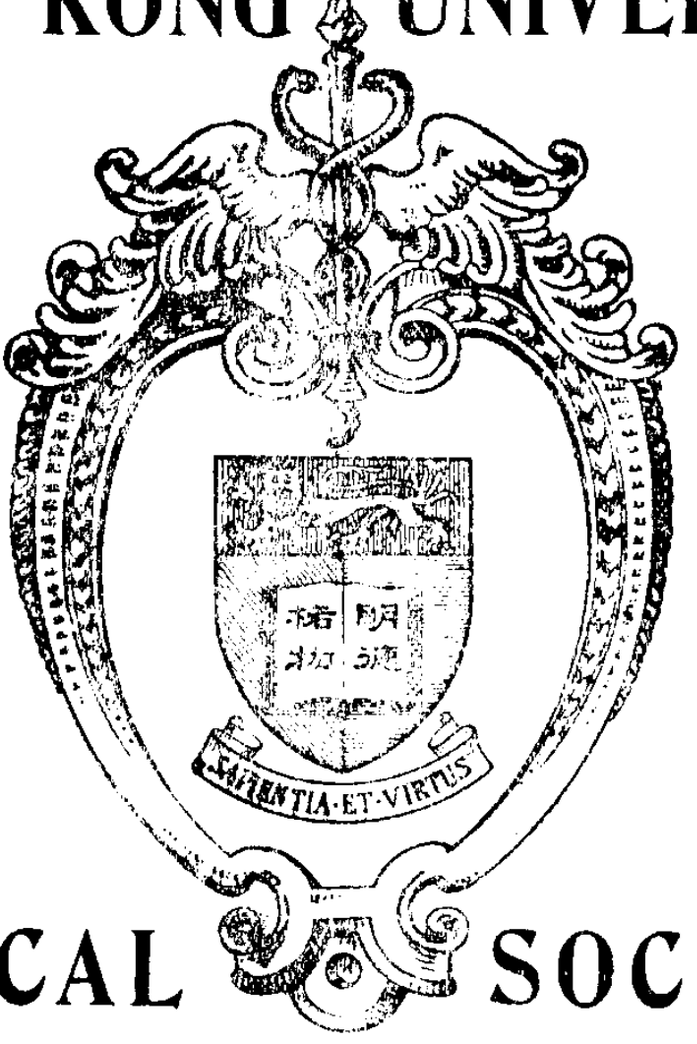
THE CADUCEUS

JOURNAL OF THE HONG KONG UNIVERSITY MEDICAL SOCIETY



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Changes of address of members of the Society and all business communications should be sent to the Business Manager, "*Caduceus*," Hong Kong University; Hong Kong.

OBITUARY.

The death of Professor C. Y. Wang early on the morning of the 15th December, 1930, has removed from the University one of its outstanding personalities and bereft an able family of one of its ablest sons.

Dr. Wang started what was destined to be a distinguished academic career at Queen's College, Hong Kong, and later passed on to the Hong Kong College of Medicine from which College he qualified with honours, in 1909. He then went to Edinburgh, and in the following year qualified for the honours degree of M.B., Ch.B. of that University. He continued his studies there for another year after which he passed the examination entitling him to the Diploma of Tropical Medicine & Health. In 1912 he took the B.Sc. degree of the Manchester University and then returned to Edinburgh to undertake some research in Pathology and Bacteriology at the Royal College of Physicians. For this work he was awarded the Freeland Barbour Fellowship of that institution and on presentation of a thesis at the University he was awarded his M.D. degree along with the University's Gold Medal. During the war Dr. Wang was Assistant Superintendent in the Bacteriological Department of the Edinburgh University, holding both the McCann and the Carnegie Research Scholarships. His work during the period was later duly recognised when he was appointed a Fellow of the Royal College of Physicians of Edinburgh.

On the 18th May, 1920, Dr. Wang was appointed Professor of Pathology in the Hong Kong University, and thus was forged a happy link binding the old Hong Kong College of Medicine and the new University's Medical Faculty.

From his appointment till his death, the late Dr. Wang was this University's Professor of Pathology and the manner in which his work was done is evidenced by the esteem in which he was held not only by all his students but his colleagues and fellow practitioners. Nor was his task a light one. Professor Wang had to build up a department and at the same time endeavour to impart a standard of knowledge equivalent to that of the home universities, to classes just as large as those at home, but with much less assistance and infinitely less equipment. With all this, Professor Wang found time to publish " A Handbook of Pathology " the merit of which is proved by the fact that it has become a standby to students in other medical schools besides our own.

After his return from long leave early in 1930 it soon became obvious to his colleagues and medical friends that Professor Wang was not well. Yet with that strength of will with which he was so liberally endowed he refused to give in. Even after being forbidden to use his voice, he remained at his post, communicating to his staff by paper, and instructing his pupils by typing lectures and having them read in class. The end of the summer however found the unequal struggle producing its inevitable results. In October Professor Wang unwillingly had to give up active work, and in spite of all that medical skill could do, mid-December saw his peaceful passing.

It is beyond the power of this pen to adequately depict the esteem in which Dr. Wang was held by student and teacher, layman and physician, Chinese and European. To his wife and children and his brothers, we offer our deepest and sincerest sympathy. His was a medical life which, begun in our own College of Medicine and nourished in the nursery of Edinburgh's laboratories, flourished to the full in its own native school, and which, at the time of full fruition, was taken.

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.

by

Prof. R. E. Tottenham.

Drs. D. K. Pillai, S. K. Lam and Miss P. C. Lai.

In December 1929 Professor Tottenham went home on leave, and since then the work of the Department has been conducted under the care of Dr. Pillai.

During the year from May 1929 to April 1930 the number of patients admitted was 1778 of whom 1616 were delivered.

Mortality Rate :—

Tsan Yuk Hospital	.17%
Government Civil Hospital	.73%
Total Mortality rate...	.45%

The causes of death were as follows :—

Post Partum Hæmorrhage	1
Shock...	1
Hydatid mole...	1
Accidental Hæmorrhage and Eclampsia ...	1

Morbidity Rate :—

Tsan Yuk Hospital	5.7%
Government Civil Hospital... ...	5.0%

Cæsarean Section Rate :—

Out of a total of 1616 deliveries Cæsarean section was performed once.

Operation Rate :—

Repair of lacerations excluded one operation to every 28 patients approximately.

OBSTETRICAL OPERATIONS.

The number of obstetric operations performed during the year was 58. A summary of these cases is given below.

1. FORCEPS.

Forceps was applied 31 times, or in about 1.9% of cases.

All the mothers were alive after delivery, 4 infants were born dead, one died in a few hours due to prematurity.

The indication in 26 cases was delay in the 2nd stage. Foetal distress was the indication for 2 cases, Eclampsia one case, prolapse of cord one, and maternal distress 3 cases.

2. PRESENTATION AND PROLAPSE OF CORD.

There were 5 cases of prolapse of cord all the mothers recovered, 3 children were born alive, one was a case of miscarriage (6 months) and one foetus was still born.

The cases were :—

C.P.Y. aged 24, para. 3, and a lateral placenta praevia with prolapse of the cord and profuse hæmorrhage: os fully dilated, immediate delivery with forceps, live infant extracted.

Mrs. S. aged 25, para. 1, admitted with prolapse of cord and vertex presentation, no pulsation felt, cord not replaced, spontaneous delivery. Stillborn baby.

T.K. aged 24. para. 1, admitted with prolapse of cord and vertex presentation no pulsation felt, cord not replaced spontaneous delivery. Weight of infant 1 lb., 12 ozs.

S.K. aged 43, para. 2, admitted with vertex, presentation and prolapse of cord, spontaneous delivery, live infant.

L.L. aged 33. para. 7, a case of twin pregnancy, admitted with prolapse of cord and breech presentation, 1st infant born alive, weighing 4 lbs., 4 ozs., 2nd vertex normal delivery, alive.

HÆMORRHAGES.

Placenta Prævia.—Our routine treatment for this condition is bipolar version; there were 11 cases, all being multipara, 3 infants were born alive and no maternal mortality.

Accidental.—There were six cases of accidental hæmorrhage five of the mothers recovered, four of the infants were born alive.

One case died of secondary concealed postpartum hæmorrhage, 2½ hours after delivery. The treatment carried out in all cases was rupture of membranes, tight binder, and ¼ c.c. pituitrin repeated every ¼ hours up to 1 c.c.

Cæsarean Section.—This operation was performed once in 1616 deliveries; under ½% novocain anæsthesia. Indication—stenosis of the cervix.

W.T. aged 36, para. 2 was admitted to the Tsan Yuk Hospital on December 1929 in her ninth month of pregnacy.

The first pregnancy 3 years ago was complicated by eclampsia, and forceps were applied in a neighbouring hospital, and a still born baby extracted.

The patient started labour on the 23-12-29 at 5 p.m. Vaginal examination at 7.30 p.m. the same day showed a closed os and the head low. The next day the patient had strong pains but made no progress, on vaginal examination, it was found that the head was low, and the os completely closed, the cervix distended by the fœtal head came down to the vagina like a bag of membranes. Under ½% novacain infiltration anæsthesia, the classical section was performed, as the lower segment relations were altered by the occlusion of the os.

Before suturing the fundal incision the largest size Hegar's dilator was passed into the cavity of the uterus, dilating and opening the occluded os. The infant weighed 7 lbs., 6 ozs. The patient made an uneventful recovery, the highest temperature chartered was on the 2nd day 100.4.

DESTRUCTIVE OPERATIONS ON THE FŒTUS.

T.Y.H. 3.

G.C.H. nil.

H.O. age 42, para. 12, breech presentation delivery of breech, head retained, all attempts to deliver the head failed, perforation of after coming head.

C.L.Y. age 21, para. 1, admitted with repeated rigors high temperature and pulse, dead fœtus: craniotomy. Putrid discharge during the puerperium.

H.S. age 34, para. 4, maternal distress, pulse 120 and vomiting. Failed forceps, dead fœtus—craniotomy.

Ruptured Uterus.—There were two cases of rupture of uterus, their particulars are as follows:—

F.T.H. age 23, para. 2, was admitted 18/9/29, for a second Cæsarean section. The indication for the 1st section was marked degree of contracted pelvis after this operation, the patient developed pneumonia and typhoid.

The fœtal parts were easily palpated and the fœtal heart sound was heard, the presentation was transverse.

On opening the peritoneal cavity free blood escaped and the uterus was found ruptured at the fundus anteriorly, at the site of the old scar, the placenta being partly attached to the rupture and the sac intact. A still born fœtus was extracted and the rupture sutured, hysterectomy was not performed, the puerperium was uneventful.

D.M.S. age 30, para. 4, was admitted on the 5/11/29 with a ruptured uterus. The second twin was lying transversely with the shoulder impacted. The first twin was delivered at home. She was suffering from shock. On laparotomy, the fœtus was found to have passed completely outside the uterus through a rent, extending from left side of the broad ligament, along the length of the left side of he uterus, up to the left tube, rupturing the left uterine artery with free hæmorrhage.

The fœtus was delivered, and the rent was sewn over gauze, and drained through the cervix into the vagina. The patient did not recover from post operative shock, and died 2 hours later.

TOXÆMIA OF PREGNANCY.

During the year there were 74 cases of albuminuria at the Government Civil Hospital, 70 slight to moderate, and 4 considerable; of the 4, one case development Eclampsia.* In the same year at the Tsan Yuk Hospital there were 324 cases; slight to moderate 323, and one considerable which developed Eclampsia. The history of the Eclampsia cases is as follows:—

Tsan Yuk Hospital:—

H.W.K. age 21, primipara admitted 15/8/29 and had her 1st fit in hospital.

Patient was in labour and on Vaginal examination the os was found fully dilated, under Aether Anaesthsia Forceps was applied and a live child extracted.

Following the delivery and the modified Tweedy treatment for Eclampsia, the patient had no further fits.

Govt. Civil Hospital.

* This case was seen in consultation by us in another department and is only shown here as an interesting case.

C.L. age 33, admitted 10-12-29 to the Medical Ward of Government Civil Hospital, pregnant 7½ months. She was suffering from Hemiparesis of the left side of the body. The histiry is as follows:—6 days ago patient had severe headache and flashes of light across her eyes and vomiting; the next day patient lost sensation and movement of left side of the body (from face down to the legs).

Three days after the onset of hemiparesis patient had no fits, was able to talk hospital for treatment. While in the Hospital, patient had no fits, was able to talk and give a clear history of her short illness, was able to take light diet, and urine examination was clear. She was transferred to the Government Civil Hospital on an ambulance on the afternoon of the tenth. That same evening at 10.50 p.m. she had a fit lasting 5 minutes. In the morning she had a series of fits, with coma intervening between the fits; she was given morphia ¼ grs. and Haustus Sed. 1 ozs.

The question of the diagnosis arose, as to the cause of the fits, whether they were eclamptic, or uræmic. The blood pressure was—systolic 100, disastolic 65. Eye examination showed no retinal changes. The urine contained 3 plus albumin, granular casts, pus cells, and red blood cells.

On the 11-12-29, 11.30 a.m. the patient was treated as an eclamptic, she improved a little, the urine secretion increased and bowels acted freely.

During the evening the condition of patient became worse, pulse 160, and deep coma. In all she had 19 fits. On the 12th morning 5 a.m. she was delivered of a still born child, labour was normal.

Her condition improved slightly after delivery but later in the evening became much more serious. She had a series of fits, total number 42), the coma was deep, temperature rose to 106, and patient did not regain consciousness before death, which occurred at 2.30 a.m. on 13-12-29.

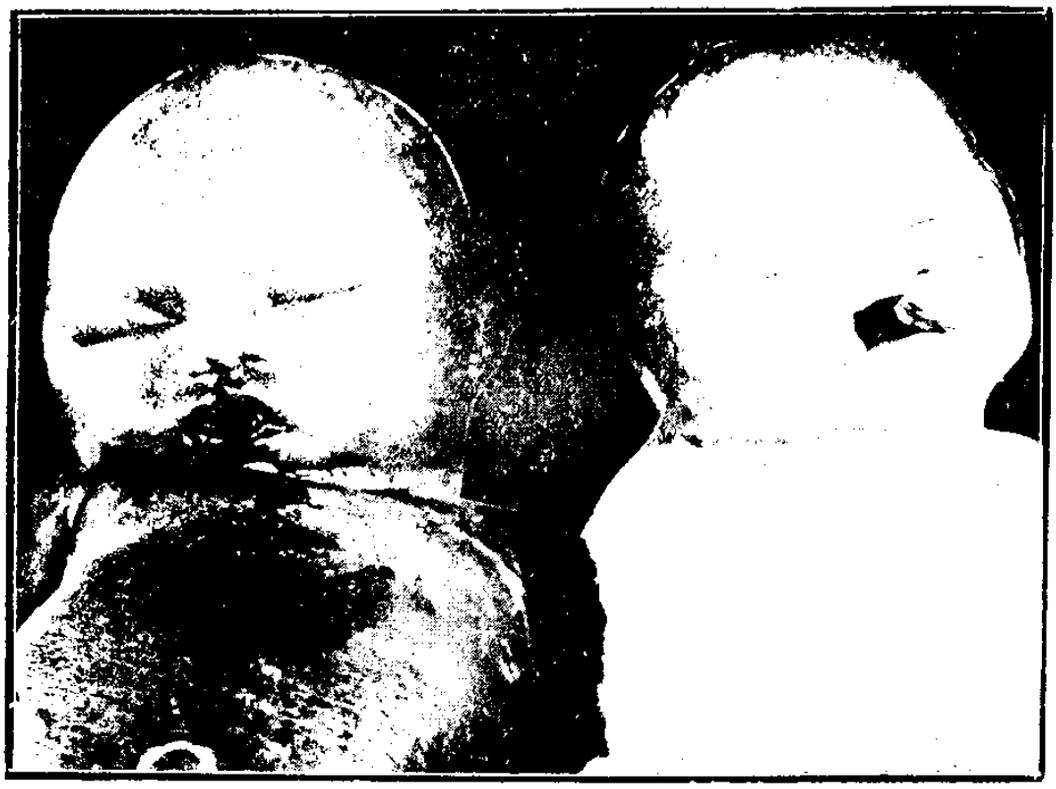
Post Mortem Findings.—Subdural hæmorrhage, with destruction of brain matter, anterior and posterior to the central Gyrus on the right side, liver yellow, fatty and fragile, with areas of hæmorrhage and necrosis. Acute nephritis, œdema, and congestion of lungs with extensive pleural adhesions (chronic). Uterus undergoing involution. Other organs relatively healthy.

