relevant to the case, and then show how the application of this knowledge may be of use to the clinician.

With the former of these two tasks we shall now deal, leaving the latter to be dealt with in later chapters.

Mendel's Experiments on Characters Involving a Single Pair of Factors.

Mendel in 1865 published his monumental work on the inheritance of certain characters of the edible pea. The members of one race of pea which he used were all tall and when bred together under normal conditions, always produced tall plants. He assumed that these plants possessed the character of 'tallness' and since the only connection between one generation and the next was the germ cells—pollen and ovules—, this character must have been transmitted in these cells. The members of another race were all short, and these likewise when bred amongst themselves under similar conditions to the first lot, always produced short plants. They therefore possessed the character of 'shortness' which also was transmitted through the germ cells. (The actual height of the tall race was 6—7 feet and that of the short race about 1 foot.) When he artificially crossed a member of the tall race with one of the short race (this parental generation is designated P1) he obtained a generation of peas all the members of which were tall. (This generation—the result of the P_1 crossing — is known as the *first fillial generation*, or for short F_1.) Why were the members of F_1 all tall? What had happened to the character of shortness? The first experiment of breeding talls with talls, and shorts with shorts, proved that the characters of tallness and shortness were handed on to the next generation, therefore the same handing on must take place in the second experiment when the hybrid is formed. The hybrids must thus contain both characters; (since such a character is an attribute of the grown plant, it can hardly be correct to say a plant has the character of shortness when it is not short but tall; there must therefore be a difference between the *'character'* which appears in the individual and that entity which is handed on and which causes the appearance of the character. This entity is termed a *'factor'* and

hence it would be more correct to state each hybrid contains both factors.) But since both factors concern height, the result can only be one of three things.

Each F_1 individual must be

either (*a*) tall) thus exhibiting one character in preference to
or (*b*) short) the other,

or (*c*) both tall & short) i.e., intermediate in height between the parents and thus exhibiting both characters to a limited degree.

Note the phrase "each F_1 individual." Another possibility is that some F_1 individuals may be tall, some others being short, but this possibility is not considered because it is not found to occur. Actual experiment proved that all F_1 individuals were tall, and so, although they possessed both *factors*, they exhibited only one *character;* the tall character must have *dominated* over the short. The character of tallness is said to be *dominant* to that of shortness. This is implied by the use of symbol "*T*" for the factor which will produce tallness and "*t*" for the factor which may result in shortness, the capital letters always being used to denote the factor producing the dominant character.

Mendel's next step was to prove that the hybrid did actually contain both factors. This he did by self-fertilizing members of the F_1 generation. The result was the second filial generation (F_2). (Note that F_2 is not synonymous with 'grand-children', a mistake sometimes made in medical literature. An F_2 generation is the result of mating individuals from the same F_1 generation, and in order for the F_2 individuals to be 'grandchildren' the F_1 mates would have to be brother and sister an event very rare in human work.) In all, Mendel produced 1064 members of the F_2 generation and he found 787 were tall and 277 were short. The F_1 plants therefore *must* have carried the short factor although the character of shortness did not appear in the stature of the adult plant. The character which *appears* is known as the '*phenotype*,' in this case it is "tall". It was not till the F_2 generation was produced that the characters of the grandparents were again produced in separate individuals, and the ratio of talls to shorts was 2.84 to 1, or approximately 3 : 1. Even in his classical experiment Mendel did not find exactly 3 : 1, and why therefore should we use the occurrence of the exact 3 : 1 ratio as a yard stick to measure the applicability of genetics to human material?

The F_1 members thus carried both the factors *T* & *t*, in other words each plant carried a pair of factors which, under suitable circumstances to be defined later, were able to affect one attribute of the adult plant—in this case its height. Such a pair of factors is known as an ***allelomorphic pair,*** each factor being known as an ***allelomorph.*** The

members of the parental generation must each have also carried a pair of factors, and since the talls when bred together always produced talls, their pair must have been *TT*, and similarly the shorts must have carried *tt*. Here should be introduced the term *'genotype'* which is so common in genetical literature. The genotype or genetical type of the plant is the expression used to describe the faculties possessed by the plant for developing characters, which faculties it is capable of handing on to its offspring. In the case of the parental talls the genotype is *TT*, of the parental shorts *tt* and of the F_1 hybrids it is *Tt*. The term *phenotype* does not distinguish between the parental tall and the hybrid tall. They are both of the phenotype "tall", but in their genotypes they are different, that of the former being *TT* and the latter *Tt*. Of these two factors possessed by the hyprid the *T* must have been inherited from the tall parent and the *t* from the short parent, and the only possible means of such inheritance is by way of the germ cells. Therefore each germ cell must have received *one* of the allelomorphic factors possessed by the plant which produced it, in other words during the germ cell formation there must have been a separation of the factors forming the allelomorphic pair. This process is known as *SEGREGATION* and its enunciation formed Mendel's first law. The allelomorphic pair of factors in the tall parent was *TT*, hence *every* germ cell from that parent contains *T*. The allelomorphic pair in the short parent was *tt*, hence *every* germ cell from that parent contained *t*, hence the allelomorphic pair in every member of the F_1 generation must have been *Tt*. Such an individual is said to be *heterozygous* because it contains one factor of each kind, while individuals such as the members of the parental generation are said to be *homozygous* because they contain two factors of the same kind, the genotypes being either *TT* or *tt*.

If this law of segregation be true, what happens when the hybrid F_1 plant forms its germ cells? Some germ cells will receive *T* and others *t*, and moreover, the chances of such cells receiving *T* or *t* being equal, 50% of the germ cells will receive *T* and 50% *t*. When fertilization of two germ cells of two F_1 hybrids takes place:—

(a) the germ cell of the one carrying *T* may meet a germ cell of the other carrying *T* forming a homozygous individual—genotype *TT*, phenotype "tall,"

(b) the germ cell of the one carrying *T* may meet a germ cell of the other carrying *t* forming a heterozygous individual—genotype *Tt*, phenotype "tall,"

(c) the germ cell of the one carrying *t* may meet a germ cell of the other carrying *T* forming a heterozygous individual—genotype *tT*, phenotype "tall," or

(d) the germ cell of the one carrying *t* may meet the germ cell of the other carrying *t* forming a homozygous individual—genotype *tt*, phenotype "short."

All these possibilities should have an equal chance of happening, so that in a large number of experiments we should find three "talls" to one "short." This, as we have seen above, Mendel actually found to be the case by experiment. This happening is better shown in the following chart, Figure 1.

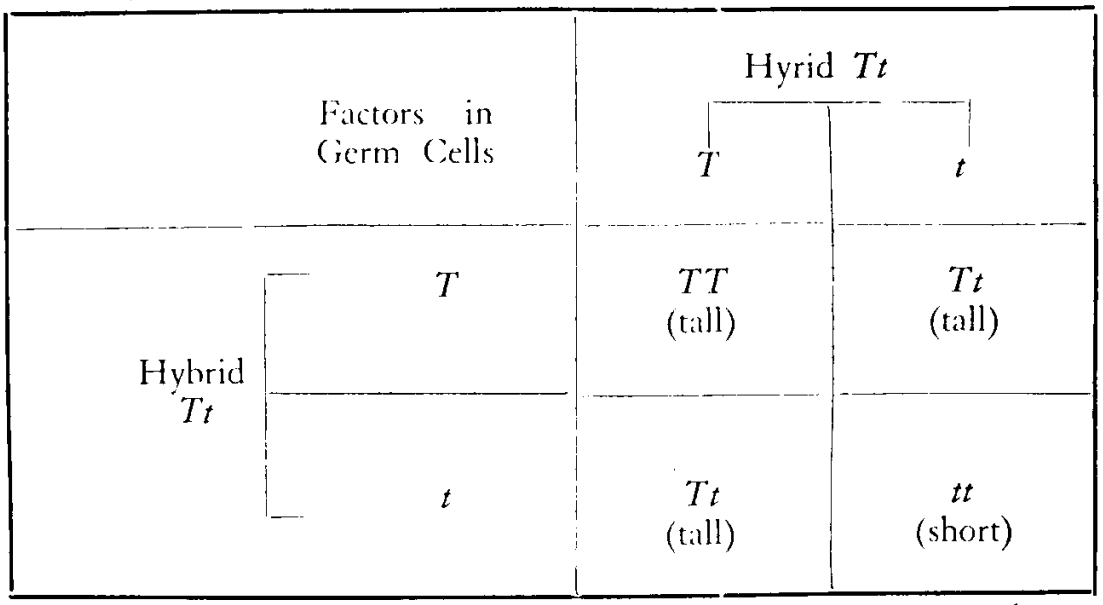

Figure 1. A chart showing how the hybrid genotype '*Tt*' gives two types of germ cells, one carrying '*T*' and the other '*t*,' and also how the crossing of the two hybrids gives three 'talls' (one homozygous '*TT*,' two heterozygous '*Tt*') and one 'short' '*tt*.'

Mendel next self-fertilized members of this F_2 generation and proved that 1/3 of the "talls" always bred "talls", i.e., were homozygous "talls" (*TT*), and 2/3 of the "talls" bred both "talls" and "shorts" in the ratio of approximately 3 : 1 proving these F_2 talls to be heterozygous (*Tt*), while the shorts always produced shorts, proving them to be homozygous (*tt*). It should be noted that it is only when "short" factors are present in a cell unaccompanied by the factor for "tall" that short factors can express themselves by making the plant short. The character "short" is then said to be *recessive* to the dominant character "tall".

To put the proof of segregation beyond doubt, Mendel then performed an experiment which has since become a standard method of experimental genetical procedure, back-crossing the hybrid F_1 to the recessive parental stock, in this case crossing a *Tt* individual with a *tt* individual. Here the latter parent can only contribute the genetical factor *t* to the progeny, and if no segregation takes place, the former parent must hand on *T* in its germ cells, with the result that the progeny, all receiving a *T* factor would be tall. If segregation takes place 50% of the germ cells of the former parent would contain *T* and the other 50% *t*, in which case half of the offspring would be tall (*Tt*) and the other half short (*tt*). Mendel's experiments showed the result to be half talls and half shorts, (Fig. 2).

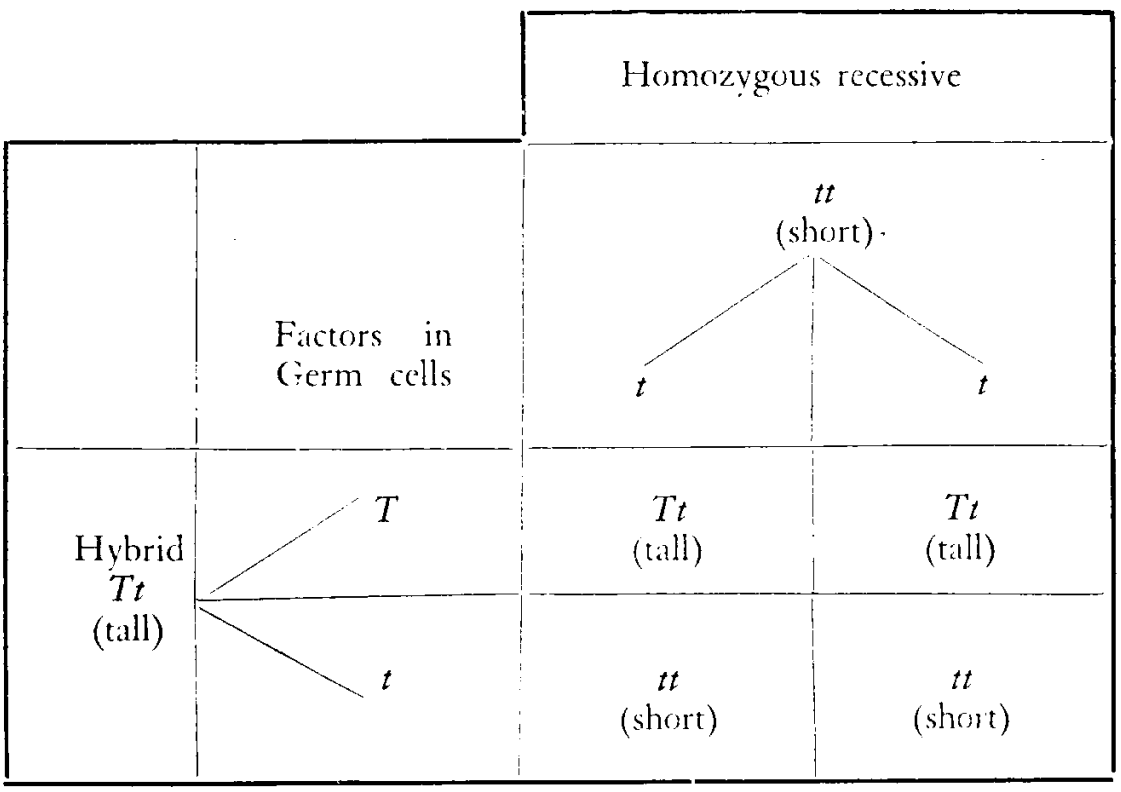

Figure 2. A chart showing the result of back-crossing a hybrid (*Tt*) with the parental recessive (*tt*) resulting in equal numbers of heterozygous talls (*Tt*) and homozygous shorts (*tt*).

Thus did Mendel prove the segregation in the *hybrid* of the pair of factors governing height. But he did not stop there. He showed the same law to hold with many other pairs of factors which governed the colour of the cotyledons, their nature (smooth or wrinkled), colour and form of the pods, etc., and in each case the results have been amply verified by subsequent investigators.

Incomplete Dominance.

Thus far we have been considering characters which Mendel showed to be completely dominant or recessive. His work lay forgotten until 1900 when it was rediscovered independently by three workers, de Vries, Correns and Tschermak, and its immense value began to be realised. Its rediscovery was the stimulus for the launching of investigations into the behaviour of many other characters in both plants and animals, and it was soon found that some of these characters did not behave in the same way as we have seen those for tallness did.

On page 59 it will be remembered we stated that of two contrasting characters, each F_1 individual must either exhibit one character only (of its parents) in preference to the other, or exhibit both characters to a limited degree. Mendel's experiments dealt with the former types. Now we must consider the latter type. The classical examples given in text books deal with certain flowers (four o'clocks) and fowls.

There are white-flowered and red flowered varieties of the ordinary four o'clock (*Mirabilis jalapa*), each variety when self-fertilized breeding true. The white variety thus carries the factor for "white", and the red variety the factor for "red." When a cross is made between a red and a white plant, the F_1's are all the same, but each individual F_1 plant is neither completely red nor completely white; every F_1 individual is pink. Unlike the F_1's obtained from crossing talls and shorts, these do not completely resemble either one of the parents in regard to the characters under consideration, but resemble them both to a certain degree. Each individual F_1 thus *not only carries the factors* for both red and white, but also *shows the characters* red and white simultaneously in each of its flowers. Neither character is dominant to the other. It is an example of *incomplete dominance.*

If members of this F_1 generation are crossed, the F_2 generation contains red, pink and white flowered varieties in the ratio of 1 : 2 : 1, the reds breeding true on self-fertilization proving them to be homozygous for "red", the whites breeding true on self-fertilization proving them to be homozygous for "white," while on self-fertilization the pinks again give reds, pinks and whites in the same ratio of 1 : 2 : 1, showing them to be heterozygous genotypically (as well as phenotypically) for "red" and "white". The phenotype of the heterozygote is here distinct and different from that of either of the homozygous plants. (Fig. 3).

The disappearance of the 3 : 1 ratio in this experiment is, however, only apparent, for if we regard pink as red plus white, there are three plants in the F_2 generation which contain red to every one which does not contain it, and from this point of view red is dominant to white. But there are also three plants that contain white to every one that does not contain it and therefore white may be considered dominant to red. It is really immaterial which way one regards it.

The points worthy of note here are (1) that although we cannot say that one character is dominant to the other, the whole experiment when investigated genetically still conforms to the law of segregation and (2) (which is more important in our argument) the apparent absence of the 3 : 1 ratio can be quite easily explained by our already meagre genetical knowledge.

The other example referred to above is that of the Andalusian fowl which is an F_1 formed by crossing a splashed-white fowl with a pure black, details of which can be found in almost any genetical text book. In these cases the hybrid is found to be mid-way between its parents in regard to the character under investigation, and we can still put the F_1's into their three respective phenotypical categories.

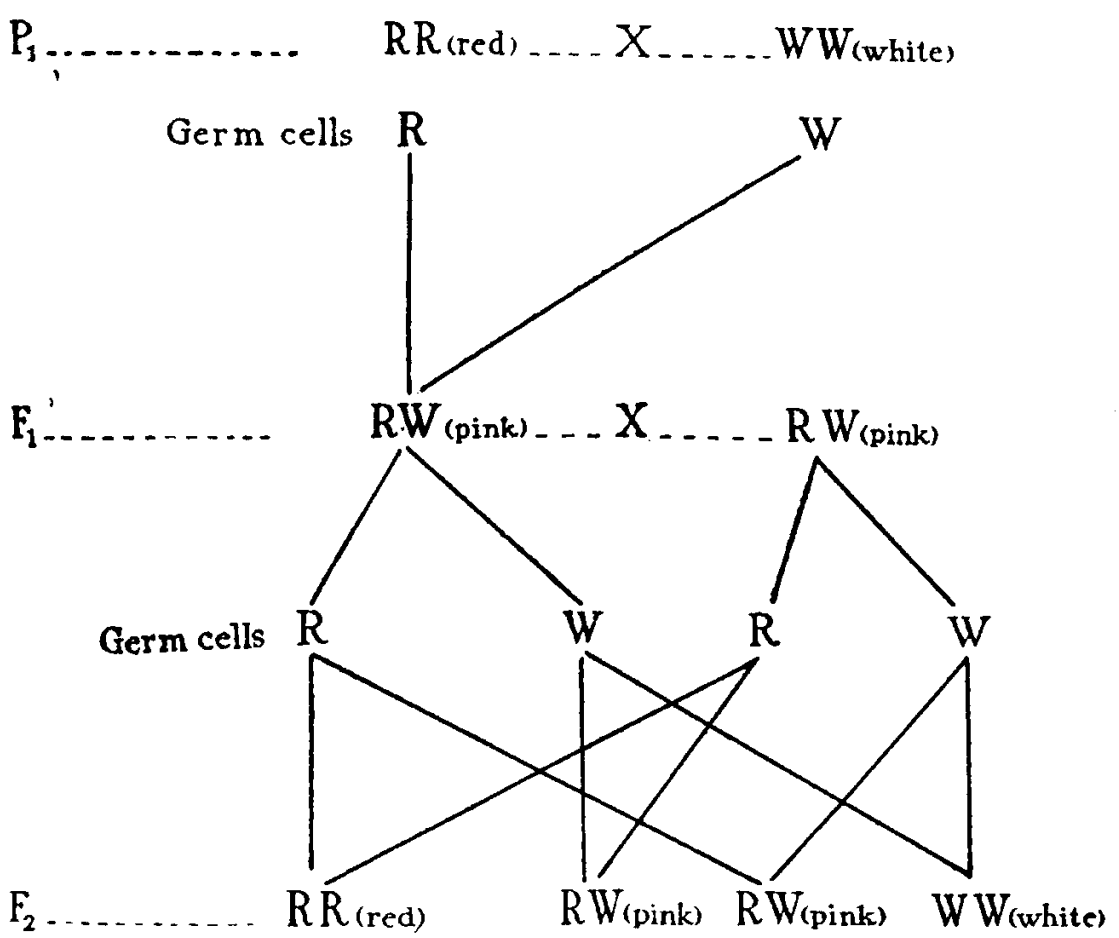

Figure 3. A chart showing three generations of *Mirabilis jalapa* (four o'clock): the P1 is a crossing between homozygous members of the red (*RR*) and the white (*WW*) varieties; the F1 members are all pink (heterozygous) and when two of these are crossed, the F2 shows one red (*RR*) to every two pinks (*RW*) to every one white (*WW*). Genotype represented thus *RR*, phenotype thus (red).

Inheritance of Characters Forming a Continuous Series.

We must now consider cases where the F_1's are not even phenotypically homozygous and where it is very difficult and almost impossible to divide them up into different categories. The work of Morgan and his school in America has supplied, if not the greatest biological advance of the century, certainly the most important genetical work since the time of Mendel. It furnishes an outstanding example of how science and even human biology can be advanced by a thorough study of an almost insignificant form of life. In the fruit fly, *Drosophila melanogaster*, over 200 pairs of factors have been examined, and in addition to the making of new discoveries and the enunciation of new laws the results have always been the same—the verification of Mendel's laws.