Remarks.—The history is suggestive of Cerebral Hæmorrhage complicated by eclampsia. Though the patient improved slightly with the modified Tweedy treatment for eclampsia, the cerebral lesion as found in autopsy was incompatible with life.

INTERESTING CASE OF HYDRAMINOS AND TWINS WITH HARE LIPS AND CLEFT PALATES.

L.Y.S. age 46, para. 12, Pregnant 27 weeks was admitted on the 20-11-29 to Government Civil Hospital. She had Dyspnœa and the abdomen was the size of a full term pregnancy. She had œdema of legs. Heart and lungs were normal.

Abdomen was tense, fœtal parts could not be felt, presenting part not fixed. On admission the os was the size of two fingers. Membranes intact with good tension, head presenting, high, and not fixed.



The [illegible] tumour, [illegible] eye, and [illegible] palate.

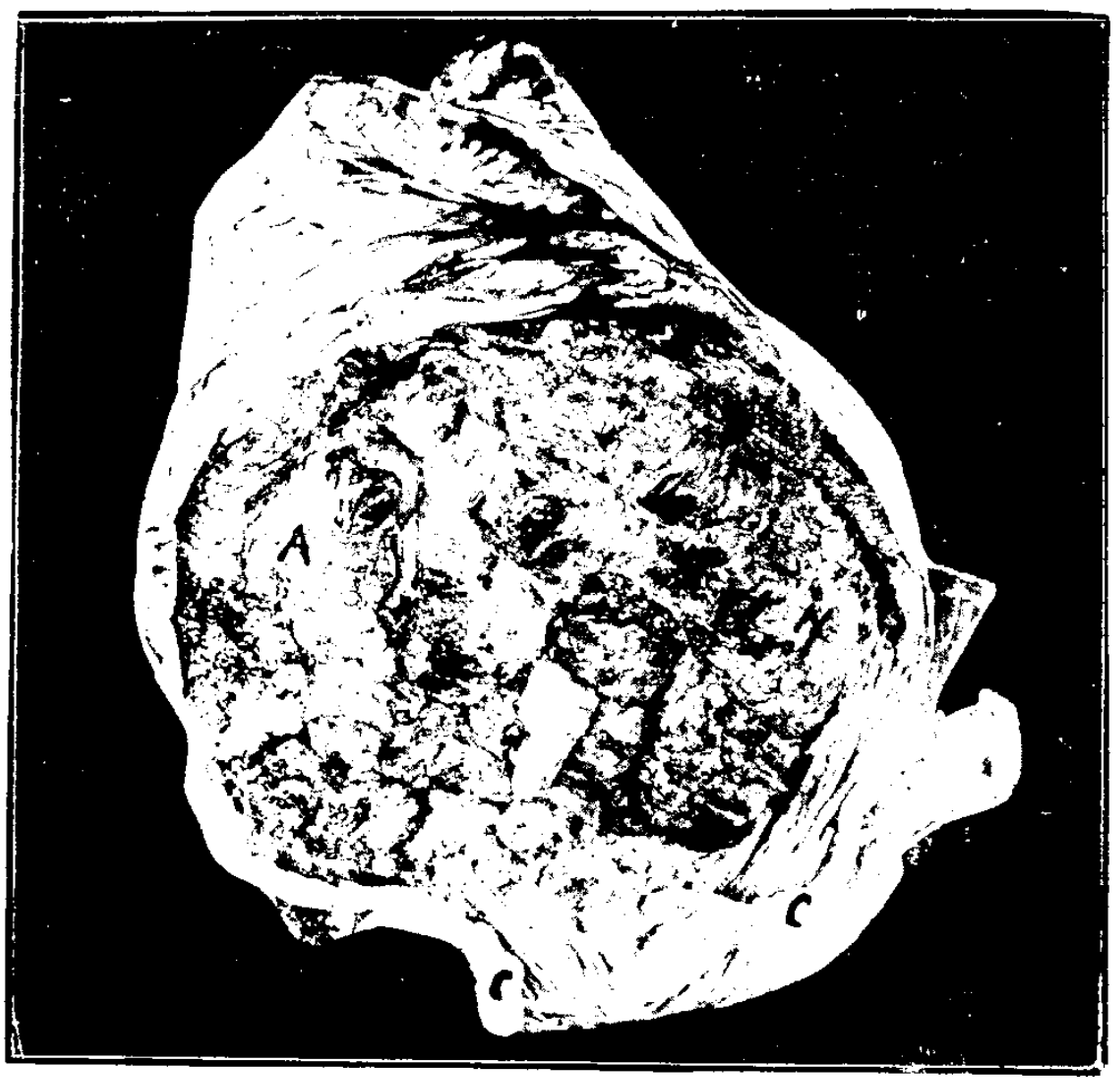


Fig. [illegible] double.

A. [illegible]

N. [illegible]

C. C. [illegible]

One plus albumin. Semi Fowler's position adopted and patient was kept on low diet, and carefully watched for 3 days. Condition improved, on the 4th day dyspnœa was marked and it was decided to induce labour. The membranes was ruptured on the 23-11-29 at 2.30 p.m. and 12 pints of liquor amnii evacuated slowly. General condition of patient good. Pulse 122. Temperature normal.

At 7.20 p.m. 1 c.c. pituitrin was given. Labour started half an hour later.

At 9.25 p.m. First male child was born 4½ lbs. The child breathed a few minutes, but there was no actual cry, and died 5 minutes after. The 2nd bag of membranes was ruptured, and another male child weighing 2 lb., 14 ozs. was born normally.

Both children presented by the vertex, and had cleft palates and hare lips.

The first child had fœtal Ascites, second child though smaller was healthy, and lived for 5 days.

The umbilical cords of both the fœtuses were very short. One chorion and two amniotic sacs.

Placenta measured about 10" in diameter. Half of the placenta, to the naked eye, appeared normal, the other half was pale and friable. The two halves being sharply demarcated. The Wassermann was returned negative.

Condition During Pregnancy.—Patient gave no history of any fright. She had 3 falls during the last 3 months of pregnancy. She believes that after the fall her abdomen became bigger and she noticed discomfort in the chest. Her abdomen has been rapildy growing in size during the last 3 weeks. Three days before she was admitted into hospital she could not take any food, and vomitted all what she took.

Previous Pregnancy.—The present pregnancy is the twelfth. All previous children were born alive. Six are alive and five died during infancy. There is no history of abortion or miscarriage. None of the eleven children had any deformity. The patient's husband is free from any deformity.

Remarks.—The case is interesting as half the placenta is abnormal and sharply marked of from the other normal half.

The first bag of membranes which was artificially ruptured containing 12 pts. of liquor amnii and the child weighed 4½ lbs. and had fœtal ascites; died shortly after delivery.

The second sac, on rupture, had a normal quantity of liquor and the child weighed 2 lb., 14 ozs. and lived for 5 days. Both the children had hare lips and cleft palates.

Morbidity.—The Morbidity rate of the two Hospitals is as follows:—

Government Civil Hospital...	5%
Tsan Yuk Hospital	5.7%

According to the pyrexia standard of the B.M.A. there were 88 cases of puerperal pyrexia during the year. Giving an average of one in 18.3 of cases and a total percentage, morbidity of 5.4%.

The morbidity rates in both the hospitals are the lowest since 1925. This is probably due to better facilities for the management of the puerperium.

Morality:—There were altogether 5 deaths, 2 in Tsan Yuk Hospital and 3 in the Government Civil Hospital giving a mortality rate of .45% (see mortality statistics for details).

D. K. PILLAI.

Table No. I.—*STATISTICS OF MATERNITY DEPARTMENT.*

Nature and number of cases treated:	*T.Y.H.*	*G.C.H.*
Total admissions	1,231	547
„ deliveries	1,072	544
„ multiparæ	67	365
„ primiparæ	395	179
Presentations:		
Vertex, normal rotation	1,076	489
„ I.	69.8%	62.04%
„ II.	25.9%	35.7 %
„ III.	1.1%	1.36%
„ IV.	3.2%	.9 %
face to pubes	1	3
Face	—	—
Breech	32	23
Transverse	1	3
Twins	6	5
Miscarriages	2	1
Hæmorrhages:		
Placenta Prævia	5	5
Post-partum	22	6
Accidental	3	5

	T.Y.H.	*G.C.H.*
Abnormalities:		
Prolapse of cord	3	2
Prolapse of hand	—	4
Hydramnios	1	4
Eclampsia	1	1
Albuminuria:		
Slight to moderate		70
Considerable		4
X-Ray Diagnosis	—	2
Operations:		
Suture of perineal lacerations:		
Complete		1
Incomplete		166
Multiparæ		67
Primiparæ		100
Suture of cervical lacerations	2	2
Forceps	14	17
Destructive Operation on fœtus	3	—
Manual removal of placenta	3	4
Cæsarean section	1	—
Rupture uterus operation for	—	2
Version:		
Bipolar	5	8
Internal	1	—
Accidental Complications:		
Puerperal Ulcers	8	1
Malaria	—	1
Dysentery	4	—
Cellulitis of finger	1	—
Mumps	2	—
Cystitis	1	—
Hookworms	2	—
Asthma	1	1
Oedema of legs	2	1
Round worms	—	1
Diarrhœa	—	1
Hydatidiform Mole	1	1
Gastritis	1	—
Hæmatoma of vulva	1	—
Myoma of anterior lip	1	—
Bronchitis	2	—

Morbidity B.M.A. Standard:

Average	one in	17.6	20.1
Percentage		5.7%	5.0 %

Mortality:

Total		2	3
Average	one in	615.5	136.8
Percentage		.17%	.73%

Table No. II.—*INFANT STATISTICS.*

Total births	1,072	544
Alive	1,031	503
Dead	41	31
Premature		
Full term	18	23
Macerated	23	6
Children born alive who died in Hospital	3	8

Abnormalities:

Abnormal fœtus	—	1
Hare lip and cleft palate	1	1

Complications:

Hydrocephalus	—	1
Cerebral hæmorrhage	—	1
Anencephalic monster		3
Ascites	—	1
Cephalo hæmatoma	2	—
Ophthalmia	3	—
Condyloma	1	—

Table No. III. *Pelvic Presentation.*

Para	Total	Dead Children	Remarks
T. Y. H.			
Primiparae	9	Macerated 1	One case the after coming head had to be perforated.
		Premature 1	One died half an hour after birth. Two cases with extended legs.
Multiparae	18	Macerated 3 Premature 2 Full term 1	Two cases occurred in twin pregnancies.
G. C. H.			
Primiparae	9	Macerated 1 Premature 2	One abnormal foetus, weighing 12 ozs. Both ears absent. Skull bones, membraneous.
Multiparae	14	Macerated 2	Two cases with extended legs
		Premature 2	One case of delay of the after coming head was due to Hydrocephalus, the spine was tapped and 15 pints of clear fluid was evacuated.
		Full term 1	Two cases with prolapsed hand.

Table No. IV.

Placenta Praevia.

Name	Age	Para	Period of Pregnancy	Presentation	Variety	Result to Mother	Result to Child	Remarks
T. Y. H.								
Y. Y.	26	3	9 months	Vertex	Central	Recovery	Dead	Bipolar Version. 2 fingers os.
K. R. S.	28	7	8½ ,,	,,	Lateral	,,	Recovery	Bipolar Version. 3 fingers os.
L. S.	38	5	6 ,,	,,	Central	,,	Dead	Slight Hæmorrhage. Os admits 1 finger. Bipolar Version.
W. L.	33	12	6 ,,	Transverse	Marginal	,,	,,	Bipolar Version. Severe hæmorrhage before admission. Pulse irregular. Os 4 fingers.
C. C.	41	7	8 ,,	Breech	Marginal	,,	,,	Bipolar Version. Profuse hæmorrhage. Os admits 2 fingers.
G. C. H.								
K. Y.	38	6	35 weeks	Breech	Marginal	Recovery	Dead	2 fingers os. Bipolar Version.
C. P. Y.	24	3	32½ ,,	Vertex	Lateral	,,	Recovery	Lateral placenta prævia with prolapse of cord Forceps delivery—live baby. Velamentous insertion of cord and battledore placenta.
F. L. M.	38	9	37½ ,,	,,	Central	,,	Dead	Hæmorrhage for 3 days. On admission signs of air hunger. Condition did not permit of immediate version. Treated for shock; pulse improved and Bipolar Version performed.
S. Y. T.	35	7	24 ,,	,,	Lateral	,,	Recovery	Spontaneous delivery. Infant died 4 hours after.
K. Y.	28	5	7 months	Breech	Central	,,	Dead	Spontaneous delivery.
L. C. S.	28	5	8½ ,,	,,	Marginal	,,	Recovery	Bipolar Version. 2 fingers os. Slight Post Partum hæmorrhage.

Table No. V. *Prolapse and Presentation of the Cord.*

Name	Age	Para	Weight of Child	Presentation	Treatment	Result to Mother	Result to Child	Remarks
T.Y.H.								
T. K.	24	1	1 lb. 12 ozs.	Vertex	Spontaneous	Recovery	Dead	Immaturity at birth
L. L.	33	7	4 lbs. 4 ozs. 4 lbs.	Breech Vertex	—	Recovery	Recovery	Membranes ruptured on admission. A loop of cord ontside the vulva.
S. K.	43	2	4¾ lbs.	Vertex	—	Recovery	Recovery	
G.C.H.								
C.P.Y.	24	3	4¾ lbs.	Lateral placenta praevia and vertex presentation	Forceps delivery	Recovery	Recovery	Admitted with haemorrhage and prolapsed cord and os fully dilated Immediate forceps delivery.
Mrs. S.	25	1	5 lbs.	Vertex	Replaced	Recovery	Dead	No pulsation of cord felt. Normal delivery.

Table No. VI.

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....	12	14	12	14	—	—	11	12	1	2	One case of P.O.P. Two cases had a rise of temperature and pulse.	
Foetal Distress.....	1	—	1	—	—	—	—	—	1	—	Child alive when born, artificial respiration for ½ hour, but the foetal heart stopped beating.	One case with mitral stenosis, and Dyspnœa.
Eclampsia.....	1	—	1	—	—	—	1	—	—	—	Albuminuria++. Had one fit before forceps.	
Prolapse of Cord.....	—	1	—	1	—	—	—	1	—	—	——	
Maternal Distress.....	—	2	—	3	—	—	—	2	—	1	——	

Table No. VII.

Number of Pregnancy of Patients in whom Forceps were applied.

PARA.	Number of forceps cases.		
	T.Y.H.	*G.C.H.*	*Total.*
1	9	10	19
2	—	—	—
3	—	5	5
4 and over	5	2	7
Total	14	17	31

Table No. VIII.

Age of Patients in whom Forceps were applied.

PARA.	Number of forceps cases.	
	T.Y.H.	*G.C.H.*
17—25	5	6
26—30	4	5
31—35	3	5
36 and over	2	1
Total	14	17

Table No. IX.

Destructive Operation on Foetus.

Name	Age	Para	Indication	Operation	Remarks
T. Y. H.					
H. O.	42	12	After coming head retained in uterus for over half an hour in spite of all efforts to deliver it.	Perforation of the after coming head.	——
C. L. Y.	21	1	Repeated rigors. Temperature and pulse high.	Perforation and craniotomy.	Very offensive discharge from uterus.
H. S.	34	4	Mother in distress. Pulse 120 vomiting.	Perforation.	Forceps failed.

Table No. X.

Morbidity, B.M.A. Standard.

	MAY		JUNE		JULY		AUGUST		SEPTEMBER		OCTOBER		NOVEMBER	
	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.	T.Y.H.
Total Deliveries..	41	67	55	92	65	102	52	121	50	126	57	125	42	93
Cases Morbid.....	1	2	8	10	—	10	6	6	5	6	2	4	1	2

	DECEMBER		JANUARY		FEBRUARY		MARCH		APRIL		TOTAL		GRAND TOTAL
	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.	
Total Deliveries..	40	101	42	106	29	70	35	76	36	58	544	1072	1610
Cases Morbid....	1	5	3	3	—	7	—	3	—	3	27	61	88

	G.C.H.	T.Y.H.	GRAND TOTAL
Total number of morbid cases..	27	61	88
Total average morbidity	20	17.6	18.3
Total percentage morbidity	5%	5.7%	5.4%

Table No. XI.

Comparative Morbidity in Primiparae and Multiparae.

Primiparae	MAY		JUNE		JULY		AUGUST		SEPTEMBER		OCTOBER		NOVEMBER	
	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.	T.Y.H.
Total Deliveries..	11	17	18	25	23	36	22	39	19	45	19	59	11	29
Cases Morbid.....	—	2	5	5	2	7	6	2	3	1	1	4	—	1

Primiparae	DECEMBER		JANUARY		FEBRUARY		MARCH		APRIL		TOTAL		GRAND TOTAL
	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.	
Total Deliveries..	17	33	11	42	6	24	15	23	7	26	179	395	574
Cases Morbid.....	1	3	—	3	—	7	—	1	—	1	18	37	55

	G.C.H.	T.Y.H.	GRAND TOTAL
Total average morbidity	9.4	10.7	10.4
Total percentage morbidity	10.1%	9.3%	9.5%

Table No. XI.—*(Continued)*

Comparative Morbidity in Primiparae and Multiparae.

Multiparae	MAY		JUNE		JULY		AUGUST		SEPTEMBER		OCTOBER		NOVEMBER	
	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.	T.Y.H.
Total Deliveries..	30	50	37	67	42	66	30	82	31	31	28	69	31	64
Cases Morbid.....	1	—	3	5	1	3	—	4	2	5	1	—	1	1

Multiparae	DECEMBER		JANUARY		FEBRUARY		MARCH		APRIL		TOTAL		GRAND TOTAL
	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.	
Total Deliveries..	23	68	31	64	23	46	20	53	28	35	365	677	1042
Cases Morbid.....	—	2	—	—	—	—	—	2	—	2	9	24	33

	G.C.H.	T.Y.H.	GRAND TOTAL
Total average Morbidity	40.5	28.2	31.5
Total percentage Morbidity	2.9%	3 5%	3.2%

Table No. XII.

Extra-genital causes of Morbidity.

	T.Y.H.	*G.C.H.*
Dysentery	3	—
Mumps	1	—
Puerperal Ulcer	2	1
Cough	1	1
Cystitis	1	—
Hookworms	1	—
Diarrhœa	—	1
Malaria	—	1

Table No. XIII.

Caesarean Section.

Name	Age	Para	Date	Nature of Operation	Indication	When Performed	Result to Mother	Result to Child	Remarks
T. Y. H. W. T.	36	2	24-12-29	Classical. Cæsarean Section.	Os completely closed.	During Labour	Recovery	Recovery	Complained of great pain during labour. On examination, the os was found to be completely closed, the cervix distended by the fœtal head and came down to the vagina like a bag of membranes.
G. C. H.				**RUPTURE OF UTERUS**					
F. T. T.	23	2	18-9-29	Ruptured uterus sutured.	Quiet rupture of the uterus.	During Labour	Recovery	Dead	Owing to lack of conveyance patient was admitted late into the hospital, with ruptured uterus patient's general condition good. Fœtal parts easily felt. A year ago patient had a classical section done for contracted pelvis. Rupture taking place in the site of the old scar.
D. H. S.	30	4	5-11-29	Suture of complete tear of uterus extending from left broad ligament involving the left uterine artery to the left tube.	Neglected transverse presentation with prolapsed hand and ruptured uterus followed by the delivery of the 2nd twin. The 1st being delivered spontaneously at home.	During Labour	Dead	Dead	Patient admitted with prolapsed hand, and ruptured uterus, and signs of shock. 3 hours after delivery of 1st twin.

Table No. XIV. *Eclampsia.*

Name	Admission	Age	Para	Condition on Admission	Urine	Number of Fits			Treatment	Result to Mother	Result to Child	Remarks
						Before Labour	After Labour	During Labour				
T.Y.H. H.W.K.	31.7-29	27	8	Brought in a fit.	Albumen ++	1	—	—	Morphia ½. Rectal wash out. With Sod. Bicarb Rectal mag. sulph.	Recovery	Alive	Patient had a fit. Os fully dilated. Forceps applied. Bowels acted freely. Patient pulse good.
G.C.H. C.L.	10-12-29	33	6	Hemiplegia of left side of body brought in an ambulance.	Albumen +++ Blood corpuscles granular and Hyaline casts.	8	35	6	Routine treatment Rectal æther 1 oz. repeated 3 times Digitalin.	Dead	Dead	Hemiplegia complicating Eclampsia. Admittd with no albumen in urine after fits 3 + albumen improved with routine treatment 24 hours after delivery patient became comatose, continuous fits, died without regaining consciousness, P.M. Extensive hæmorrhage of right side of brain with cerebral softening and acute nephritis, incompatible with life.

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

Name of Operation	Number		No. of Mobidity		Percentage		Average		Remarks	
	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.
Forceps.	18	14	4	2	21%	15·6%	1 in 3·9	in 6	One case of placenta accreta. 2 manual removals.	
Internal Version.	—	2	—	1	—	50%	—	1 in 2		In labour head unfixed.
Bipolar Version.	6	3	2	2	33⅓%	66%	1 in 3	1 in 5	One case central placenta prævia. Examined before admission.	One case of placenta prævia bad hæmorrhage before admission.
Suture of Perineal laceration.	142	117	10	22	14%	18·8%	1 in 1·42	1 in 5·3		
Destructive Operation on the fœtus.	—	3	—	—	—	—	—	—		
Manual Removal of placenta.	5	3	3	1	75%	33%	1 in 1·3	1 in 3	One case admitted 12 hours after delivery with retained placenta.	Profuse hæmorrhage.

Table No. XVI.

Duration of Stay in Hospital of Morbid cases.

	T.Y.H.	*G.C.H.*
Less than 10 days	44	12
10—19 days	16	14
20—29 days	1	1
30 and over	—	—
Total	61	27

Table No. XVII.

Duration of Temperature.

	T.Y.H.	*G.C.H.*
Less than 5 days	52	22
5—9	9	5
10—19	—	—
20 and over	—	—
Total	61	27

Table No. XVIII.

Highest Temperature Charted.

	T.Y.H.	*G.C.H.*
100°—100.9	18	3
101 —101.9	30	11
102 —102.9	6	8
103 —103.9	5	3
104 and over	2	2
Total	61	27

Table No. XIX. *Mortality.*

Name	Age	Para	Adimited	Delivered	Died	Cause of Death	Remarks
T.Y.H.							
L. H.	18	1	18-7-29	18-7-29	18-7-29	Post Partum haemorrhage	Patient had normal delivery. On examination cervix found torn. There was no time to stitch as patient's pulse was very feeble. Uterus and Vagina plugged, breast saline given. Pituitrin and ernutin given but patient died soon after.
C. M. K.	24	1	29-10-29	30-10-29	30-10-29	Shock	A case of twins. Delayed 2nd stage, uterus tender, and there was vomitting. The 1st baby was delivered by forceps. The 2nd was lying obliquely. Internal version, but could not be delivered. Uterus found unruptured. Another attempt to deliver baby succeeded, but patient died 3 hours after.
G.C.H.							
M. S.	40	7	18-6-29	18-6-29	20-6-29	Spontaneous delivery of hydatidiform mole	Chronic ill health with hæmorrhage. Pulse, fast, acute nephritis generalised œdema, and failing heart. Patient died from acute Toxæmia, lochia normal.
C. L.	33	6	12-12-29	12-12-29	12-12-29	Eclampsia	Admitted from T.Y. to G.C.H. medical ward, for hemiplegia. Series of fits. Routine treatment of Eclampsia carried out. Albumen†††. Patient improved after normal delivery, comatose,, continous fits 49 in all. Died. (See Eclampsia).
C. K.	26	1	10-6-29	11-6-29	11-6-29	Shock	Concealed and revealed accidental hæmorrhage. Rupture of membranes. Concealed secondary Post partum hæmorrhage. 2 hours after delivery. Shock treated. Patient did not rally and died ½ hour later.

Table No. XX. *Accidental Haemorrhage.*

Name	Age	Para	Period	Variety	Result to Mother	Result to Child	Presentation	Albumin	Remarks
G.C.H.									
M.	22	2	8 Months	Concealed and Revealed	Recovery	Recovery	Vertex	+	Spontaneous.
K. L. Y.	20	1	36 Weeks	Concealed and Revealed	Recovery	Dead	Vertex	+	Bleeding in the 1st and 2nd stages of labour. Normal delivery.
Y. S.	39	1	38 Weeks	Revealed	Recovery	Recovery	Vertex		Spontaneous.
Y. S. H	29	1	38 Weeks	Revealed	Recovery	Recovery	Vertex	+ + +	Forceps delivery.
T.Y.H.									
C. W. I.	22	2	39 Weeks	Concealed	Recovery	Recovery	Vertex	+	Bleeding from vagina on day of admission when labour started and continued till head appeared on vulva. Since then hæmorrhage stopped. No signs of shock.
G.C.H.									
C. K.	26	1	Term	Concealed and Revealed	Dead	Dead	Vertex	+	Concealed and revealed postpartum hæmorrhage Rupture of membrane. Postpartum hæmorrhage 2½ hours later had concealed postpartum hæmorrhage. Died.

GYNÆCOLOGICAL REPORT.

This year there were 328 cases admitted. 195 to the Tsan Yuk Hospital and 133 to the Government Civil Hospital. The total number of operations was 169.

OVARIAN CYST.

There were 20 cases of ovarian cyst treated by operation. In 19 cases the cysts were removed. One case was treated by marsupialization as it was malignant and inoperable, 18 pints of blood stained fluid being evacuated during the operation. There was another case of malignant cyst which was partly adherent to the abdominal wall; section of the ovary showed a malignant adenoma.

Good results generally attended the operations. One patient died shortly after operation. On post mortem examination it was found that both kidneys were cystic and atrophied. Large unilocular cysts were generally tapped before excision, in such cases only a small incision was made.

PROLAPSE.

This condition was present in 15 patients. According to the degree of prolapse a combination of the following operations was performed:—Anterior colporrhaphy; perinæorrphapy; amputation or reconstruction of the cervix; vaginal suspension; ventral suspension; shortening of Mackenrodt's ligaments and vaginal hysterectomy. Results of all the operations were satisfactory.

HYSTERECTOMY.

Abdominal hysterectomy was performed five times, one of these was performed for fibrous stricture of the vagina which barely admitted a finger, the other four for uterine fibroids.

MORTALITY.

There were 4 deaths:—

K.Y.B. Age 37. *Extrauterine pregnancy.*

Patient collapsed 24 hours after operation. Cold sweats, clammy, feeble pulse, limbs rigid and left pupil dilated, cause of death (clinically) Embolism of brain.

P.M. findings:—

Plastic peritonitis.
Free blood in peritoneal cavity;
Blood stained fluid in pleural cavities.
Oedema and congestion of lungs.
Soft toxic spleen.
Excess of fluid in cranial cavity.
Oedema of brain.

C.Y. Age 19. *Puerperal sepsis* (Delivered 15 days ago in a neighbouring hospital).
Died 2½ hours after admission.

Ovarian cyst:—

W.L.H. Age 18. P.M. findings:
Blood in middle cranial fossa (left side).
Both kidneys atrophied and cystic.
Congenital polycystic kidneys.
Dilatation of both ureters.
Soft flabby, somewhat atrophic liver.
Oedematous lungs.

L.X.. Age 29. *Carcinoma of sigmoid and pelvic colon:*—(Patient had diarrhœa (profuse) 1 week before operation. After operation—profuse diarrhœa, rapid pulse, and death.

Post mortem findings:

Malignant growth of left ovary involving various parts of colon " deposits in liver and glands.
Subacute kidneys.
Ascites (not blood stained).
Multiple fibrous adhesions of viscera.

Table No. I.

Statistics of Gynæcological Department.

	T.Y.H.	*G.C.H.*
Nnmber of admissions	195	133
,, ,, Operations	69	100

Table No. II.

Nature and Number of Operations.