Especially do we want to consider here the case where a race with ebony wings is crossed with one with sooty wings. Here the whole gamut of wing colour from apparently pure ebony on the one hand,

through mixtures of both, right down to pure sooty on the other hand, is represented in the F_1 generation; and the gradation is by steps so small that it is very difficult to place the individuals into categories which would be purely arbitrary. When these F_1 flies are inbred, we again get a series with ebony at one end and sooty at the other. The F_2's show no signs of a 3 : 1 ratio, in fact no sign of any ratio at all. It seems as though the *characters* have blended, but the important thing is that the *factors* have not. They still undergo clear cut segregation as can be proved by back crossing the F_2's to the pure parental stocks. As the result of this we find that there is one group of F_2's which, when mated to the P_1 ebony, gives only ebony progeny. There is another group which gives again a varying progeny, most of those occupying the more ebony end of the scale resulting from the ebony—F_2 back-cross, and those in the sooty end from the F_2-sooty back-cross; there is yet another group which, when mated to P_1 sooty gives only sooty progeny. These three groups are approximately in the ratio of 1 : 2 : 1. This back-crossing experiment proves that even in this case where we get the utmost variation possible in the characters exhibited by the hybrid, clear cut segregation of factors takes place.

The study of this type of inheritance leads us medical people into rather deep water, but it is here that the pure geneticist makes his most valuable discoveries, and we must then accept his word that, even when things are beyond our depth so that personal testing is to us impossible, the law of segregation still forms the rock bottom. This dependence on the genticist is an important fact to realise. Most of us have enough mathematical ability to cope with problems such as the earlier ones quoted above, where the F_2's all fall into easily recognised classes and form a discrete, integral or discontinuous series, but only the expert in modern statistics can hope to cope with the situation such as that found in the F_2's in the drosophila experiment quoted. There the differences form a continuous series, and it would be an uneconomic use of his time for the clinician to learn the mathematical methods of dealing with such data. Other parts of his work are much more important, but what is vital is that he should appreciate the foregoing, and be able to collect the data in the form usable by the expert. When we come to human work we find very few examples of the discrete series of characters. This series may be typified by the hereditary agglutinogens and agglutinins found in the blood, (all human individuals fall into one of the four blood groups and no cases can be intermediate between any two groups), and deaf-mutism, (the individual under consideration is either a deaf-mute or not.) By far and away the greater majority of human characters form a continuous series and the data has to be supplied to an expert geneticist for his decision as to whether the factors concerned behave according to Mendelian laws. Examples of such characters are eye-colour (we see all grades ranging from light blue to dark brown), hair colour, stature, cephalic index, resistance to disease etc.

Multiple Factors.

We must now consider what happens when the development of a character depends on more than one factor. This type of inheritance is proving to be more common that was originally thought and it is especially in evidence in such characters as colour in both plants and animals. One or two classical examples here will be sufficient to make our point clear.

Bateson and Punnett working with two varieties of white sweet pea, found a remarkable thing when these varieties were crossed. As there was no chance of an intermediate colour between the white of one parent and the white of the other, all the F_1's might reasonably have been expected to yield white flowers, but they all turned out to be purple. The explanation lies in the fact there are two pairs of factors involved in the formation of the purple colour, (*a*) a pair governing the ability to produce any pigment and (*b*) a pair governing the development of the particular colour (purple in this case) itself. This will be better understood from the following. Let the power to develop pigment be represented by the symbol *C* which is dominant to the inability to develop pigment *c*, hence an individual of genotype *CC* or *Cc* can develop a colour if the factor for that colour is also present, but an individual of genotype *cc* cannot develop a colour under any circumstances. Let the factor causing the appearance of a particular colour be represented by *R* which factor is dominant to the factor *r* which is unable to cause the appearance of the colour in question.

Now if one parent be of genotype *CCrr* (phenotype white, because although the developer factor is present, there is no pigment factor present to be developed) and the other be genotype *ccRR* (again phenotype white, but this time because pigment factor present cannot be developed owing to the absence of the developer,) the law of segregation shows the F_1's will all be genotype *CcRr* (phenotype coloured). Now when the F_1's are self-fertilized we get an F_2 generation—as we see from the accompanying Punnett square (Figure 4)—composed of "coloured" and "white" in the ratio of 9:7.

We are now right away from any semblance of a 3:1 ratio but we can still explain our experimental findings as shown above using just the same laws as before. This type of inheritance will most certainly prove very common in man and one character that has already been shown to react thus is that of skin colour. The skin colour of the mulatto is intermediate between that of his white and black parents. If two mulattoes marry, the skin colour of their children may vary anywhere between Negro-black and European white, and Davenport has shown that the explanation lies in the fact that skin colour depends on a number of factors whose identity he has established and by means of which the actual findings can be adequately explained. Thus we see

that blending inheritance can not only be caused by incomplete dominance (four o'clocks) but also by multiple factors each pair being capable of influencing the development of the character in question.

		CcRr (coloured)			
	Gametes.	*CR*	*Cr*	*cR*	*cr*
CcRr (coloured)	*CR*	*CCRR* (coloured)	*CCRr* (coloured)	*CcRR* (coloured)	*CcRr* (coloured)
	Cr	*CCRr* (coloured)	*CCrr* (white)	*CcrR* (coloured)	*Ccrr* (white)
	cR	*cCRR* (coloured)	*cCRr* (coloured)	*ccRR* (white)	*ccRr* (white)
	cr	*cCrR* (coloured)	*cCrr* (white)	*ccrR* (white)	*ccrr* (white)

Figure 4. A chart showing how the F1 self-fertilization in the white sweet pea may result in coloured and white flowered plants in the proportion of 9:7.

Under the heading of multiple factors, we should also mention modifying factors. In the investigation of the eye colour of *Drosophila melanogaster*, Morgan and Bridges have ascertained that the 'eosin' colour may be modified by 6 or 7 other factors, some intensifying it and others diluting it. In fact just as we shall endeavour to show later that dominance is not absolute but merely relative, so the idea that a certain character exhibited by the individual is due to the presence of one factor, would be more accurately expressed if we said that the *most obvious* effect of that factor was to produce that certain character. In many cases 'most obvious' implies 'to the exclusion, as far as we can ascertain, of all other effects', but in some cases e.g., eosin eye colour, it allows of the conception of modifiers, or to quote Morgan (31) "It cannot too insistently be urged that when we say a character is a product of a particular factor, we mean no more than it is the most conspicuous effect of the factor."

Reversible Dominance.

Another cause of departure from the 3 : 1 ratio is the phenomenon of reversible dominance. It is clear from what we have already seen that the terms dominant and recessive are not as clear cut as might have originally been supposed. In the case of tall and short peas, if we arbitrarily assume complete dominance to be represented by 100% and complete recessiveness by 0%, the dominance of "tall" over "short" must be represented by 100%, but in the case of the red and white four o'clocks, the dominance of "red" is but 50% as is also the recessiveness of "white", i.e., neither character is definitely dominant nor recessive. In the case of the ebony and sooty wings of the fruit fly, the dominance varies. If this variation were relatively small, let us say from 80% to 90%, we could still say that the character is dominant, but if it were to vary say from 90% to 10%, then in some cases at least its dominance has been reversed. We do not understand completely the mechanism of this change, but we do know that in some cases external environment can cause it, and in others some particular combination of genetical factors seems to have some definite effect. (For further discussion on the effect of environment, see Chapter VIII.)

Tennent (43) has shewn that the dominance of some characters in the echinoderm may be changed to recessiveness by altering the chemical nature of the sea water. Again in the normal *Drosophila melanogaster* the abdomen has a series of black bands arranged with a definite regularity, but there is another race in which the regularity of these bands may be interfered with, the bands in some cases being actually broken up and only represented by scattered patches of pigment. If these latter flies are kept in the ordinary moist environment with plenty of fresh food, their abnormal condition persists, but let the food and moisture be curtailed and the bands will become less and less abnormal until at last they are indistinguishable from the normal flies.

In the cross between these two races under normal moist conditions all the F_1 individuals are abnormal. We would therefore infer that the abnormal condition was dominant to the normal one. But let the culture dry and we find all the abnormal F_1's change to normal ones. Now we should say that the normal was the dominant character. Which statement is correct? To be as accurate as possible our answer should be that under moist conditions "abnormal" is relatively dominant, whereas under dry conditions it is relatively recessive. Further work has definitely proved that this change is not brought about by any action of the environment on the factor itself. The factor remains unchanged but what is changed is the expression of the character which it causes to appear in the body of the fly.

If such a phenomenon is known to occur in other animals, we should not be surprised to find examples of it in human work, nor should we

look upon the strange ratios it produces as evidence of the inapplicability of genetics to the human organism. They may possibly be the explanation of some of the contradictory statements one already comes across in medical writings as to the dominance or otherwise of certain supposed disease causing factors. Such cases would however have to be very thoroughly investigated before such an explanation could be definitely accepted. The trouble is that in our disproportionate medical training we have been left with the idea that the phenomenon of dominance is one of the fundamentals of Mendelism. It is not. It is a very handy term to use in the elucidation of data, and is only accurate when we remember the reservation we made about the way it may be affected by environment and the fact that it is merely a relative attribute applied to characters that depend on factors which (and this is much more important) always behave according to Mendel's laws. *Dominance is a phenomenon* displayed *by characters of individuals (phenotypes) resulting from and depending on the reaction between genetical factors (genotypes) and the environment, a phenomenon which rightly belongs therefore to the study of phenogenetics.* And here attention might well be drawn to the common use of the terms "dominant factor" and "recessive factor." A *factor* is neither dominant nor recessive, and the use of such terms is only permissable when it is understood that they are abbreviations for "a factor which results in the appearnace of a dominant character" or "of a recessive character" as the case may be.

Lethal Factors.

Morgan and his associates have shown that in the *Drosophila melanogaster*, there are a number of factors which if not balanced in their pairs by a normal factor, cause the early death of the individual. That means to say that only the individuals possessing the normal factor survive, the homozygous lethals all die. These factors greatly complicate the ordinary Mendelian ratios that we have been considering, because only a proportion of the possibilities survive for examination and incorporation in the data. An excellent example of the way in which lethal factors effect ratios is given by Baur's work on snapdragons with yellow leaves. Self-fertilization of these plants gives rise to two types in the next generation, one type with yellow leaves and one with green, in the ratio 2 : 1. The green leaved plants breed true, but the yellow ones give yellow and green leaved plants in the ratio of 2 : 1 as before. None of the yellow leaved plants breed true. The explanation lies in the fact that 25% of the plants arising from self-fertilization of the yellow plants fail to survive as they are homozygous for yellow. Had they survived, the ratio of yellow to green would be 3 : 1 but in the homozygous condition the plant is unable to manufacture enough chlorophyll to enable it to survive and the ratios are thus altered accordingly. (Figure 5).