Vulva:	*T.Y.H.*	*G.C.H.*
Condyloma of vulva	—	1
Epithelioma of clitoris	—	1
,, ,, vulva	—	1
Hypertrophy of clitoris	—	1
Fibroma of labium	—	1
Bartholin cyst	—	1
Perineum:		
Perinæorrhaphy	2	2
Vagina:		
Vesico-vaginal fistula	—	1

	T.Y.H.	G.C.H.
Uterus:		
Currettage	26	23
Prolapse	12	3
Myomectomy	2	1
Ventro-suspension	2	14
Subtotal Hysterectomy	2	2
Total Hysterectomy	—	1
Abortion (curettage)	—	2
Cervix:		
Trachelorrhaphy	2	—
Amputation	5	3
Polypus	2	1
Tubes and Ovaries:		
Marsupialization of cyst	—	1
Ovariotomy	8	11
Salpingectomy	7	9
Pyosalpinx	—	2
Extrauterine gestation	1	6
Salpingostomy	1	—
Miscellaneous:		
Laparotomy	—	1
Removal of tissue for section	2	—
Insertion of pessary	3	—
Sebaceous cyst	1	—
Breast abscess	—	3
Ventral hernia	—	1
Implantation of ureters into rectum	—	1
Growth of colon	—	1
Gonorrhœal warts	—	1
Drainage of ovarian cyst	—	1
Vesicular mole	1	—
Pseudo Didelphys	—	1
Carcinoma of uterus	—	1
„ „ cervix	—	1
	—	1

Table No. III.

Nature and Number of Cases Treated Without Operation.

	T.Y.H.	G.C.H.
Refused treatment	56	8
Abscess of vulva	1	—
Leucorrhœa	4	—
Pregnancy	4	—
Operation contra-indicated by general health	—	1

	T.Y.H.	*G.C.H.*
No operation indicated	2	—
Pregnancy with gonorrhœa	5	—
„ „ nephritis	1	1
„ „ œdema	1	—
„ „ diarrhœa	1	—
„ „ cystitis	1	—
„ „ tuberculosis	—	1
Abortion	2	—
Salpingitis	2	—
Urethritis	3	—
Ulceration of vulva	1	—
Condyloma	2	—
Gonorrhœa	1	—
Cases for observation	1	—
Miscarriage	2	—
Hydramnios	1	—
Pelvic Tumour	1	—
Ankylostomiasis and Ascariasis	1	—
Vaginitis	1	—
Inoperable cancer	9	—
Mastitis	1	—
Infantile uterus	—	1
Uræmia complicating pregnancy	—	1
Vulvitis	1	1
Menstruation	—	2
Subinvolution	—	1
Threatened abortion	—	1
Papilliferous ovarian cyst	—	1
Puerperal sepsis	—	1
Carcinoma of vaginal wall	—	1
Carcinoma of cervix	—	5
Fibroid uterus	—	1
Retroversion (pessary)	—	1
Salpingitis	—	4
Fixed retroversion	—	2
Papillomatous growth around anus	—	1
Cystitis	—	1
Post partum Beri-beri	1	1
Vesical calculus	—	1
Prolpase of uterus	2	1
Pessary inserted	1	—
Cancer lead treatment	1	—
Inoperable cancer		—
Syphilitic eruption	1	—

Table No. IV. *Hysterectomy (Abdominal).*

No.	Name	Date	Age	Disease	Operation	Result	Remarks
T.H.Y. 225	G. C. Y.	4-2-29	50	Myoma. Broad Ligament cyst.	Subtotal Hysterectomy.	Recovery	Myoma size of 2 fists. Left broad Ligament cyst size of fœtal head removed.
25	L. S. M.	20-2-30	46	Uterine Fibroid.	Subtotal Hysterectomy Ovariotomy.	Recovery	Uterus enlarged to about 4 months pregnancy. Left cystic ovary excised. Right ovary resected.
G.C.H. 27	L. N. S.	—	23	Vesico-Vaginal fistula and stricture of vagina.	Subtotal Hysterectomy and implantation of ureters into rectum.	Recovery	Fibrous stricture of vagina, barely admitting tip of finger. Bladder m.m. can be seen presenting at the vulva.
50	L. K.	27-8-29	50	Uterine fibroid.	Subtotal Hysterectomy.	Recovery	Tumour—size of football. More on left than on right tumour, 2 finger's breath below xyphoid process. Weight 12 lbs.
76	K. A. H.	29-10-29	35	Uterine fibroid.	Hysterectomy.	Recovery	Tumour size about 6 months pregnancy, moves with cervix.

Table No. V. *Ovariotomy.*

No.	Name	Date	Age	Disease	Operation	Result	Remarks
T.Y.H. 202	S. H.	25-6-29	28	Ovarian cyst.	Ovariotomy.	Recovery	Abdomen occupied by a swelling size of a 7 months pregnancy. Weight 16¾ lbs.
215	Y. S. Y.	16-7-29	30	Medium size Cystic Tumour.	Ovariotomy.	Recovery	Cyst tapped. Brown fluid evacuated.
216	Y. M. C.	16-7-29	26	Ovarian cyst.	Ovariotomy.	Recovery	Retro-verted Uterus. Cyst size of a football. Cyst tapped Brown fluid evacuated. Omentum adherent to cyst.
188	S. C. W.	3-10-29	29	Ovarian cyst.	Ovariotomy and Ventral-suspension.	Recovery	Left ovary resected. Right ovarian cyst, size of an orange removed.
41	M. Y. M.	20-3-30	20	Right Ovarian Cyst.	Right Ovariotomy.	Recovery	Cyst partly adherent to abdominal wall. Brown fluid evacuated. Pedicle clamped and cyst removed. Section of ovary malignant adenoma.
49	C. L. C.	27-3-30	25	Left Ovarian Cyst.	Ovariotomy.	Recovery	Cyst size of full term pregnancy Tapped and fluid evacuated.

Table No. V.—*(Continued)* *Ovariotomy.*

No.	Name	Date	Age	Disease	Operation	Result	Remarks
78	C. K. H.	31-10-29	47	Ovarian Cyst (double).	Ovariotomy. (double).	Recovery	
82	C. C.	26-11-29	40	Ovarian Cyst.	Ovariotomy.	Recovery	Tumour—size of a fist felt to left of uterus which is displaced to the right and forwards.
85	L. S.	2-12-29	60	Ovarian Cyst.	Ovariotomy.	Recovery	Small retroverted uterus reflected to right—cystic tumour in pelvis—size of full term pregnancy.
86	T. K. Y.	2-12-29	25	Ovarian Cyst.	Ovariotomy.	Recovery	Tumour—size of a fist.
109	L. H. S.	20-1-30	47	Right Ovarian Cyst.	Ovariotomy (double).	Recovery	Size of a full term pregnancy, cervical polypi also removed.
121	M. U. C.	6-3-30	27	Ovarian Cyst.	Marsupialisation.	Recovery	18 pts. of blood stained fluid evacuated—inoperable malignant.
124	T. Y.	10-30-30	33	Broad ligament cyst.	Excision.	Recovery	4 cysts, each size of a goose egg.
135	L. T.	16-4-30	44	Ovarian Cyst.	Excision of left cystic ovary. ½ pt. of clear fluid, adhesions to sigmoid.	Recovery	

Table No. V.—*(Continued)* *Ovariotomy.*

No.	Name	Date	Age	Disease	Operation	Result	Remarks
57	C. L. P.	17-4-30	23	Left Ovarian Cyst.	Ovariotomy. Removal of Hydrosalpinx.	Recovery	Left ovarian cyst size of fœtal head removed. On right side Hydrosalpinx and ovarian cyst size of head removed.
58	L. H.	17-4-30	23	Left Ovarian Cyst.	Ovariotomy.	Recovery	Cyst of left ovary tapped. (Size of full term pregnancy). Pedicle twisted and congested.
G.C.H. 11	C. A. C.	21-5-29	46	Right Ovarian Cyst.	Ovariotomy.	Recovery	Uterus retroverted and displaced to the left by a cystic tumour size of a football.
19	N. S.	4-6-29	38	Left Ovarian Cyst.	Ovariotomy.	Recovery	Size of 2 footballs.
60	W. S. C.	24-9-29	25	Right Ovarian Cyst.	Ovariotomy.	Recovery	Tumour situated at anterior wall of uterus—size of a football freely movable.
75	W. L. H.	22-10-29	18	Left Ovarian Cyst.	Ovariotomy	Dead	(P. M. findings). Both kidneys cystic and atrophied

Salpingectomy and Oopherectomy.—(Continued)

No.	Name	Date	Age	Disease	Operation	Result	Remarks
G. C. H.							
64	C. F.	1-10-29	29	Retroversion Salpingitis.	Double Salpingectomy. Double Ovariotomy. Ventral-suspension.	Recovery	Right. Ovarian cyst—size of a golf ball.
74	S. K.	15-10-29	32	Retroversion Salpingitis.	Double Salpingectomy. Double Ovariotomy. Ventral-suspension.	Recovery	——.
29	L. K.	9-7-29	31	Retroversion Salpingitis.	Double Salpingectomy. Left Ovariotomy. Ventral-suspension.	Recovery	Right cystic ovary—punctured and sewn over.
132	L. W. H.	31-3-30	26	Salpingitis.	Adhesions separated and uterus suspended. Left cystic ovary resected.	Recovery	Good.
133	A. N.	7-4-30	25	Salpingitis.	Adhesions separated. Resection of part of left ovary, and excision of left tube, right tube and right ovary. Uterus suspended.	Recovery	Good.

Salpingectomy and Oopherectomy.—(Continued)

No.	Name	Date	Age	Disease	Operation	Result	Remarks
G. C. H.							
25	L. K.	18-6 29	27	Retroversion and Salpingitis.	Ventral-suspension. Double Salpingectomy. Left ovariotomy. Shortening of round Ligaments.	Recovery	———
26	L. S.	20-6-29	32	Retroversion Salpingitis.	Salpingectomy.	Recovery	———
41	L. Y.	30-7-20	33	Retroversion Salpingitis.	Right salpingectomy. Ventral-suspension. Shortening of round Ligaments.	Recovery	———
42	A. Y. Y.	20-8-29	23	Retroversion Salpingitis.	Double Salpingectomy.	Recovery	———
61	L. A. Y.	24-9-29	36	Retroversion Salpingitis.	Double Salpingectomy. Double Ovariotomy. Ventral-suspension.	Recovery	———
62	L. C.	1-10-29	36	Retroversion Salpingitis.	Double Salpingectomy. Double Ovariotomy. Ventral-suspension.	Recovery	Right. Ovarian cyst—size of a golf ball.

Table No. VI.

Salpingectomy and Oopherectomy.

No.	Name	Date	Age	Disease	Operation	Result	Remarks
T. Y. H. 189	L. M. L.	15-6-29	22	Salpingitis.	Salpingectomy with ventral-suspension.	Recovery	Tweedy's operation for sterility for left tube. Right salpingectomy ventral-suspension. Modified Gilliam.
193	Q. Y. S.	20-6-29	26	Salpingitis.	Resection of right tube with ventral-suspension.	Recovery	A hard mass in right tube resected. T.B.
208	Y. K.	20-8-29	47	Hydrosalpinx.	Resection of left tube.	Recovery	Hydro-salpinx size of a fist resected. Right tube and ovary adherent—inoperable. Myoma in anterior wall if uterus enucleated. Ventral-suspension.
223	L S. C.	28-8-29	28	Salpingitis.	Salpingectomy and Ovariotomy.	Recovery	Both tubes and right cystic ovary resected. Modified Gilliam. Two interior piles excised.
5	M. K. Y.	19-12-29	30	Salpingitis.	Salpingectomy and Ovariotomy.	Recovery	Uterus fixed to Pouch of Douglas Tubo ovarian cyst of both sides, size of a hen's egg dissected. Ventral-suspension Pouch of Douglas drained.
66	H. P. H.	29-4-30	24	Salpingitis.	Salpingostomy with freeing of adhesion and ventral-suspension.	Recovery	Dense adhesions. Left ovary cystic—resected. Left tube patent. Right ovary resected, salpingostomy done on right tube. Uterus suspended.

Table No. VII.

Prolapse.

No.	Name	Date	Age	Disease	Operation	Result
T. Y. H. 175	W. K.	13-5-29	42	Prolapse.	Anterior colporrhaphy. Perinæorrhaphy and ventral-suspension.	Recovery
186	C. R.	29-5-29	29	Procidentia.	Anterior colporrhaphy. Vaginal ventral-suspension. Circular amputation of the cervix and its reconstruction shortening of Mackenrodt's ligaments. Perinæorrhaphy.	Recovery
195	A. F.	18-6-29	30	Prolapse. hypertrophy of cervix.	Curettage. Vaginal suspension. Shortening of Mackenrodt's ligaments. Circular amputation of cervix and perinæorrhaphy.	Recovery
212	H. N.	11-7-29	40	Procidentia.	Prolapse operation under local anæsthesia. The abdomen was opened. Two broad ligament cysts and hydrosalpinx, each the size of a fist removed.	Recovery
233	S. W. H.	19-8-29	51	Prolapse.	Shortening of Mackenrodt's ligaments, circular amputation of cervix.	Recovery
262	J. A.	15-10-29	33	Prolapse.	Anterior Colporrhaphy. Amputation of cervix. Mackenrodt's ligaments shortened. Perinæorrhaphy. Ventral-suspension.	Recovery
251	C. C.	25-9-29	37	Procidentia.	Vaginal suspension. Mackenrodt's ligaments shortened. Amputation of cervix. Perinæorrhaphy.	Recovery

Prolapse.—(Continued)

No.	Name	Date	Age	Disease	Operation	Results
T. Y. H. 278	S. S.	5-11-29	44	Procidentia.	Vaginal Hysterectomy. Perinæorrhaphy.	Recovery
3	C. S. Y.	17-12-29	17	Procidentia.	Vaginal ventral-suspension. Colporrhaphy not performed.	Recovery
39	L. A. M.	13-3-30	25	Cystocele rectocele slight prolapse.	Anterior colporrhaphy. Mackenrodt's ligaments shortened. Perinæorrhaphy.	Recovery
63	L. A. M.	26-4-30	33	Procidentia.	Complete Operation for prolapse. Vaginal ventral-suspension and perinæorrhaphy.	Recovery
G. C. H. 34	Y. A. M.	16.7-29	37	Retroversion and prolapse.	Anterior colporrhaphy. Amputation of cervix. Shortening of Mackenrodt's ligaments. Reconstruction of cervix Post colpo-perinæorrhaphy. Vaginal ventral-suspension.	Recovery
53	N. S. Y.	3-9-29	28	Uterus (prolapse)	Anterior colporrhaphy—amputation of cervix. Mackenrodt's ligaments shortened. Vaginal ventral-suspension. Reconstruction of cervix.	Recovery

Prolapse.—(Continued)

No.	Name	Date	Age	Disease	Operation	Result
G. C. H. 87	T. Y. L.	3-18-29	22	Retroverted uterus. Tear of perineum. Transeverse laceration of cervix.	C. and D. shortening of Mackenrodt's ligaments Anterior Colporrhaphy. Amputation of cervix. Reconstruction of cervix. Post colpo-perinæorrhaphy.	Recovery
106	C. C.	31-1-30	--	Prolapse.	C. and D. Mackenrodt's ligaments shortened. Anterior Colporrhaphy. Amputation of cervix. Vaginal ventral-suspension. Reconstruction of cervix. Colpo-perinæorrhaphy.	Recovery
131	M. Y.	31-3-30	—	Prolapse.	C. and D. Mackenrodt's ligaments shortened. Anterior Colporrhaphy. Amputation of cervix. Vaginal ventral-suspension. Reconstruction of cervix. Colpo-perinæorrhaphy.	Recovery
116	M. H.	19-2-30	—	Prolapse.	C. and D. Mackenrodt's ligaments shortened. Anterior Colporrhaphy. Amputation of cervix. Vaginal ventral-suspension. Reconstruction of cervix. Colpo-perinæorrhaphy.	Recovery

Table No. VIII. *Miscellaneous Operations.*

No.	Name	Date	Age	Disease	Operation	Result	Remarks
T.Y.H. 2	W. F. H.	16-12-29	30	Myoma.	Enucleation.	Recovery	Myoma of anterior lip of Cervix size of a fist shelled out.
208	Y. K.	20-8-29	47	Myoma.	Enucleation.	Recovery	Small myoma in anterior wall of uterus enucleation.
266	T. W. H.	24-10-29	38	Fixed retroversion.	Ventral-suspension. modified Gilliam.	Recovery	The uterus, tubes and ovaries adherent to pouch of Douglas. Adhesions freed. Ventro-suspion. Modified Gilliam.
265	C. F. F.	24-10-29	42	Polypus.	Polypus twisted off.	Recovery	Pedunculated tumour jutting out from cervix.
199	C. H.	20-6-29	42	Polypus.	Polypus twisted off.	Recovery	Polypus size of an orange twisted off its stalk.
G.C.H. 2	L. S.	7-5-29	33	Fixed Retroversion and Salpingitis.	Laparotomy.	Recovery	Uterus found adherent to loops of intestiness and lying in Douglas pouch.

Miscellaneous Operations.—(Continued)

No.	Name	Date	Age	Disease	Operation	Result	Remarks
45	L. A. C.	5-8 29	58	Ventral hernia following abdominal operation 3 months ago.		Recovery	
100	K. F. W.	31-12-29	17	Ovarian Cyst.	Drainage of Cyst.	Went home against advice.	Cyst found adherent to peritoneum all round. Owing to bad condition of patient no attempt was made to remove the cyst.
66	W. M.	3-10-29	21	Vesico Vaginal fistula.	Plastic Operation.	Recovery	
107	C. K.	15-1 30	43	Epithelioma of clitoris with secondary glands.	Complete Exterpation of vulva and superficial and deep ingiunal glands of both sides.	Recovery	3 stage operation; ulceration of glands of right side. Section of both glands epitheliomatous.
112	L. L.	10-2-30	33	Salpingitis and retroversion.	Partial resection of both ovaries. Separation of adhesion to frimbiæ.	Recovery	Ventral-suspension.

Table No. IX. *Extrauterine Pregnancy.*

No.	Name	Date	Age	Disease	Operation	Result	Remarks
T.Y.H. 36	Mrs. R.	6-3-30	24	Extruterine gestation.	Right salpingectomy.	Recovery	Right tube pregnant removed. A large hæmatoma of the right ovary.
G.C.H. 3	M. D.	1-5-29	22	E. U. P.	Refused Operation.	Same	Uterus in front and deflected to the right. Tepder mass felt at left lateral fornix and pouch of Douglas.
12	H. Y. C.	21-5-29	31	E. U. P.	Excision of right tube. Removal of right ovary.	Recovery	Tumour-- size of a hen's egg felt in right lateral fornix.
18	H. S. C.	28-5-29	26	Ruptured extra-uterine pregnancy.	Excision of left tube.	Recovery	
56	T. Y. S.	13-9-29	24	Ruptured E. U. P. (right).	Excision of right tube and removal of right ovary.	Recovery	Pelvic hæmatoma.
67	W. A. C.	8-10-29	25	Ruptured extra-uterine pregnancy.	——	Recovery	
49	P. N.	13-8-29	27	Extruterine pregnancy.	Excision of left ovary and left tube.	Recovery	
108	W. C.	20-1-30	27	Ruptured extra-uterine pregnancy.	Excision of right ruptured ovary and tube.	Recovery	Blood clots from pouch of Douglas cleared.

Table No. X. *Mortality of Puerperal Sepsis Cases.*

No.	Name	Age	Date	Died	Diagnosis	Remarks
G.C.H.						
31	C. Y.	19	8-7-29	7 9-20-2½ hours after admission.	Puerperal sepsis. Acute lymphatic sepsis.	Patient admitted in a dying condition—delirious. Delivered 15 days previously in a neighbouring hospital.

A Short Report on Gynæcological Specimens.

(FROM THE SCHOOL OF PATHOLOGY UNIVERSITY OF HONG KONG)

As in previous years, examinations of specimens from the Obstetrical and Gynæcological Unit of the University were undertaken by the Reporting Department of the School of Pathology and the following is a summary of the investigations carried out during the last twelve months ending 31st May, 1930.

Blood for Syphilitic Reaction	486
Blood for Widal Reaction	12
Uterine Contents for Culture	3
Preparation of Vaccines	5
Tissues for Micro-sections	55

This gives a total of 558 specimens or the year.

Of the 486 samples of blood for Syphilitic Reaction 25 were positive, 3 doubtful and 458 negative. The test employed was a modification of the Szebs-Georgi introduced by Professor C. Y. Wang. For the Widal Reaction the microscopic method was used. Of the 12 samples of blood 2 were positive and 10 negatve.

Out of the 52 specimens of tissue submitted for microscoipcal diagnosis 27 were of tumour-growths while the rest were inflammatory conditions :—

Tumours		27
Carcinoma of Cervix	2	
,, ,, Clitoris	1	
,, ,, Ovary	1	
,, ,, Omentum (secondary)	1	
,, ,, Vulva	1	
Fibro myoma of Ovary	1	
,, ,, ,, Cervix	1	
Simple Cyst Ovaries and tubes		11
Papilloma of Vulva	2	
,, ,, tubes	1	
Teratoma of Ovary	1	
Inflammatory lesions		24
Inflammatory lesion of Cervix	3	
,, ,, ,, tubes	12	
,, ,, ,, uterus	4	
,, ,, ,, labium	1	
Placental scrapings	1	
,, ,, ,, labium	1	

AFFECTIONS OF THE EYE IN GENERAL PRACTICE.

by

R. Lindsay, Rea, B.SC., M.D., M.ch,, F.R.C.S.

Ophthalmic Surgeon to the London Lock Hospitals; Surgeon to Western Ophthalmic Hospital, etc.

The examination of the eye aids us greatly in localizing the seat of neoplasms in the brain.

As heretofore I will refresh my reader's memory with a brief reference to the anatomy of the middle portion of the brain, tumours of which usually involve some of the nerves connected with the eye. Illustration No. 1, shows the front view of the medulla, pons, and

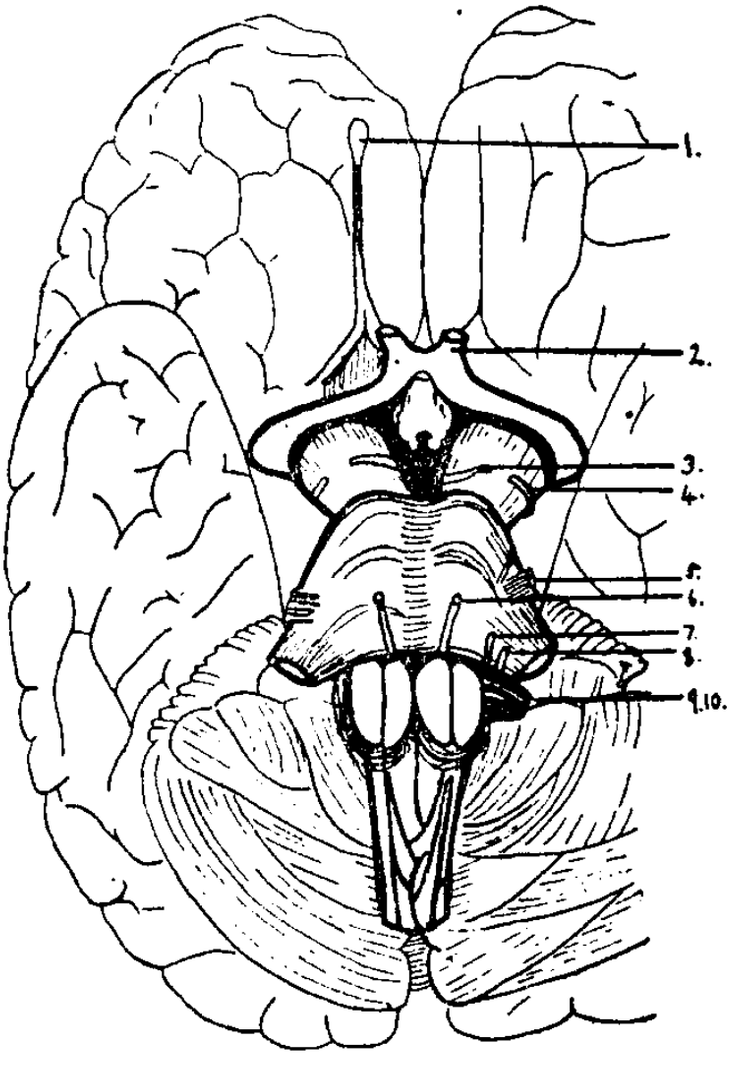


Fig. I.

the mid-brain the latter consisting mainly of the crura cerebri which will be seen to resemble two large rope-like strands emerging together from the upper portion of the pons varolii diverging as they proceed upwards to enter the cerebrum. At the point where each crus disappears into the corresponding side of the hemisphere it is embraced

on its outer side by the optic tract. From the inter-peduncular space emerge the 3rd cranial or oculo-motor nerves, this little space is therefore called the sulcus oculo-motorius. Winding around the outer side of each crus is seen the 4th cranial or trochlear nerve. From the ventro-lateral surface of the pons the 5th cranial or trigeminal nerve is seen to proceed, while from the area between the lower part of the pons and the upper part of the medulla the 6th, 7th and 8th cranial nerves are seen emerging closely together so that if a tumour or hæmorrhage implicates one nucleus most probably the other will be affected also.

The fibres of the pyramidal tract which in the pons form scattered bundles are gradually gathered together so as to cross over at the lower part of the pons and upper part of the medulla to the opposite side, this is known as decussation of the pyramidal fibres, so that a lesion affecting these pyramidal fibres on the upper part of the right side of the pons will produce a paralysis on the left side of the body and vice-versa.

Looking at the illustration one can quite easily understand that a lesion which affects the nucleus of the 3rd, 6th, 7th, or 8th nerves will most probably also affect the pyramidal bundles in their neighbourhood, so that there will be not alone a squint produced, but there will be paralysis of the limbs or of the face and limbs as well.

(1) There are several syndromes worth remembering, the first is known as " Weber's " syndrome which is a paralysis of the 3rd nerve together with a hemiplegia of the opposite side, there will also be some paralysis of the face and tongue. Where would be the situation of such a lesion? A glance at the illustration will show that such a lesion will be situated at the junction of the pons and crura or mid-brain.

(2) " Benedikt's " syndrome.—This is a paralysis of the 3rd nerve together with tremor of opposite limbs. It is a lesion of the red nucleus which is situated at the upper part of the mid-brain, the part which receives the fibres from the superior cerebellar peduncle.

(3) " Millard Gubler's " syndrome.—This is a lesion in the lower part of the pons producing a paralysis of the face and of the opposite limbs. The eyes are not affected as the nucleus of the sixth nerve escapes damage.

(4) " Foville's " syndrome.—Here the sixth nerve nucleus is involved so that there is a paralysis of the external rectus of that side. Supposing it to be the right side, the right eye will converge as the internal rectus will be unopposed by the paralyzed external rectus, therefore the right eye cannot turn to the right and this is expressed as loss of conjugate movement to the right. The 7th Cranial nerve

is involved so that there is paralysis of the same side of the face and there will be a hemiplegia of the limbs on the opposite side.

(5) "Cerebello-Pontine" syndrome.—The last post-mortem of this condition which I examined showed the cerebello-pontine tumour pushing its way along the 8th cranial or auditory nerve and gradually invading the internal auditory meatus, its progress had practically severed the 6th, 7th, and 8th nerves. It had pushed the cerebellum aside. The symptoms of such a condition then are plainly understood. Paralysis of the 6th serve supplying the external rectus allows the eye on that side to squint inwards, the 7th nerve is paralyzed, that side of the face will therefore be immobile, while on account of the destruction of the 8th cranial nerve there is deafness on the same side. There will be pressure on the cerebellum producing nystagmus together with a coarse inco-ordination of movements.

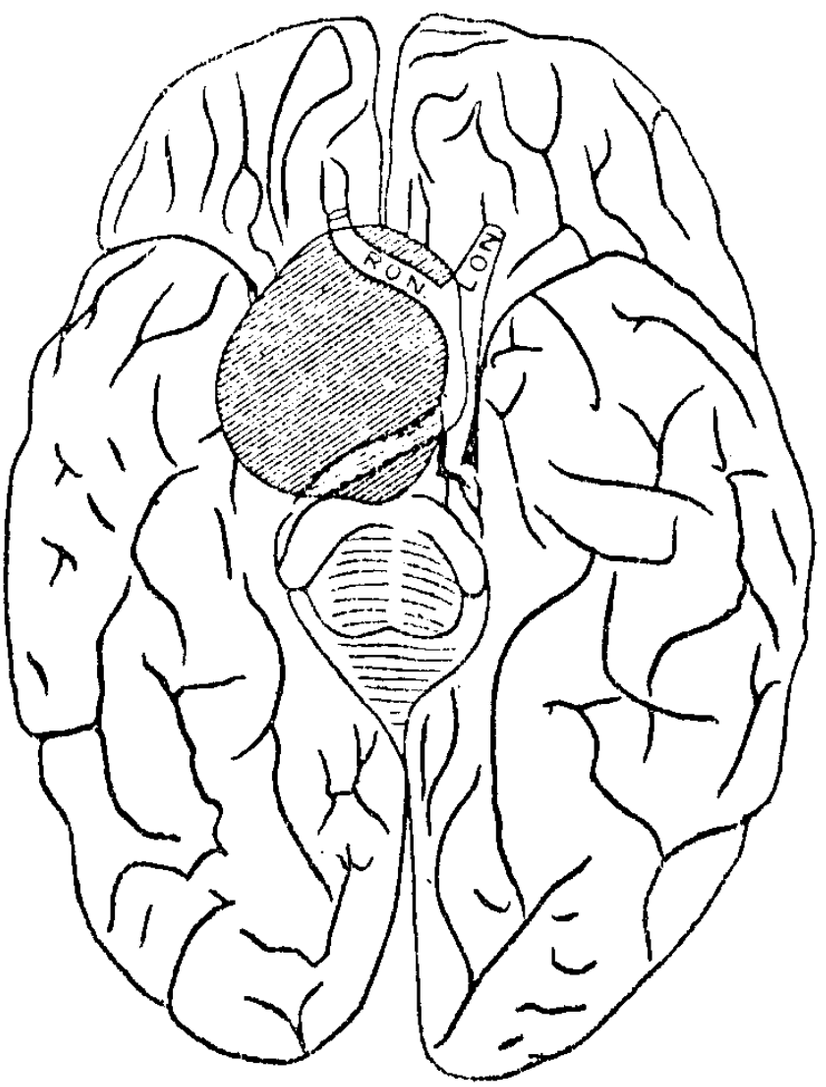


Fig. II.