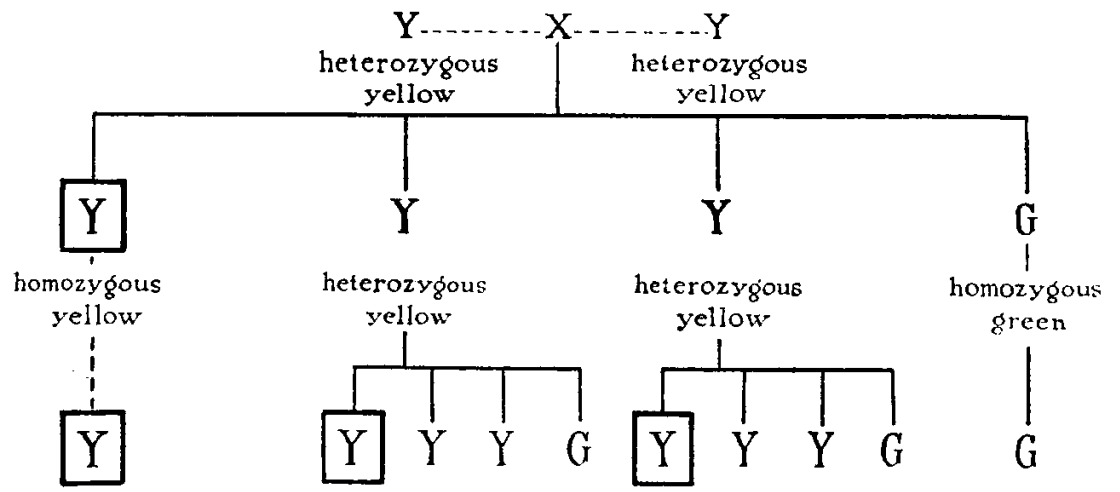

Figure 5. A diagram showing how lethal factors effect the ratios of characters appearing in a generation. Snapdragons with yellow leaves (Y) are self-fertilised and the next generation gives yellow and green leaved (G) plants in the ratio 2:1. These green leaved plants on self-fertilization breed true showing them to be homozygous, the yellows again giving yellow and green leaved plants in the ratio 2:1 proving them to be heterozygous. All the plants homozygous for yellow fail to develop (shown in the black square). Had they survived the yellow: green ratio would have been 3:1. The lethal condition of the homozygous yellow changes the ratio to 2:1.

Although we know very little about lethals in man, these factors must be very seriously considered in accounting for misconceptions, abortions and still-births which are otherwise unexplainable. A teacher, after a post-mortem examination of a newly born child in which nothing abnormal could be demonstrated, was once asked this awkward question, "Why did not the child live?" His reply was "We don't know. All we can say is that it just could not catch hold of, or keep hold of life." That is just it. The child did not posses the necessary genetical equipment (and therefore as we shall explain later had not the necessary internal forces) to combat the external forces it met with in its separate existance. With its mother's help and protection, or in other words, when its mother modified its environment, it could not only exist for a time but grow and develop, but without this help, existence was not possible.

It is interesting here to speculate on the manner in which lethals act, and again it should be noted that the term lethal itself is also relative. All living cells inevitably must sooner or later cease to exist, and all factors may be considered as having an effect on the rate of the onset of death either positively or negatively. Some factors, such as those involved in eye-colour, blood groups, etc., have such a relatively small effect that we cannot discern it and we say they are not lethal. But a factor that causes the appearance of susceptibility to a serious disease may be considered lethal to a certain extent in so far as, over a large number of cases, it will cause the earlier onset of death in a percentage of those cases. Next a factor such as that which causes amaurotic familial idiocy when it is present in the homozygous

recessive condition, may be considered to be a definite lethal, for of a certainty will that homozygous recessive individual die before middle age. Next come the factors which we recognise and designate as lethals, and the only difference is that they cause the onset of death before or just after birth, instead of after a long lapse of time.

It will be a long time before we can accurately assess the part played by lethal factors in human work, but it is a point we must constantly bear in mind when trying to explain the peculiar percentages human statistics give us.

CHAPTER III.

GENETICAL RATIOS REVEALED BY THE LAW OF INDEPENDENT ASSORTMENT.

In the last chapter we considered Mendel's first law and attempted to show how recent work has accounted for the many variations from the 3 : 1 ratio which we encounter. In this chapter the same line of argument will be followed in dealing with the other genetical law of Mendel.

Mendel's second law is that of independent assortment of factors, which simply stated, says that where one is considering two races which differ in *two* characters, each character being dependent on a pair of factors as before, but each pair being totally independent of the other pair, then each character will appear in the F_2 generation according to the 3 : 1 ratio in absolute independence of the other character.

Inheritance of Two Separate Characters Each Dependent on a Single Pair of Factors.

Mendel's experiment here involved the crossing of one race of peas characterised by round and yellow seeds, with a race yielding wrinkled and green seeds. The two characters therefore involved the shape of the seeds on the one hand and the colour of them on the other. Using a similar argument to that in the last chapter, we see that one race possessed the characters for round shape plus yellow colour, and the other race those for wrinkled shape and green colour. The F_1s were all found to be round and yellow, but the F_2 generation did not produce peas of two phenotypes only in the proportion of 3 : 1, but there appeared four phenotypes in the proportion of 9 : 3 : 3 : 1. The first group was composed of peas all both round and yellow, the second round and green, the third wrinkled and yellow, and the fourth wrinkled and green. If we examine this in the light of each character separately, we find in the F_2 generation there were 12 round peas to every 4 wrinkled (= 3 : 1), and 12 yellow to every 4 green (= 3 : 1), so that this 9 : 3 : 3 : 1 ratio is merely an extension of the old 3 : 1 ratio to fit the case of inheritance of two independent characters. If we

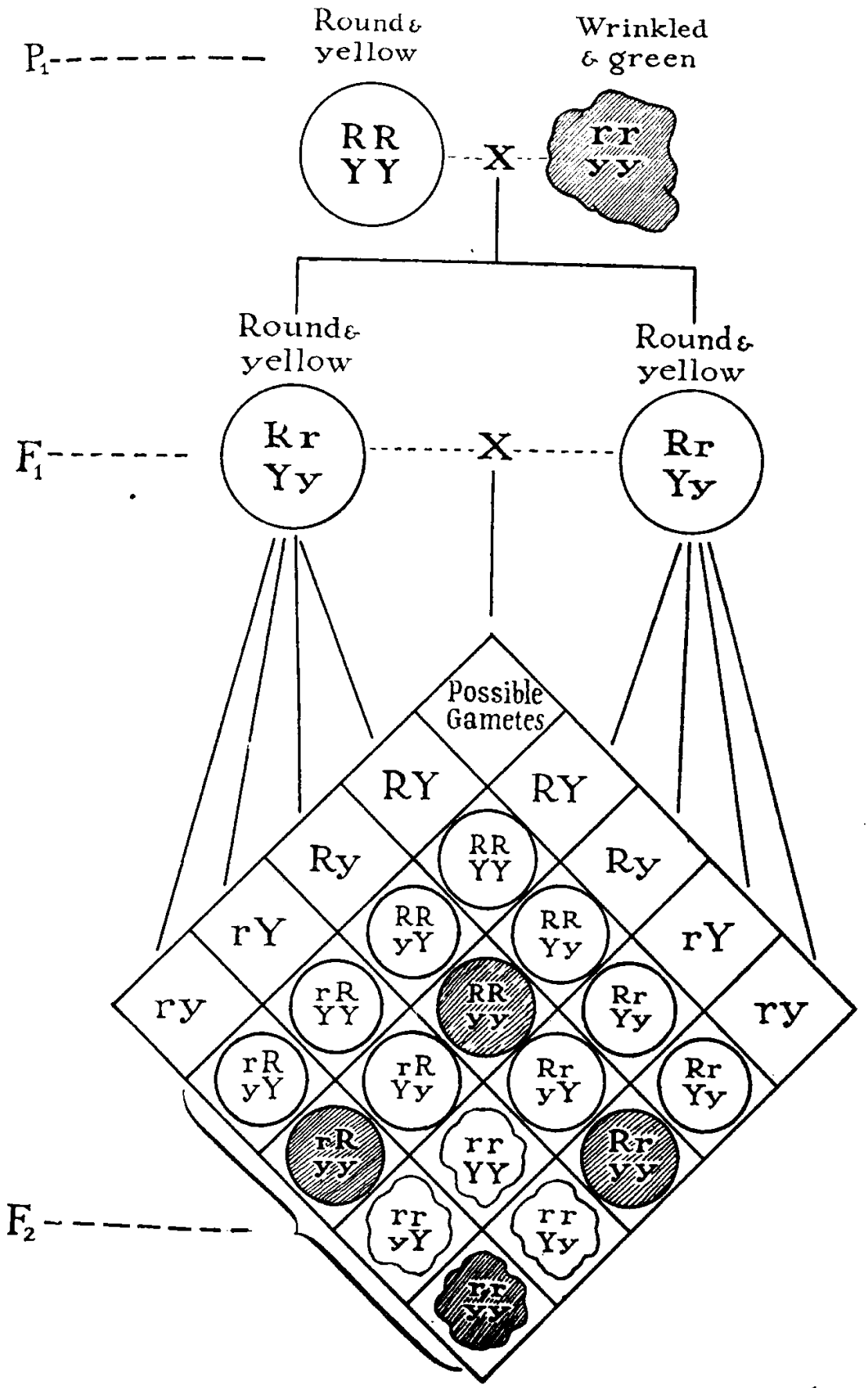

Figure 6. A chart showing the result of crossing a member of a pure race of peas yielding round and yellow seeds (*RRYY*), with another from a pure race yielding wrinkled and green seeds (*rryy*). The F1 individuals are all round and yellow (*RrYy*). heterozygous for each pair of factors. The F2 generations shows round and yellow, round and green, wrinkled and yellow and wrinkled and green in the ratio of 9:3:3:1.

represent the round factor by *R* and its allelomorph wrinkled factor by *r* (the round character being dominant to wrinkled), the yellow factor by *Y* and the green by *y* (yellow character being dominant to green), this experiment may be diagramatically represented as shown in Figure 6.

Here we should like to stress the point that no only is the appearance of four phenotypes in the F_2 really quite compatible with the 3:1 ratio, but that this appearance shows that each pair of factors segregates indepentently of the other pair. In this connection it will be to our advantage to consider the example of two independent pairs of factors given by Von Dungern and Hirszfeld in their theory of inheritance of the blood groups. *Although this theory is no longer accepted as the correct one, yet it is described here because of its importance in the development of our knowledge of the inheritance of blood group characters.* Here one pair of factors *A* & *a* was considered to control the appearance of the agglutinogen "A" on the corpuscles, and the presence of agglutinogen "A" being dominant to its absence, the genotype of the recessive condition was represented by *aa*, while that of a subject exhibiting the "A" agglutinogen was either *AA* or *Aa*. Similarly a pair of factors *B* & *b* was assumed to control the presence of the "B" agglutinogen, the genotype of a person having the "B" agglutinogen being either *BB* or *Bb*, and the genotype *bb* resulting in the recessive absence of the "B" agglutinogen.

A group O person was therefore considered to be of genotype *aabb*, not possessing either agglutinogen. Such a condition is often described as being *nulliplex*, i.e., it exhibits only negative characters of two independent character pairs.

A person belonging to group A might be either homozygous or heterozygous for the *A* factor, the genotypes being in the former case *AAbb*, and in the latter *Aabb*. In each case the agglutinogen "A" appears but "B" does not.

Similarly a person belonging to group B might be either homozygous or heterozygous for the factor *B* in which case the two possible genotypes are *aaBB* and *aaBb*.

A person belonging to group AB might be (*a*) homozygous for both factors (genotype *AABB*) and such a condition is often described as being *duplex*, i.e., each gamete has contributed similar dominant factors to the zygote: (*b*) homozygous for the dominant factor *A* and heterozygous for *B*—genotype *AABb*, (*c*) homozygous for the *B* factor and heterozygous for the *A*—genotype *AaBB*, or he may be (*d*) heterozygous for both pairs of factors—genotype *AaBb*. In this last case the condition is described as *simplex*, the zygote getting a different factor from each gamete thus having a single dose of each dominant character. The foregoing is well depicted in the table given below. (Figure 7).

PHENOTYPE.	GENOTYPE.	REMARKS.
Group O	*aabb*	*Nulliplex*. Homozygous recessive for both pairs of factors.
Group A	*AAbb*	Homozygous for both pairs of factors, the condition being dominant in respect of the A pair, and recessive in respect of the B pair.
	Aabb	Heterozygous in respect of the A pair, and homozygous recessive in regard to the B pair.
Group B	*aaBB*	Homozygous for both pairs of factors the condition being recessive in regard to the A pair, and dominant in regard to the other pair.
	aaBb	Homozygous recessive for the A factor pair and heterozygous for the B pair.
Group AB	*AABB*	*Duplex*. Homozygous dominant for both pairs of factors.
	AaBB	Heterozygous for the A pair, and homozygous dominant for the B pair.
	AABb	Heterozygous for the B pair, and homozygous dominant for the A pair.
	AaBb	*Simplex*. Heterozygous for both pairs.

Figure 7. A chart showing the possible genotypes for each phenotype according to the old (and now disproved) theory of Von Dungern and Hirszfeld.

A further consideration of this theory will be given in a later chapter, but a complete understanding of the fore-going is necessary before the argument concerning the true method of blood group inheritance can be fully appreciated.

It is clear that the 9 : 3 : 3 : 1 ratio is really no departure from the original 3 : 1 ratio but merely an application of the latter to the case where we are considering two pairs of independent factors. Just as when considering the 3 : 1 ratio we found that the reversible dominance, lethal factors, etc., may affect the ratio, so we find the 9 : 3 : 3 : 1 ratio may also be modified as shown by the phenomena of Cryptomery and Epistasis; in the former phenomenon the genes involved are not absolutely independent, but one modifies the ability of the other to express itself and here the ratio 9 : 3 : 4 appears. In epistasis one gene has the power to inhibit completely the phenotypical expression of the other, the former being epistatic, the latter hypostatic, and the ratio becomes changed to 12 : 3 : 1. Concerning cryptomery, it is interesting to note that Sanders (41) has recently come to the conclusion

that this phenomenon is concerned in the causation of the rare condition of periodic paralysis and muscular atrophy found to occur in a family reported by Biemond and Daniels.

Cytological Evidence.

Before passing to more recent work, we must here consider whether there is any cytological evidence in favour of the laws cited above. With this part of the work, medical men are all well acquainted, so we will content ourselves with merely mentioning the stages in the line of argument rather than supplying a detailed explanation as we have done in the case of the genetical factors.

The only connection as we have seen between one generation and the next is the germ cell, which therefore must be the vehicle for the transport of the genetical factors. In the female germ cell or ovum we have present the nucleus with its contained nucleolus, chromatin and achromatin material and the cytoplasm. In the male cell in most cases however the cytoplasm is reduced to a minimal amount (hardly detectable) around the head and the middle piece of the sperm and in the tail. The head of the sperm is almost completely composed of condensed chromatin material with, in certain cases a centrosome at its base. It is generally believed that at fertilization only the head of the spermatozoön enters the egg, therefore the cytological entities that may be involved in the handing on of factors from one generation to the next are the large amount of cytoplasm and the nucleus (including their component parts) in the ovum, and the nucleus and a minute amount of cytoplasm in the sperm.

With the exception of a few special cases to be cited and explained later, we do not in general find any difference in results depending or whether a certain character enters into the cross from the male or female side, and it is therefore assumed that the method of transference must be the same in both male and female sex cells. The great inequality of female and male cytoplasm entering into the formation of the zygote has therefore caused attention to be turned away from it towards the nuclei in search of the factor carriers. Cytological investigation has shown that the chromatin material in the nucleus is the only substance which can be seen to remain constant all through successive cell divisions; even the nucleoplasm appears to be completely mingled with and lost in the cytoplasm after the disappearance of the nuclear membrane, and even the spindle fibres disappear during resting stages of the cell. Only the chromatin remains permanently recognisable in various cell stages and further since this material regularly condenses into individual chromatin rods of constant shape, size and number for each species, this material has for some time generally been considered to be the vehicle for storing and transmitting the genetical factors. These chromatin rods are known as *chromosomes*

and may be considered as being made up of structural units which are now called *genes,* which genes are supposed to be the carriers of the factors we have been considering. This term now universally used, supplies the anatomical background necessary for the physiological idea of the word *'factor,'* and hence they are often used synonymously, a loose habit which always leads to unnecessary complications. Johannsen first introduced the term *gene* in order to describe how monohybrids differed from one another; their difference was a single *gene* but he refused to recognise the material basis underlying this difference. It was Morgan who later visualised the material structure as a *gene,* a conception which not only has the advantage of being more easily understood but which also fits in well with recent genetical advances. With this explanation there should be no difficulty of understanding the meaning of the word we have already used a great deal—*'genotype,'* which term along with *'phenotype'* was also suggested by Johannsen.

Cytological and embryological evidence is almost overhwelmingly in favour of the chromosomal theory of heredity. The constancy of the chromosome number, size and shape in successive generations we have already mentioned. Their occurrence in pairs fits in with Mendel's idea of paired factors; their conjugation seen to take place after fertilization would allow of the chance mixing of factors, and the separation of each pair into single chromosomes provides the occular means of demonstrating the method by which the fundamental assumption of Segregation of Factors may be carried out; the halving of the chromosome number during the reducing division of the germs cells not only fits in with the theory that only one of a pair of factors passes to each germ cell, but it also explains why the total number of chromosomes in each succeeding generation is not doubled. If $2n$ be the number of chromsomes in the cells of one parent, then the number in the gamete is n, and this when it combines with the n of the other gamete results in the zygote having again the same number of chromosomes ($2n$) which each of its parents possessed.

Cytoplasmic Inheritance.

We have seen that the zygote receives its cytoplasm to a small extent from the paternal gamete and to a very much larger extent from the maternal gamete. In any theory of heredity the possibility of the cytoplasm of the germ cell conveying some of the hereditary characters from one generation to the next must be considered; if this were the case one might reasonably expect the maternal influence to greatly outbalance the paternal. In general we have seen that this is not so, but in certain cases it may be so. Conklin (7) came to the conclusion that "the general polarity, symmetry and pattern of the embryo are determined by the cytoplasm of the egg" but although later these views

were modified, one cannot but realise that this cytoplasmic inequality is an ever present volcano which, at any time, new evidence may prove to be active rather than extinct or passive. In fact recent work of Little (27) on the inheritance of cancer in mice has reawakened the slumbering genetical world to the possibility of activity in this direction.

But apart from that, the importance of this new work, if it is substantiated, will not be that it will disprove Mendelian inheritance as we now know it, but rather that it will fill in some of the gaps in our knowledge which Mendelian theories have not been capable of filling, gaps which are causing medical men to hold back their whole-hearted acknowledgement of the importance of genetical principles in relation to human inheritance.

From this rapid and by no means detailed consideration we can see that the cytological evidence, while being almost wholly in favour of it, does not by any means prove the chromosomal theory of heredity. The proof, if we can yet say that we have any such thing, remains genetical, and the importance of this genetical proof lies not so much in the fact that it agrees with existing laws and theories, but that it serves to supply, not only an explanation of apparent deviations from these laws, but also of those new laws of chromosomal heredity which have placed the science of genetics on the solid foundation it occupies to-day. We shall now turn our attention to this new work.

(To be Continued)

THE PERSISTANCE OF TRANSFUSED RED BLOOD CORPUSCLES IN THE HUMAN CIRCULATION.

by

LINDSAY RIDE & LING KE DIEH

from

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During blood investigations at present being carried out in this department, it occurred to one of us (L.T.R.) that the phenomenon of hetero-haemagglutination might provide an excellent opportunity for investigating the length of life of human red cells and in this paper we intend to record the application of this method to two cases and the results.

In 1928 Landsteiner and Levine (5) recorded their findings that in addition to the agglutinogens *A* & *B* whose presence on red cells is demonstrable by the ordinary iso-haemagglutination technique, there also existed three other agglutinogens on human red cells which they named M, N, & P. Among other serological and genetical differences these latter differ from A & B in that they are hetero-agglutinogens i.e., specific agglutinins for M, N, & P do not occur naturally in human serum and hence their presence can only be demonstrated by specially prepared immune sera. The preparation of these sera are adequately described in papers by Landsteiner and Levine (5) and by Wiener (11).