Winding around the outer side of the crus is seen the 4th nerve a thin fine structure. This comes from the region of the corpora-quadrigemina so that if this part is the seat of the lesion there will be loss of up and down head movements together with a diplopia produced by paralysis of the superior oblique.

In order to show the optic tracts in the illustration the temporal lobes have been pulled apart. Tumours in this region may press on the optic tracts, they produce loss of visual field and sometimes one half of the entire field is obliterated. This is known as a complete hemianopia. Jackson described uncinates attacks or dreamy states in such cases—there are visual halucinations sometimes together with generalized convulsions which form a complete symptom-complex of temporal lobe tumours. A tumour situated in the frontal lobe may not produce optic neuritis or as it is now called papillœdema (" Paton ") but if the tumour affects the cerebellum the papillœdema is intense, therefore we cannot always find the three cardinal symptoms of tumours of the brain to be present. These three you will remember are :—

1. Optic Neuritis, or as it is called Papillœdema.
2. Vomiting.
3. Headache.

Illustration No. 2 is taken from a brain which I recently examined, it shows the presence of a tumour the size of a tangerine orange at the base of the brain intimately associated but not forming part of the pituitary gland. The patient, a man aged 51, came to me on February 5th, 1925, complaining of blindness of the right eye. The history was that the sight of the eye had failed somewhat suddenly just before Christmas. On examining the eyes nothing abnormal could be found except that the edge of the right disc appeared very slightly blurred. Although the edge was not quite sharply defined there was not a definite papillœdema present, the eye was quite blind, the central vision in the left eye was normal, and its field of vision was normal also. There was no change in the appearance of the vessels that would suggest a cause for the failure of vision in the right eye. The Wassermann reaction of the blood-serum was negative. The examinations by the rhinologist and neurologist were negative also. One thing I omitted to do, namely, to make an X-ray examination of the skull, the Hospital unfortunately was not well enough off to possess such a a valuable piece of apparatus. On March 9th the right disc appeared slightly pale. The general health was perfect, and each day his usual work was done. Then for the first time on May 11th I noticed slight blurring of the disc, and three days later there was a definite papil-lœdema in the left eye. Suspecting its cause the patient was then

transferred to the West End Hospital for Nervous Diseases. A radiogram was taken which showed at the base of the skull a very much flattened sella turcica; (see illustration on No. 3 which is the actual drawing of the bone.) The shadow of the pinna of the ear caused some confusion making it appear to the physician as if a partly ossified tumour occupied this region, but such an appearance I had seen often in the radiograms of wounded soldiers in France. A decompression operation was performed, but the patient died two days later from meningitis extending from the base of the brain. The post mortem examination, including that of the brain was made by Dr. Carnegie Dickson. The actual specimen showed a large tumour, forty millimetres across, occupying the pituitary region. On cutting it was found to be an endothelioma intimately connected with the wall of the cavernous sinus. The tumour had flattened out the fossa. Extending upwards on the outer side of the right optic tract, it had pushed the tract across to the left side. The right optic tract and optic nerve were stretched and flattened, lying on the tumour, hence the blindness of the right eye. The left optic nerve and tract were not stretched nor pressed upon by the tumour. Not only was it pressing on the right optic nerve, but it pressed against the right optic foramen so that there was not the slightest possibility of cerebro-spinal fluid passing down the sheath of the right optic nerve to the eye, hence the

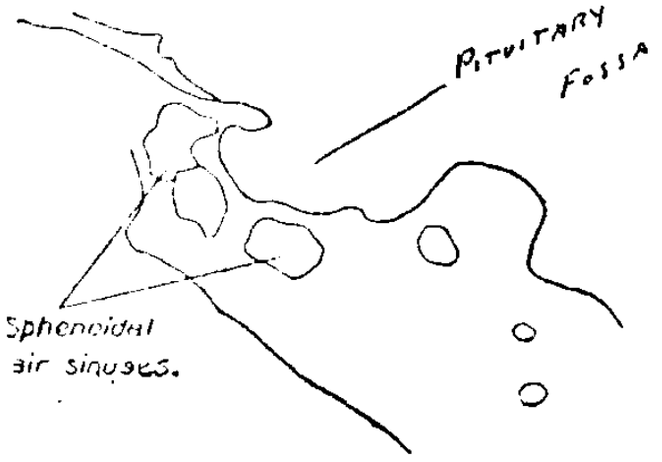


Fig. III.

absence of papillœdema. It was only when the tumour continued growing and the intra-cranial pressure arose that papillœdema was found in the left eye. The entire absence of symptoms suggestive of cerebral lesion was remarkable. The patient sang a solo at his church on the Sunday before his admission to Hospital.

Many surgeons including the late Sir Victor Horsley, have believed that the optic neuritis by its earliest appearance or greater severity on one side, is diagnostic of the side on which the tumour is. But this sign is of much greater value in the case of intracranial abscess.

There may be optic neuritis on the side of the abscess only. Partial paralysis of the 3rd nerve, *e.g.*, ptosis, with a dilated pupil on the same side, is almost pathognomonic of an ipso-lateral cerebral or cerebellar abscess.

In tuberculous meningitis one-fourth of the cases show a slight optic neuritis. But tubercle of the choroid is much more frequent. Parsons believes that tubercle of the choroid is not so uncommonly found in tuberculous meningitis as is usually thought. But it is often found only a day or two before death. Round yellow spots are seen near the optic disc, or, less frequently, scattered over the choroid.

In epidemic cerebro-spinal meningitis there may be a descending neuritis causing a swelling of the head of the optic nerve without œdema. Other eye symptoms are often found, *e.g.*, conjunctivitis, swelling of the lids, photophobia, pupils unequally dilated or contracted, keratitis or ulceration of the cornea, or even a uveitis may be present.

During the last few years the epidemic disease known as encephalitis lethargica has been brought very prominently before the mind of the profession. Along with the acute general infection there are found paralyses of the cranial nerves. The investigations of the Local Government Board, London, showed that 75 per cent. had some ocular palsy. Ptosis was the most common defect. In 25 per cent., it was unilateral, papillœdema was rare. Foster Moore did not find any fundus change in seventeen cases which he examined ophthalmoscopically. The ocular paralysis passes away soon, but some lasted as long as nine weeks after the onset of the disease. The writer has observed some cases of failure of convergance which defect has remained permanently. The most advanced changes were found in the region of the third nerve nucleus in the mid-brain.

THE HYDROLYTIC THEORY OF HYDROCHLORIC ACID SECRETION IN THE STOMACH.

by

Lindsay T. Ride and S. Y. Wong.

From the Dept. of Physiology, The University, Hong Kong.

During our fractional test meal investigations on normal Chinese students, we came up against some facts the explanations of which demanded enquiries into the chemical method of hydrochloric acid secretion.

The method whereby animal cells can secrete a strong mineral acid has always been a matter of wonder and conjecture to zoologists and still awaits solution. The finding of hydrochloric acid in the gastric juice of all vertebrates is the universal rule, but in animals below that phylum, none has as yet been demonstrated with any certainty. In all invertebrates, gastric digestion is of the pancreatic type, the enzymes working in an alkaline medium; even in some invertebrates however, observers have reported the presence of acid salts in the gastric juice. It will thus be seen that gastric digestion as we know it in vertebrates, is a comparatively late zoological development, and perhaps achlorhydrics should not be looked upon as something totally irregular in the animal kingdom, but rather as living anachronisms pointing unmistakably to a recent advance in protein digestive methods.

This does not mean however that the world had to await the advent of the vertebrate before witnessing the miracle of turning alkaline fluids into a strong mineral acid, for certain molusces have long been known to have the power of secreting sulphuric acid. Baylis (2), in discussing the secretion of this acid by a large mollusc, *Dolium galea*, states "It seems desirable that the fact of secretion of 5 per cent. sulphuric acid should be be reinvestigated." He then goes on to say "the production of hydrochloric acid in the stomach (of vertebrates), of decimolar or even higher concentration, has not yet received a satisfactory explanation." Carlson (3) in dealing with the 'border cells' in his review on *Gastric Secretion* states that, although the evidence is fairly conclusive that these cells produce the hydrochloric acid of the stomach, "we do not know how or where these cells produce it."

In general the theories of hydrochloric acid secretion are based on one or other of the following, (*a*) the acid as such is actually formed in the oxyntic cells of the gastric mucous membrane and then poured out into the stomach along with the rest of the gastric juice, or (*b*) the acid is secreted by these cells in the form of a chloride, and that it is not till after this chloride has reached the lumen of the stomach that the acid is definitely formed. The facts are that from an alkaline

fluid such as the blood, certain cells of the gastric mucosa are able to separate off an acid of such concentration that, if it gained entrance to any animal cells, it would certainly prove fatal to them.

An aqueous extract of the stomach mucous membrane is neutral in reaction and therefore the acid cannot be stored in the cells as such. Neither can it be stored in the cells in an inactive form for although the cells contain more chlorine than any other body cells, there is not enough to account for the large amount of hydrochloric acid found in the gastric juice. Nencki and Schumova-Simonowski (5) give the percentage weight of chlorine found in the various wet tissues as follows:—

Stomach mucosa	0.093
Panniculus adiposus	0.076
Intestinal mucosa	0.040
Bone marrow	0.034
Muscle	0.033
Bones	0.033
Kidney	0.032
Liver	0.025

Heidenhain, by means of pouch experiments, proved that the fundic secretion was definitely more acid than that of the pyloric end, and Pawlow, by similar experiments in which the nerve supply was left intact, proved beyond doubt that the secretion from the fundus is acid in reaction. Furthermore the chlorine content of the fundic mucosa is found to be greater than that of the pyloric mucosa. It is now generally agreed that the place of ultimate origin of the acid is the oxyntic or parietal cells which are limited in their distribution to the glands in the wall of the fundus of the stomach.

Claude Bernard tackled the question by injecting potassium ferrocyanide into one vein of a dog, and into another vein, iron lactate. In the presence of acid these two substances form prussian blue, and on killing the dog the prussian blue was found in the ducts of the fundus glands and in the stomach, but not in oxyntic cells. Fitzgerald repeated this work of Claude Bernard at a much later date and, although finding the blue colour in the stomach and in the necks of the glands, she found some of the parietal cells stained with blue. She therefore concluded that the secretion was acid, whereas Bernard's conclusion was that it was alkaline. In 1912 Harvey and Bensley found that although some of the parietal cells took the blue stain, the vast majority of them did not, and finding too that many other cells in the body which were not concerned with the formation of acid showed the prussian blue stain, they came to the conclusion that the results obtained by this method were unreliable. Other workers have brought forward evidence which still leaves the question in this undecided state, but

taken on the whole it seems certain that whether the secretion be alkaline or slightly acid, it does not contain acid in amounts to account for the concentration that we find in test meal observations.

It was Foster who first suggested that the acid was actively secreted by the oxyntic cells as an organic compound, which compound was decomposed in the lumen of the stomach, setting free the acid. Later Harvey and Bensley came to the conclusion that the secretion of the fundus glands was very rich in organic matter and was slightly alkaline or neutral in reaction, but when the foveola was reached the acid was formed. These observations fit in well with Foster's ideas.

This brief account shows the unsatisfactory state in which for many years our knowledge of the exact method of acid formation in the stomach has been stagnating. In this paper we hope to produce evidence of a totally different nature from that of previous workers, which evidence seems to us to add weight to Foster's theory, and we have formed the opinion that the oxyntic cells secrete the acid in the form of a compound of hydrochloric acid and a weak base, and that this chloride is hydrolysed in the stomach, the basic portion being reabsorbed leaving the hydrochloric acid free.

Experiments.

The method of procedure was the same as that explained in a previous paper (7). A test meal consisting of 50 c.c. of a 2% arabinose solution made up to 500 c.c. with the ordinary gruel was given, and the free and total acid, total chlorides and pentose dilution were estimated in each fraction according to the method described in the above mentioned paper. In the cases quoted, the method of double test meal was used, and the following are the details of each individual case.

In Case IV the stomach was found to be empty 1½ hours after taking the meal, only about 5 c.c. being obtainable as sample G. Accordingly a second meal consisting of 475 c.c. of the usual gruel was given without removal of the tube and a second series of samples H, I, J, K and L were obtained and analysed. This method of double test meal was repeated in Case V, but here the emptying was so rapid that three meals were able to be examined in four and a half hours; the gastric digestion of the last meal however was not completed when the investigation had to be discontinued. The first meal consisted of 450 c.c., the second a similar amount, and the third 400 c.c. each of the usual gruel.

Similarly, at H, 1¾ hours after the first meal in Case VI, the stomach was found to contain only 6 c.c. which was removed for estimation. Again a second meal consisting of 450 c.c. was given, but this time it contained 1 gm. of ammonium chloride dissolved in it. This strength of ammonium chloride was found to be quite palatable and the meal consequently taken with the same ease as the first one.

In Case VII the stomach was emptied with sample K, 2½ hours after the first meal which consisted of 450 c.c. of the gruel. The second meal consisted of 425 c.c. but also contained 2 gms. of ammonium chloride in solution, and was therefore passed into the stomach through the tube in order that the salty taste should have a minimum effect on the secretion of gastric juice.

Results.

The procedure whereby the dilution was estimated by means of the pentose was followed on 13 separate occasions on 5 different subjects, and results obtained on four of these occasions are given below.