Since these new substances are of the nature of hetero-agglutinogens they do not concern us in the ordinary operation of blood transfusion but in the method about to be described it will be seen that they enable us to detect in the blood drawn from a patient the actual corpuscles which have been transfused from another human individual.

The method will be best understood from the description of the first case to which it was actually applied. A Chinese male aged 24, was admitted into the Hong Kong Government Civil Hospital, suffering from an acute abdominal condition which was later diagnosed as haemangioma of the liver. His condition ultimately necessitated a blood transfusion, and samples of his blood together with some from possible donors were submitted to us for grouping. The patient's corpuscles turned out to be type O, M−N+ that is to say iso-agglutinogens A & B, and hetero-agglutinogen M were absent from his corpuscles, but hetero-agglutinogen N was present. We recommended the use of blood from a donor of type O, M+N− and the transfusion was duly performed on 9th January 1935, using 1 pint of blood from the recommended donor. One hour after the transfusion a sample of blood was taken from the ear of the patient and on naked-eye as well as microscopic examination by the open slide method it was found to consist of two types of

corpuscles, one type (present in very large numbers) which was agglutinated by anti-N serum, and another type (present in much smaller numbers) which was agglutinated by anti-M serum. Now since the recepient's corpuscles before transfusion were proved to be *unagglutinated* by anti-M serum, and the donor's cells were proved to be agglutinated by this same serum, it was obvious that the cells from the patient after transfusion that were agglutinated by the anti-M serum were the actual cells given by the donor.

Similar tests were applied to samples of blood taken from the patient 9¼ hours, 22 hours, 45¾ hours and 57½ hours after the transfusion had been performed and in each case some of the corpuscles were found to agglutinate on addition of anti-M serum. The death of the patient made it impossible to continue this investigation any further.

Discussion of Earlier Methods.

Many methods have been used in order to try and ascertain the life span of human red cells. Indirect methods such as calculating from the haemoglobin content of the blood and the daily excretion of bilirubin give the life span as 30-40 days according to Macleod (7). Wright (12) merely states that it is believed be some authorities to be 4-6 weeks, Flack and Hill (3) state it is possibly 3-4 weeks but give no indication as to how this figure is arrived at. Starling (9) states "How long a corpuscle continues to exist in the circulating blood is not known"; Howell (4) states "How long the average life of the corpuscles may be has not been determined"; Burton-Opitz (1) after stating that the life has been estimated at 4 weeks is of the opinion that the point has not been definitely settled, and he further exposes some of the falacies in the bilirubin method. Stewart (10) states that results obtained by this method "are not sufficiently trustworthy to warrant their citation" Lovatt Evans (6) in his chapter on blood destruction also quotes this method as giving 30-40 days and also points out its limitations and further mentions a method similar to ours, namely, investigating the persistance of transfused cells belong to a different but compatible blood group of the same species. This method places the life of a red cell at 28-100 days.

We have not access to the records of this work, but we can easily deduce the experiments that must have been used and hence can discuss them now.

Corpuscles from an individual belonging to group O (universal donor) could be used for this purpose by transfusing them into the circulation of a person belonging to any of the other groups A, B, or AB. In any of these cases the demonstration of type O cells must have been by agglutinating the A, B, or AB cells and by showing a number of cells left unagglutinated. This method is open to the criticism that even in a suspension of type A, or B or AB cells alone, there are always

some cells left unagglutinated after mixing with the corresponding agglutinating serum, and therefore there is no absolute proof that the unagglutinated cells are really of type O and have come from the donor. If the recognition of the time of the disappearance of these cells was made by quantitatively estimating the time at which the unagglutinated cells become a constant minimum it must be, of necessity, inexact and would almost certainly give an exaggerated length of life of the cells. In any case the entrance of $\alpha\beta$ agglutinins of high titre into the circulation of the recipient, although diluted by the receiver's blood, may cause agglutination and consequent destruction of the receiver's own cells, and thus interfere with the normal equilibrium of the cell formation and destruction of the recipient.

The only other possible experiment is the use of type A or type B cells for transfusion into an individual belonging to Group AB. Here the same objections apply; the persistance of the transfused cells can only be inferred when some cells are left unagglutinated after the addition of β serum in the former case and α serum in the latter, and these cells cannot be distinguished from the patients own unagglutinated AB cells. These methods therefore do not allow of the actual demonstration of the transfused cells themselves, but merely allow the possible inference of their continued existance, nor do they meet the possible (though we think improbable) criticism that the transfused cells may be very short lived, and be quickly be replaced by the recipient's own cells which in their early stages may not agglutinate readily. It is a negative method and as such is open to all the criticism of negative evidence.

The direct methods usually employed are those of introducing into the human circulation, red blood corpuscles from another species, corpuscles which are morphologically distinguishable from human red cells. The objections to these methods are obvious; one is introducing foreign cells and cells which may easily be either more or less resistant than human red cells to human methods of red cell destruction. In any case this method yields evidence not on the question of human red cell longevity but on the length of life of *foreign cells in an unusual environment.*

Here we must mention an ingenious method that has been used in animals by Paul Eaton and Damren (2). They pointed out that after haemorrhage, the bone marrow responded by increased output of red cells including reticulocytes as shown by a rise in the reticulocyte count. Compared with the normal daily birth rate of red cells, the production on the days immediately following the bleeding was high and hence when the end of the life span of these cells was reached there would be a corresponding period of days of abnormal death rate, in fact another physiological haemorrhage would occur, with therefore another rise in the reticulocyte count. A series of daily reticulocyte counts revealed that

this did actually happen, the rise in the count getting smaller and smaller in each successive occurrence; after 50 days the wave disturbance in the reticulocyte frequency curve disappeared. The length of time elapsing between each rise in the reticulocyte count was taken as a measure of the average life of a red cell in the animal investigated. In a rabbit it was found to be 8½ days. We have no knowledge of whether this method has been applied to man or not, but it may well be that it has and with useful and reliable results.

Advantages of the New Method.

Its first advantage is that use is made of transfused corpuscles that are as similar as possible to those of the recipient. The donor and recipient were both males, both Chinese, both belonged to the same blood group, and therefore both cells and serum were as similar as possible. The only known and detectable difference was that one had M hetero-agglutinogens and the other N, but since no corresponding agglutinins occur naturally in the human, the result of this difference must be as far as we know negligible.

The other advantage is that it provides an accurate quantitative method which can be used to estimate blood volume during rest or exercise in normal or pathological conditions, and longevity of corpuscles as well as their rate of destruction. A necessary preliminary to this application is the proof of the fact that, when corpuscles of a certain type are agglutinated out of a mixed suspension of cells by the addition of an anti-serum, the clumps thus formed are composed of that certain type of cell only and do not include cells of any type which may be entangled in the clumps. This problem is now being investigated in this laboratory. If it can be shown that a clump contains only cells that have been actively clumped, then having ascertained the blood counts of donor and recipient before transfusion, the volume of blood transfused, and the percentage composition of the two types of cells in the recipient's circulation immediately after transfusion the volume of blood in the receiver's circulation can be estimated. If the recipient is immediately subjected to some form of exercise the increase in circulating blood volume may similarly be deduced. Reports on this aspect of the work will be published later.

Second Case.

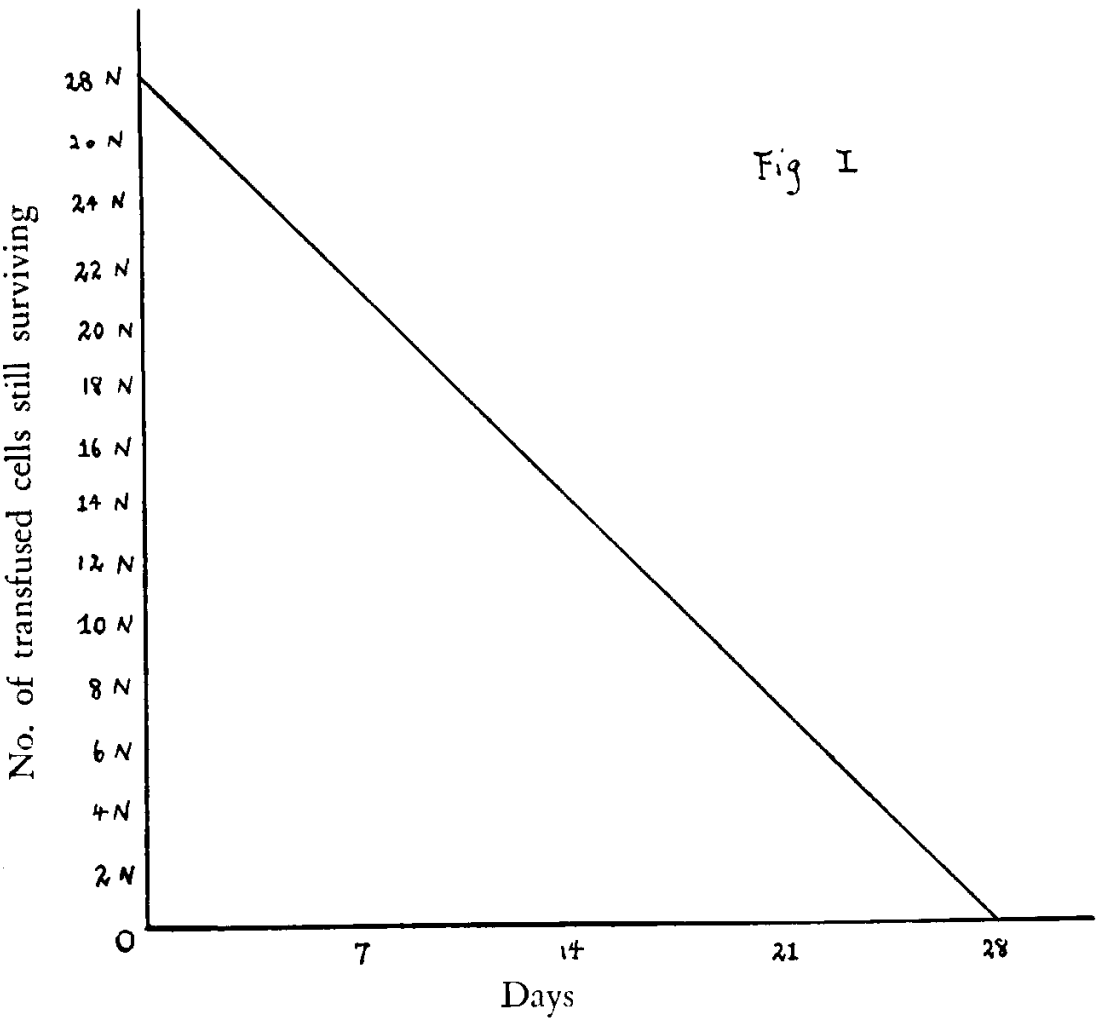
Mrs. L, a patient of Dr. E. W. Kirk required transfusion and he submitted to us a sample of the patient's blood together with samples from possible donors. The patient turned out to be type B, M− N+ and the recommended donor was type B, M+ N−. 300 ccs of blood from the recommended donor were transfused by Dr. Kirk and the patient's blood was examined at frequent intervals afterwards as shown in Table I.

TABLE I.

Blood Sample	*Time Taken*	*Agglutination Tests*	
		With anti-M serum	*With anti-N serum*
Patient's	Before transfusion	−	+
Donor's	,, ,,	+	−
Patient's	5 hours after ,,	+	+
,,	24½ hours ,, ,,	+	+
,,	48 ,, ,, ,,	+	+
,,	70½ ,, ,, ,,	+	+
,,	93¾ ,, ,, ,,	+	+
,,	6 days ,, ,,	+	+
,,	11 ,, ,, ,,	+	+
,,	13 ,, ,, ,,	+	+
,,	15 ,, ,, ,,	+	+
,,	20 ,, ,, ,,	+	+
,,	25 ,, ,, ,,	+ (faint trace)	+
,,	42 ,, ,, ,,	−	+

Discussion.—The second case shows that some at least of the transfused corpuscles survived in the recipient's circulation for about 4 weeks. The significance of this finding depends on the correlation between the rate of destruction of the red blood corpuscles and their age. There is a belief in existance—although on what evidence it is founded it is hard to say—that all the red cells live for a certain number of days and then disintegrate. If the number of red cells in the body of any one individual is constant under physiological conditions, and if the above belief be true, the cells living for *n* days, then every sample of blood taken at random from that individual will contain *n* equal-sized age-groups of cells.

Transfused blood forms just such a random sample of a donor's cells, and provided the conditions in the recipient's circulation are the same as those in the donor's (a similarity which our experimental method has tried to make as complete as possible) then each successive day should witness the reduction in the number of these transfused cells by 1/nth of their original number. A graph made by joining points corresponding to the number of surviving transfused corpuscles on each successive day would be a straight line as shown in Figure 1, and under these conditions our results would lead us to believe not only that a red cell exists in the recipient for about 28 days but also the average life of such a cell is 28 days.



There is however no evidence that human blood cells do all live for the same number of days. In fact it would be just as reasonable to suppose that red cells like any other biological entity may show a curve of survivors such as depicted by Pearl (8) for human beings. If such conditions apply also to red cells, then a random sample of blood cells will not consist of equal-sized age-groups, and equal number of cells would not be destroyed on successive days from each age group.

The only way to ascertain which of these two possibilities is correct is to make a quantitative observation on the number of transfused cells surviving each day, and if the number decreases in arithmetic progression then the interpretation of our results would be that not only do transfused cells survive in the recipient's body for about 4 weeks but that 4 weeks is also the average life of a red cell. If the number does not decrease in this manner, then our conclusion must merely be, that red cells existed after transfusion for a maximum period of about 4 weeks, and the average life of such cells would have to be calculated from the survival curve obtained.

SUMMARY.

1. A method involving the use of the hetero-agglutinogens M & N of Landsteiner, is described whereby the maximum period of survival of transfused red cells in the circulation of a recipient may be accurately determined.

2. Earlier methods used in the estimation of the life of red cells are discussed, and the advantages of the new method detailed.

3. Two cases are described in which the new method was applied, and in the second case a few of the transfused corpuscles could be demonstrated existing 25 days after transfusion.

4. The significance of these results is discussed and it is shown how the daily quantitative estimation of the number of cells surviving is necessary to deduce the average length of life of a red cell.

Our special thanks are due to Dr. E. W. Kirk, who not only allowed us to carry out these observations on his patient, but who found time during his busy practice to collect the blood samples himself and send them along to us; to Mrs. L. the patient who so readily consented to let us have the blood samples so frequently; to Prof. J. L. Shellshear for the references to the work of Eaton and Damren.

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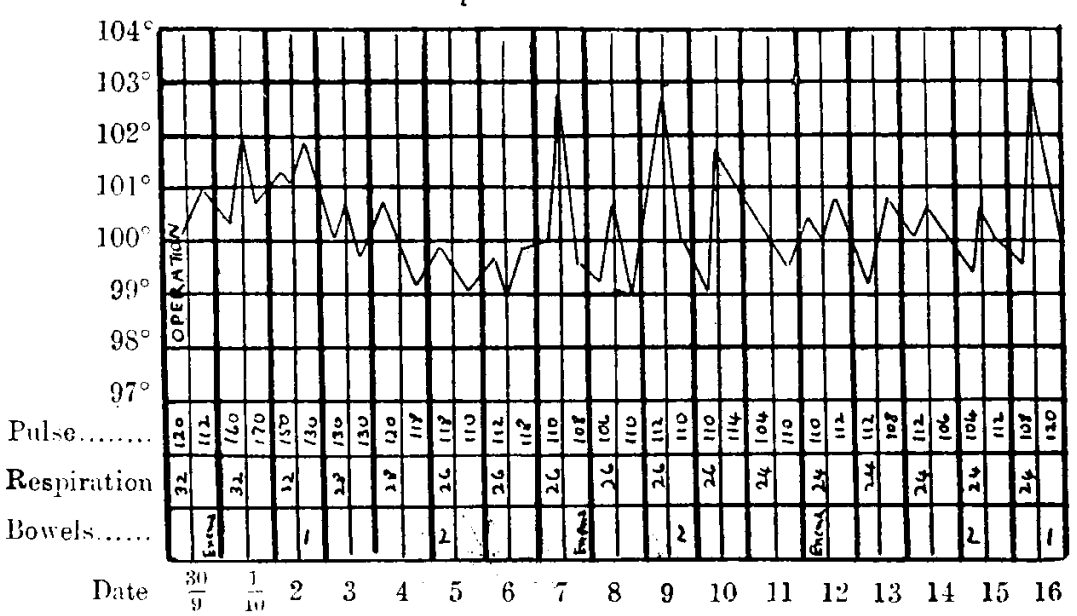
A CASE OF ACUTE INTUSSUSCEPTION COMPLICATING TYPHOID FEVER.

By DR. K. C. CHENG
& DR. F. Y. KHOO (Surgical Unit).

Diarrhoea and enteritis are stated to be the commoner causes of acute intussusception. One can therefore imagine that typhoid fever must have accounted for quite a number of cases, although we have not been able to find any mention in any of the standard textbooks we have read. We can only find one such case on record (1) in the journals after an extensive search—possibly there might have been a few more which have escaped our notice—hence we think it might be of interest to record this case.

Temperature Chart



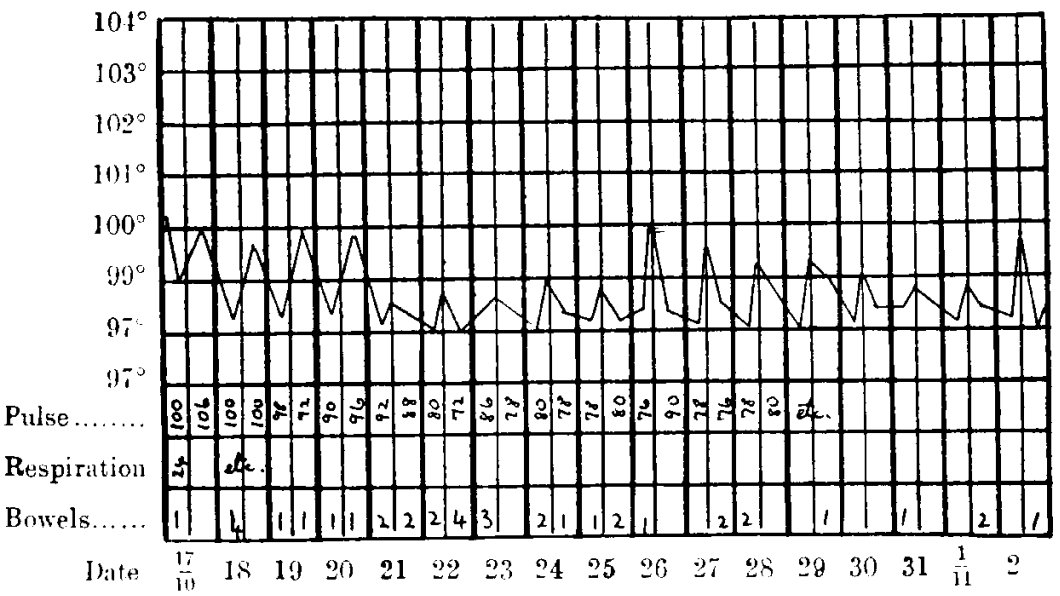
September 26th. Patient, W. K. C., male, age 5 who was previously healthy, fell ill with a temperature of 103°F. Examination showed that the heart, lungs, liver and spleen were normal. The tongue was coated and dry, and the pulse strong and rapid. He had one motion on the day of the attack.

September 27th. Morning: The abdomen was more distended; peristalsis and three very distended coils of intestine were stated to be seen. The patient vomited twice.