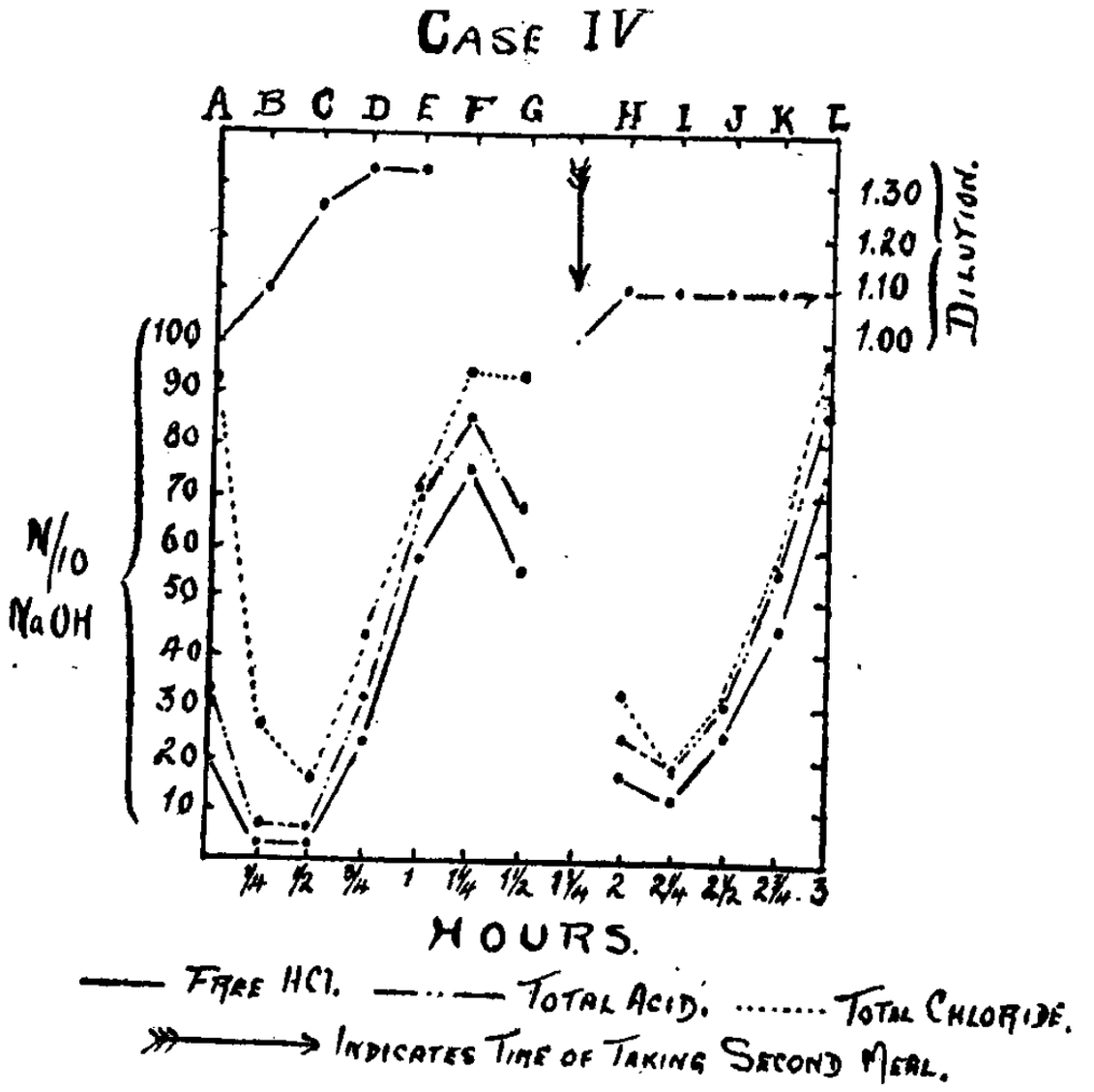


Fig. 1.

An example of a double test meal without removal of the tube. The curves at the top of the graph in each case show the dilution of the stomach contents as measured by estimating the concentration of the pentose in each sample.

In Case IV (fig. 1) it will be seen that between three-quarters of an hour and one hour after the meal, the pentose estimations showed

that the food in the stomach did not undergo any dilution and yet both of the acid curves and the total chloride curve showed a very marked rise during this period. In the second meal it will be seen that the dilution of the pentose rose during the first quarter of an hour, but for the next hour samples I, J, K and L showed no change in dilution of the pentose at all. It will also be noticed (especially if the curves for the two meals are superimposed on one another as in fig. 2, q.v.) that except in the first one or two samples, the curves are remarkably coincident.

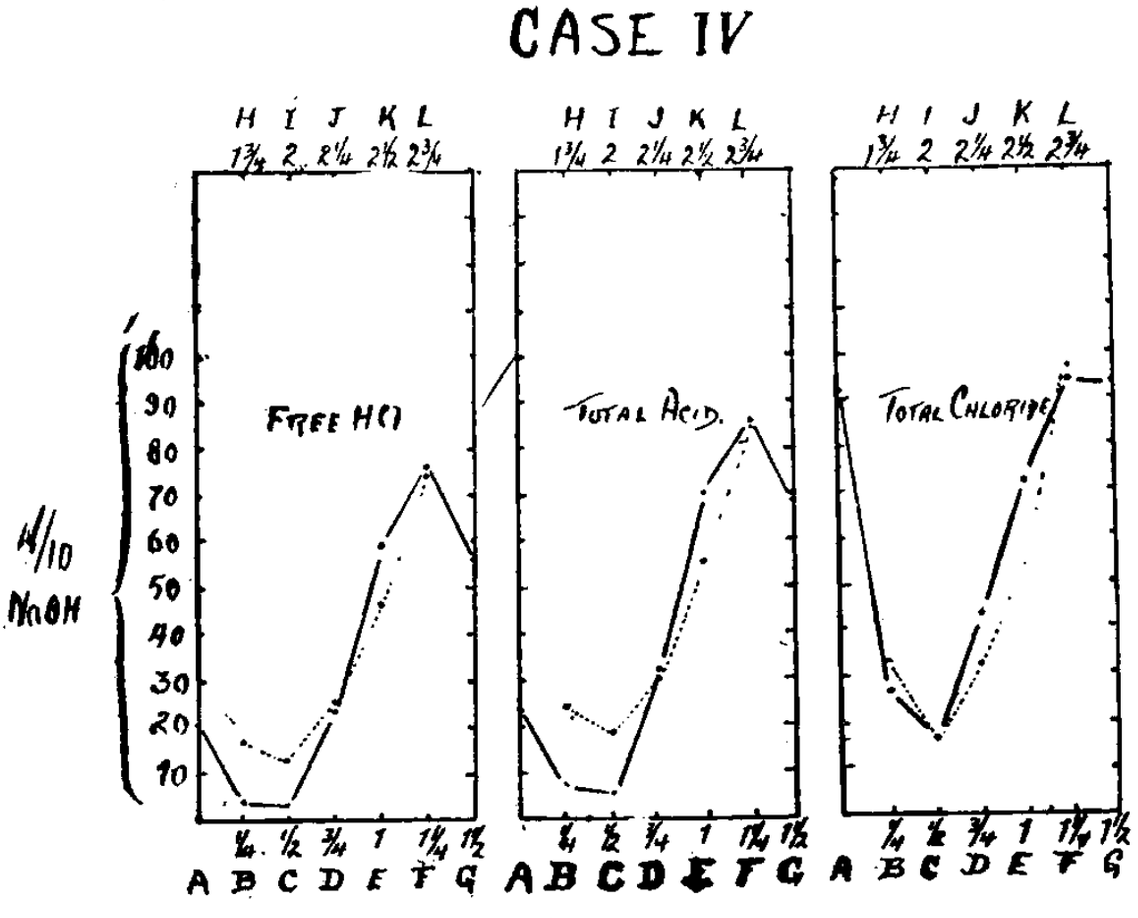


Fig. 2.

These are the curves of the same experiment as shown in fig. 1. In this case, the continuous line represents the 1st meal, the dotted line the 2nd. In the graph on the left the free acids of the two meals are plotted, in the middle one, the total acids and on the right, the total chlorides. The samples and corresponding times of the 1st meal are shown underneath the graphs, and those of the 2nd meal are marked above the graphs.

Case V (fig. 3) gave very similar results. One hour after the first meal was given the stomach was found to be empty, no gastric fluid being obtainable at all. Dilution estimations were carried out on only one of the samples. The second meal was given one hour and a half after the first, and here the third and fourth samples showed no change at all in the dilution of the stomach contents, although again both of the acid curves and the total chloride curve all showed a very marked rise. Here too the stomach emptied in 1½ hours and so 2 hours after the second meal a third one was given. On this occasion the

second, third and fourth samples showed no change in the dilution, while yet again there was a marked rise in the acid and total chloride content. It should be noticed here that between the second and third fraction the three curves are parallel but between the third and fourth the free and total acids, though parallel, show only a small rise, whereas the total chloride curve continues its previous steep ascent.

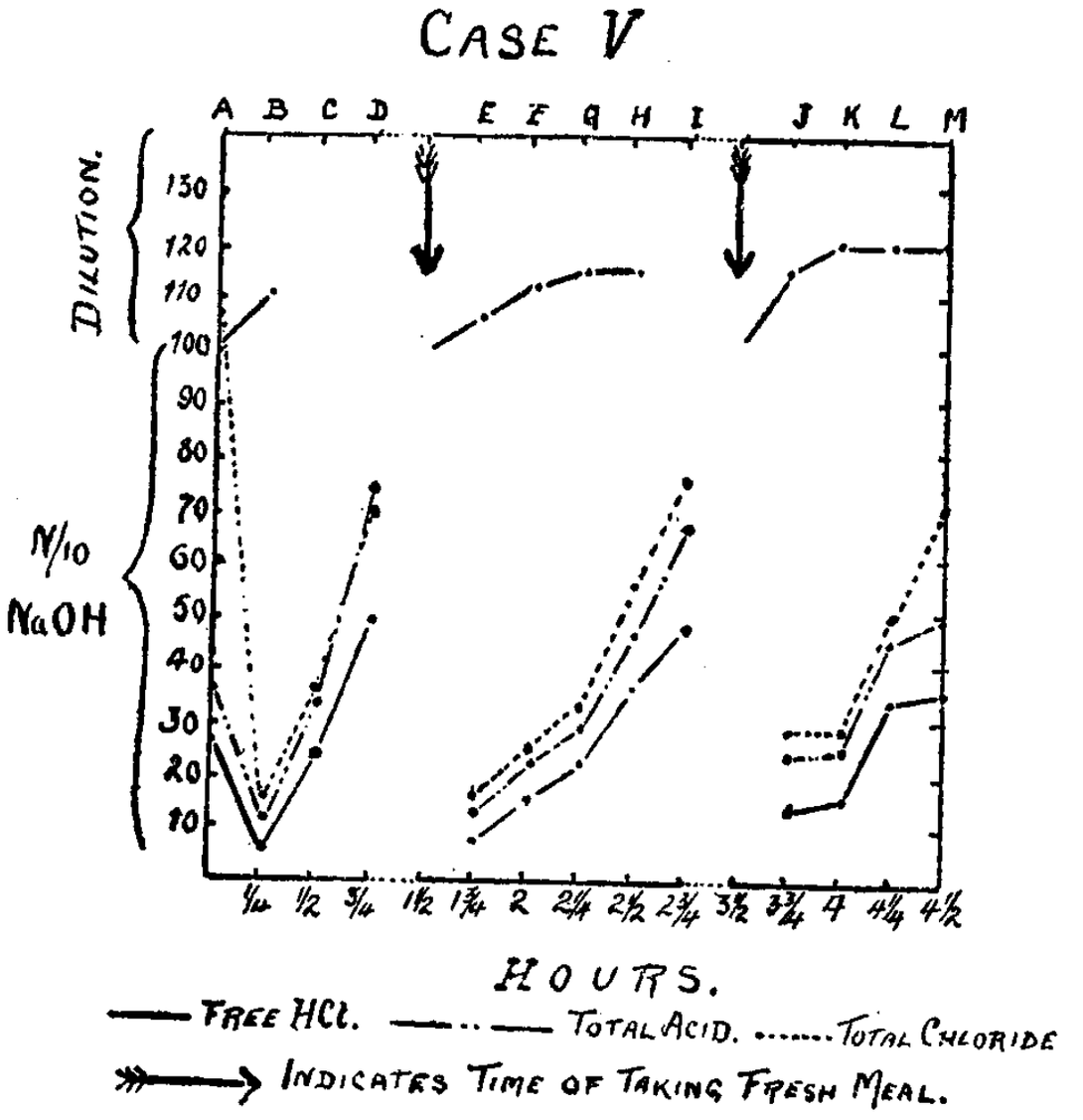


Fig. 3.

A case of triple test meal showing (*a*) the similarity of curves and (*b*) that the secretion of acid in second and third meals is slower than in the first.

In fig. 3 it will also be noticed that the general trend of the curves given by each meal is similar, this being strikingly so in the case of the total chlorides, especially after the fisrt sample in each meal. Furthermore it should be noticed that what variation there is, is such as to make the values in the second and third meals lower than those of the corresponding samples in the first meal.

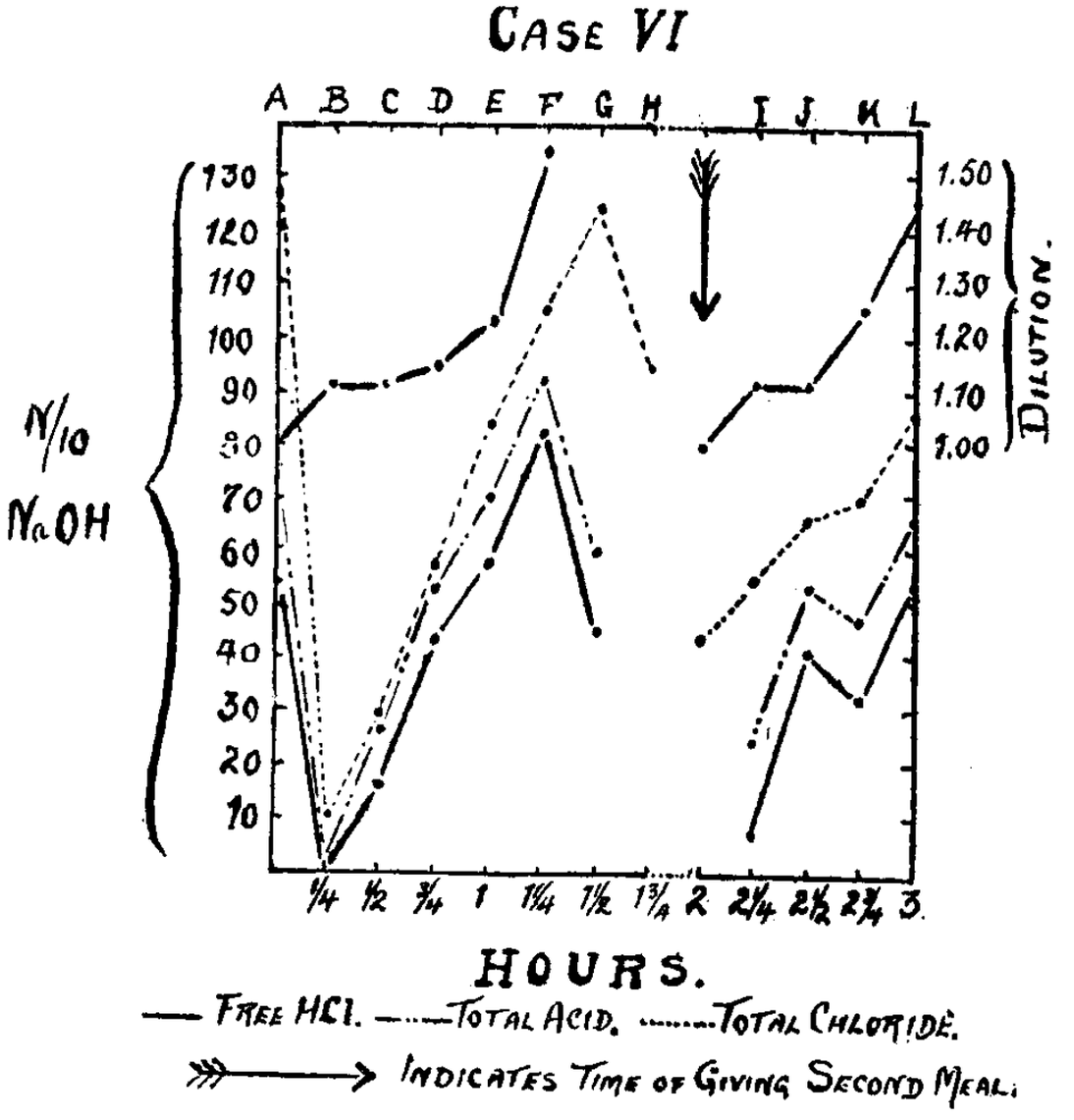


Fig. 4.