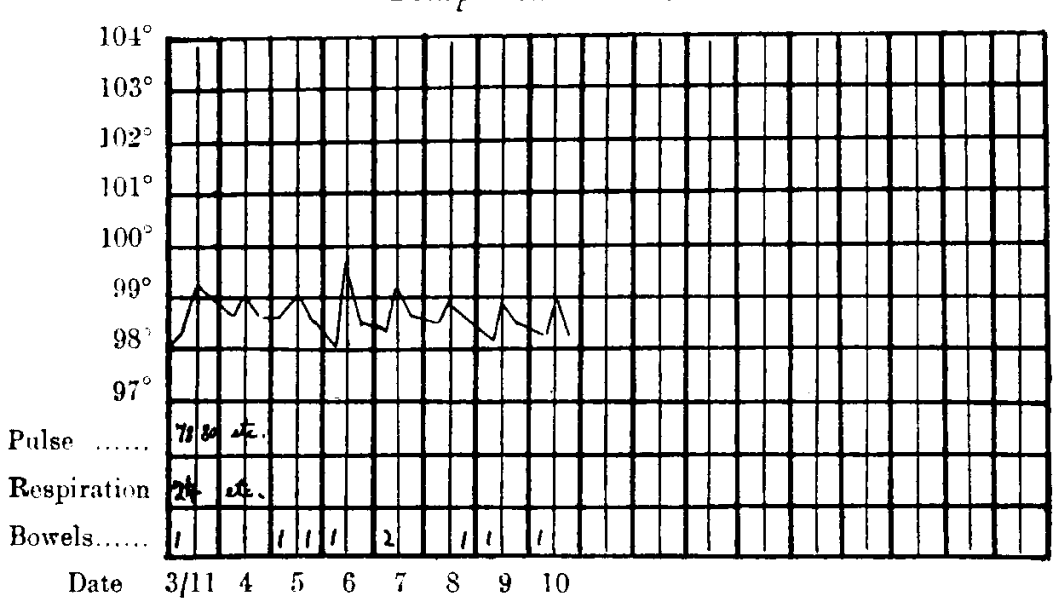
Afternoon: The patient was admitted into the Canossa Hospital. He was in a distressed condition. The eyes were sunken and the tongue moist with whitish-grey fur in the centre. The pain was chiefly to the right and above the umbilicus, in the nature of colicky attacks which

caused the patient to roll about in bed. There were distended coils of intestine, ladder pattern, at times. A rectal examination revealed faeces in the rectum—with something ? faeces, felt higher up outside the rectum.

Temperature Chart



Temperature Chart



The urine was free from albumin and sugar.

An examination of the blood showed a haemoglobin percentage of 90; a white count of 11,000; and a differential count of 85% polymorphonuclears, 12% lymphocytes, and 3% monouclears.

The following diagnoses were considered :—

Intestinal obstruction, typhoid fever, appendicitis, worms, and tuberculous peritonitis.

Later:—Visible peritalsis, ladder pattern in type, was distinctly seen. Two enemas were given—the first result was a constipated motion, and the second showed a fair quantity of blood and mucus. A revised diagnosis of intussusception was then made.

The patient was operated upon by Professor Digby and ourselves at once. Open ether was given. A mid-line incision was made, and an ileo-colic intussusception, of a fairly good colour and about 3″ long, was reduced by manipulation. The Peyer's patches were found to be inflamed, and the glands in the lower part of the mesentery were enlarged. The affected portion of the gut was sewn by a single catgut stitch to the anterior abdominal wall, and the wound was closed. Blood was taken for Widal test on the 7th. and the 14th. day after the onset of the illness. The first test was returned as negative, and the second as positive (dilution 1/100 against "O" & "H" antigens). The patient made an uneventful recovery, and the temperature settled to normal six weeks after the operation.

We are indebted to Professor K. H. Digby for his encouraging us to record this case.

REFERENCE.

(1). Intussusception following typhoid fever—excision and recovery. by A. Burhaneddin Central blatt für Chirugie.

973—976. April 16th. 1927.

Review of Books.

"WELCOME" PHOTOGRAPHIC DIARY, 1935. Burroughs Welcome and Co., 60, Hong Kong Road, Shanghai.

This little volume contains in addition to the photographic diary almost every type of information that any amateur photographer may require. The size is such as to permit its being slipped into one's waist coat pocket.

Its contents has been revised, the exposure and development factor having been brought up to date. These factors enable the amateur photographer to ascertain with ease and precision the exposure required for any one of the multitude of films and plates now available and about which the maker offers little or no data for use or for correct development.

The only criticism one might bring forward is that on many occasions only the name of Tabloid proprietary chemicals is given in the formulae. This point might disappoint those who believe in preparing every thing for themselves, though this is a procedure which is always expensive and time wasting in the hands of amateurs.

This concise photographic vade-mecum though intended for the general public is especially valuable to members of the medical profession who make photography an aid to their record of work, clinical as well as scientific and who are at the same time too busy to consult the big photographic manuals.

L. K. D.

THE CARBOHYDRATES. 5th Edition. E. F. Armstrong and K. F. Armstrong. 15/- Net. Longmans Green and Co., Ltd.

The publication of this monograph completes the revision of a work which has long been familiar to chemists under the title of "The Simple Carbohydrates and the Glucosides." The original monograph has been divided into two volumes, the first of which "The Glucosides" appeared in 1931.

The authors explain that the reason for the delay in the publication of the companion volume was due to a desire to be reasonably certain that the newer conception of the structure of the sugars had gained general recognition.

During the last eight years, the pyranose and furanose ring structures may be said to have been definitely established, this being largely due to the work of Haworth and the Birmingham school.

The present monograph gives an authoritative and up to date review of sugar chemistry, without, however, entering into discussions of intricate structural problems.

The earlier chapters deal principally with hexoses, then follow chapters on the pentoses, the carbohydrate alcohols and inositol.

The authors use the term "Oligosaccharides" originally proposed by Freudenberg to include the di-, tri- and tetrasaccharides and to differentiate them from the polysaccharides.

The two final chapters deal with the relation between configuration and biological behaviour, and the synthesis of sugars in the plant.

A perusal of this work enables the reader to realize the great advance in our knowledge of sugar structure upon the foundation so well and truly laid by Emil Fischer. Though much has been done there still remains much more to do.

For example the usual explanation of the mutarotation of glucose as due to the establishment of an equilibrium between the α and β forms cannot be considered as adequate when it is realized that an aqueous solution of glucose probably consists of α and β glucopyranose α and β glucofuranose together with the aldehydic form of the sugar. The laboratory synthesis of sucrose has yet to be achieved and our knowledge of the chemistry of the polysaccharides is only now assuming a concrete form. This book is essential for chemists and others who desire to keep in touch with the recent numerous developments in sugar chemistry. This especially applies to those in the Far East, to whom the original sources may not be readily accessible.

The biochemical importance of the sugars has been emphasized throughout the work, thus making it of special value to biochemists. At the end of each chapter there is a useful list of references.

It is impossible to conclude the review of this book without referring to the tragic death of Mr. K. F. Armstrong a few months ago while on holiday. He was a young man of the third generation of a renowned family of chemists and as he was already worthily maintaining the family tradition, his untimely death is an irreparable loss not only to his parents but to the profession of chemistry.

G. T. B.

"A SHORT PRACTICE OF SURGERY."

It is a pleasure sometimes to turn from large multivolumed systems of surgery to a shorter and more vivid presentation of the subject. The authors have set out to present the essentials of surgery in a bright and attractive manner and to include "only such material as is completely up to date and yet generally acceptable". There is also for the most part a good sense of proportion so often wanting in writers of surgical

text books. We are glad to see advanced surgery and rare conditions relegated to small type in a work of this kind.

Bacteriology is wisely condensed. Perhaps in such a compact account, the date of discovery of the gonococcus might have been omitted.

On the other hand one is pained to find that "asepsis" and "antisepsis" do not appear in the index and that "Lister" is only indexed for his metal sound. A section on the general question of shock should have been included. After all it is one of the three great bugbears of the surgeon.

On the other hand the romantic little tit-bit about the use of the "larang" in China might have been blue pencilled. It is certainly news to us in Hong Kong, and we feel that it is an undeserved slight upon the intelligence of murderers in China. Why use a distinctive weapon with a Malay sounding name to kill a person with enlarged spleen when a blow with a blunt instrument or even the hand will do so as effectively and far more secretively?

Such criticisms are not excessively condemnatory. To compress a large subject and yet leave it readable and interesting is bound to provoke adverse comment from some one or other.

This reviewer has a more serious quarrel with a paragraph on page 347 which appears to include just one of those "time honoured shiboleths" which the Preface claims are excluded:—

"The vermiform appendix is present only in man, certain anthropoid apes, and the wombat. Morphologically it is the undeveloped distal end of the large caecum found in many lower animals, just as the caecum of these animals is but a rudiment of the comparatively enormous caecum of herbivora". (And this to medical students who have presumably dissected a rabbit in their biology class!)

One would like this and the following paragraph to be rewritten to read:—

"In nearly all mammals, the apex of the caecum contains a mass of lymphoid tissue. Sometimes this forms a caecal tonsil. At other times it occupies the wall of a highly specialised tube the vermiform process or appendix. This is well developed in man, certain anthropoid apes, some marsupials, most rodents, and a few ungulates, less developed in some others. In the entirely herbivorous rabbit, it reaches its maximum complexity."

"The vermiform appendix, like the other sub-epithelial lymph glands, such as Peyer's patches and the faucial tonsils, probably plays a part in immunising the individual against bacteria in the lumen. But the very nature of its work in ingesting bacteria renders it liable to damage, whereupon its protective nature is converted into a menace.

The plentiful occurrence of other subepithelial lymphoid tissue permits of its removal without obvious ill-effect."

Objection may also be taken to a statement on page 797 anent Pott's fracture. "In some cases the internal malleolus projects through the skin." Surely this should read, "In some cases the sharp edge of the tibia, from which the medial malleolus has been fractured projects through the skin."

In spite of these grumbles we welcome this work which is sure to be very popular with students. The clear forceful dogmatic expression supported by excellent diagrams drives home its teaching; though just occasionally (as is not uncommon with such teaching), this is at the expense of strict scientific accuracy.

We love the coloured picture of an exploding appendix!

Title of Book:— "A Short Practice of Surgery". 2nd. Ed. (in one volume).

Authors:— Hamilton Bailey, F.R.C.S. (Eng.).
R. J. McNeill Love, M.S. (Lond.), F.R.C.S. (Eng.).

Pages:— VIII—988.

Illustrations:— 731 (83 coloured).

Size:— Demy 8vo.

Price:— 30s. net.

Publishers:— H. K. Lewis & Co., Ltd.,
136, Gower Street,
London, W.C.1.

K. H. D.

"MEDICINE IN ITS CHEMICAL ASPECTS", Reports from the Medico-Chemical Research Laboratories of the I.G. Farbenindustrie Aktiengesellschaft. Vol. II.

The first volume of the series of essays and reviews published under this heading, was noticed in the February number of this journal for 1934. This second volume is dedicated to Dr. Horlein on the occasion of the twenty-fifth anniversary of his joining the firm of Bayer & Co., of whose research laboratories he is now director. The volume consists of forty chapters reviewing recent advances in the many diverse fields in which chemical, and chemico-physiological methods have been successfully applied to the elucidation of medical problems. Each chapter is by a member of the Bayer staff who has been actively engaged in the particular field of research dealt with. The whole forms a fitting tribute to the effectiveness of the Bayer research organization and to the ability of its director.

L. J. D.

Acknowledgements

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