A case of double test meal in which the second meal contained 1 gm. of ammonium chloride. The top (continuous) curve in each case represents the dilution.

Fig. 4 provides us with examples of the same kind; the dilution remains the same between samples B and C, and between I and J, and rises very slightly between C and D. In all these the other curves rise very rapidly.

Case VII (fig. 5) shows that on four occasions there was an absence of change of dilution in consecutive samples, between C and D, F and G, H and I and L and M. The other three curves show a definite rise on each occasion with the exception of the two acids which show a fall between F and G.

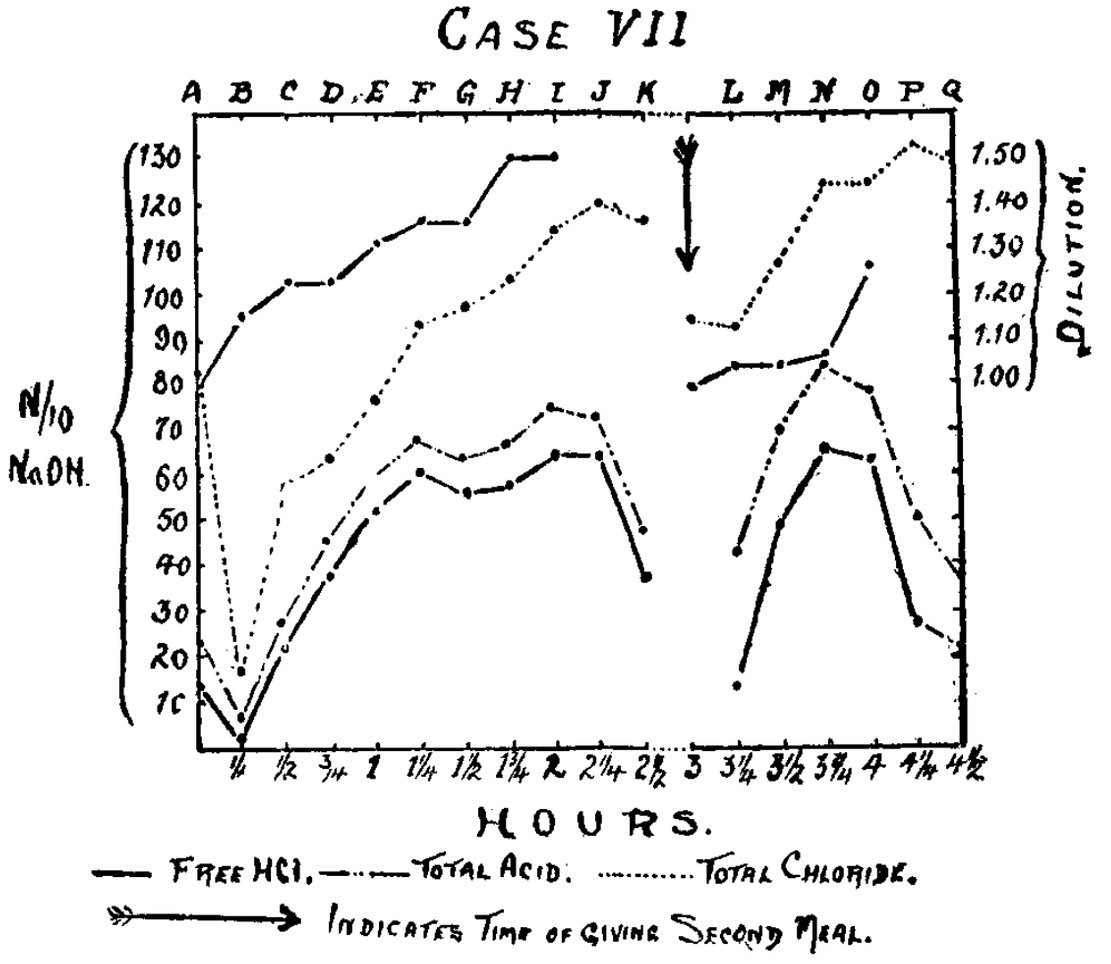


Fig. 5.

A case of double test meal in which the second meal contained 2 gm. of ammonium chloride. The upper continuous curve in each case represents the dilution.

Discussion.

By means of the dilution estimations, it is seen that the stomach contents become, in general, more and more dilute as digestion progresses. This goes on until, just before the stomach is empty, its contents are practically merely digestive fluids that have been added since the meal was taken. From the small number of curves already done, it is not yet possible to state whether there are any definite types of dilution curves, common to different individuals, or whether they are all essentially different. We have examples of cases where the dilution is small at first, but increases more rapidly with each successive sample, whereas on the other hand there are cases in which the increase in dilution, although great at first, gets smaller and smaller in each successive sample.

In no case was the dilution in one sample ever less than its predecessor, but, as mentioned above there were many examples where the dilution was the same or just slightly greater. What are the possible sites of origin of this diluting fluid? They are (*a*) salivary

glands (*b*) glands of the œsophagus and stomach, and (*c*) regurgitated duodenal contents. Throughout our experiments dilution due to the first cause was reduced to a minimum by the subject continually expectorating into a beaker provided for the purpose. It should be noted here that addition of saliva would result in a rise in the dilution curve, and either a definite fall or a diminution in the rate of rise of the acid and chloride curves. Dilution due to the last cause would also be shown by the rise in the dilution curve, and in the acid curves a corresponding absolute fall or a noticable deviation from the steady upward trend of the curves. Here however, the total chloride curve would vary in a manner totally different from that of the acid curves. The acid curve variations are due to neutralization plus dilution; the chloride curve variation is due to the change in dilution alone. An excellent example of the rise in dilution due to regurgitation is shown in fig. 4, samples J and K. Here the acid curves have both fallen, the dilution has risen, and so has the total chloride curve. Where therefore we see the acid and chloride curves all rising in a steady manner we can assume that there has been no gross regurgitation, and applying this principle we see that in none of the cases mentioned above where the dilution remains unchanged, has there been any gross regurgitation.

Diluting fluid from the glands of the œsophagus and of the stomach, although very different in character is considered under the one heading because it is impossible to separate them practcically. Since the gastric juice contains no pentose, its secretion must be accompanied by a rise in the dilution curve as measured by our method, but the change in the other curves will depend on the concentration of chlorides and acid in the secreted fluid. If the secreted fluid contains hydrochloric acid only and in the same concentration as that already in the stomach, the curves of both acids and chlorides will remain horizontal; if there is acid alone in the secreted fluid, but in greater concentration than that already present in the stomach, the curves will show a parallel rise in each case. (This would not necessarily follow if the chloride concentration were much greater than the acid and the concentration of the acid in the added fluid happened to fall between these two, but our experience is that this happens very rarely in a normal investigation for the chloride curve is nearly always very close to, and remarkably parallel to the acid curve for at least the first hour. The significance of this and the ultimate deviation of the curves we shall deal with in the next paragraph). If there be acid alone in the secreted fluid, but in a lower concentration than that already in the stomach, there will be a corresponding fall in the chloride and acid curves.

But this parallel rise in these curves would be just as efficiently explained if the secretion contained nothing but chloride and if the

acid could be formed from the chloride in the stomach. If both acid and chloride were secreted simultaneously in the juice this persistent parallelism would not be so likely to occur. From this argument therefore we are inclined to think that in the early part of digestion the gastric fluid secreted contains either acid or chloride and not both. But we still have to explain the latter part of the curves where we get a marked deviation of the acid from the chloride curve. The chloride curve generally gradually attains a height which Apperly (1) has named the 'chloride point,' and this height is generally maintained by the chloride curve after the meal, while the acid curves gradually fall. There have been two explanations put forward for this, one is that regurgitation takes place and neutralises the acid, but as we have seen above, there would also be a fall in the chloride curve as well as in the acid curves, due to the diluting of the chlorides by the added intestinal juice; there would also be a rise in the dilution curve. Fig. 5, case VII, samples F and G show that this is not always the explanation for there is no rise in the dilution corresponding to the fall in acids. The other explanation is that the stomach ceases to secrete acid, and substitutes for it, sodium chloride. This is the theory advocated by MacLean and Griffiths (4). They contend that the gastric glands secrete at all times 'a fluid having a fixed concentration of chloride ion' and 'that the chloride ion brought to the glands by the blood as sodium chloride is secreted at a definite fixed concentration, part of it unchanged as sodium chloride and part changed into hydrochloric acid and that the extent of this change governs the acidity of the secreted juice.' We hope to discuss some of the evidence for this (carbon dioxide estimations) in a later paper, but as far as this paper is concerned, we have seen that the type of parallel chloride and acid curves one so often finds is more likely to happen when the stomach secretes either acid alone or chloride alone and not both together. Furthermore, the point where the acid curves begin to deviate from that of the chloride is where the stomach is presumably secreting strong acid. This would therefore mean that the stomach suddenly switches over from secreting a highly concentrated acid to a highly concentrated chloride. It seems to us much more reasonable to expect to find that the actual secretion contained but one type of chloride all through digestion, and that this salt was converted into acid in the stomach itself.

We are now in a position to examine the major evidence for this theory. As far as we have reasoned at present, we have seen that any change in the acid or chloride curves due to addition of acid or chloride must be accompanied by a rise in dilution and yet we have cited definite examples of cases where there is an undoubted rise in acid concentration without any change in the dilution curve. During this period the acid and chloride must have been secreted by a process which leaves the ***resultant volume of fluid equal to the initial volume.*** If the chloride

be secreted as the salt of a weak base XOH, it will be hydrolysed in the stomach according to the following equation

$$XCl + HOH = XOH + HCl$$

If the mucous membrane of the fundus is permeable to the base XOH, it would drive this action from left to right and the HCl which is not absorbed would be left in the stomach. If now the XOH is absorbed in the same concentration as the XCl was secreted, the volume of the fluid in the stomach would be exactly the same as at the beginning of the secretion whereas the acid and chloride concentrations would have risen.

Because the gastric mucosa is very permeable to ammonia and because of its large ammonia content we chose ammonium chloride as our example of the form in which the acid is secreted; but we by no means contend that this is the actual substance secreted. We rather suspect a much more complex organic compound, and we are about to try the effect of the hydrochlorides of various amino acids. If acid can be formed from such chlorides, it is obvious that the addition of ammonium chloride to the meal should result in an increase in acid formation. First of all Case IV was given two identical meals according to the method of double test meal, and figure 2 shows how close the resulting curves are. Case V, figure 3 was given three meals with again very similar curves resulting. If anything, succeeding curves tend to be lower than preceding ones, at any rate they are definitely not higher. (This is shown graphically in figure 2 where the graph of each constituent of the second meal is superimposed on that of the corresponding constituent of the first meal). When however ammonium chloride is added to the second meal, the acid curve is noticably higher as figure 4. Case VI and figure 5. Case VII show.

Since the presence of ammonium chloride is the only difference between the two meals it is reasonable to suppose that it is the cause of the only change found in the results, namely the rise in the acid curves. In our series we have many other examples of this effect of ammonium chloride on the acid curves.

That this rise is an absolute one and not merely relative is shown by the fact that the actual amount of acid secreted in each quarter of an hour is greater when ammonium chloride has been added to the meal than when it has been omitted. Furthermore it is generally found that the effect of ammonium chloride lasts generally for about three quarters of an hour or an hour only.

To explain the fall in the acid curves towards the end of the meal there is, under the hydrolytic theory, no need to suggest a change in type of secretion nor is there any necessity to fall back on regurgitation (although this certainly does occur). Towards the end of the meal the amount of absorbed base accumulating in the membrane cells

must tend to retard the rate of absorption; this would result in less acid being formed from the secreted chloride, and while the total chloride curve would thus remain up about the same level, the acid curves would show a fall simultaneously with the continued rise in the dilution curve. Furthermore, by applying the Law of Mass Action to the above equation we see that the mere rise in acid concentration itself will tend to decrease the amount of dissociation of the secreted chloride, and hence the accumulation of acid alone tends to regulate its own formation.

Additional secretion of chloride in the same concentration as that found in the stomach will therefore result in a rise in the dilution, a fall in the acids and will not affect the chloride curve which will remain at the level of the chloride point.

On this theory we see that the variations in acid curves found in different patients or even in the same patient at different times would be accounted for by a variation in the permeability of the gastric mucosa to ammonia or the basic substance actually involved. A higher permeability would produce hyperchlorhydria and similarly low permeability would produce hypochlorhydria. If the mucous membrane is impermeable to the base or if the chloride is secreted in a non-hydrolysable form we should have a state of achlorhydria. This theory further explains why the presence of large quantities of mucus in the stomach causes a low acid curve and that if the mucus be removed, a normal curve is often obtained; for the mucus prevents the gastric fluid from coming into intimate contact with the membrane cells and therefore hinders the absorption of the base. The clinical application of this theory was treated more fully by one of us elsewhere (6).

The method of double and triple test meals which we have used has shown the effect of quickly repeated meals on gastric secretion. Many of our cases show that the acid curves of the second and third meals tend to be lower than the first, and this affords scientific proof of the wisdom of the treatment of hyperacidity in peptic ulcer cases by giving small meals at frequent intervals. This is definitely conducive to lowering of the acid secretion especially in the first three quarters of an hour which is as long as gastric digestion would last with very small meals.

CONCLUSIONS.

By the addition of pentose to a fractional test meal, it is found that during gastric digestion the stomach contents become more and more diluted by the addition of digestive fluids.

As the result of measuring the dilution of the pentose we suggest that the acid of the stomach is secreted as a chloride of a weak base. The chloride undergoes hydrolysis in the stomach, the basic portion being reabsorbed, leaving hydrochloric acid free.

The fall in the acid curves towards the end of digestion is due to the lessened absorption of the basic ion, the chloride concentration remaining level while that of the acids falls.

SUMMARY.

1. A short description of our present knowledge of acid secretion is given.
2. Experiments embodying double test meals and dilution estimations are described.
3. Results are discussed in the light of acid secretion theories.
4. Hydrolytic theory is explained.
5. The theory is applied to certain clinical states.

REFERENCES.

1. Apperly F. L.—" Medical Journal of Australia," June 1925.
2. Baylis, W. M.—" Principles of General Physiology."
3. Garlson, A. J.—" The Secretion of Gastric Juice in Health and Disease," Phys. Reviews 1923, Vol. III, p. 1.
4. MacLean & Griffith's.—" Jl. of Physiology," LXV, p. 63, 1928.
5. Mathews.—" Physiological Chemistry."
6. Ride, L. T.—A Paper read before the Far Eastern Association of Tropical Medicine at Bangkok on December 10, 1930.
7. Ride, L. T. & Wong, S. Y.—" The Caduceus," Vol. 9, No. 4, p. 159, 1930.

SOME CLINICAL APPLICATIONS OF THE HYDROLYTIC THEORY OF HYDROCHLORIC ACID SECRETION.*

by

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THE HYDROLYTIC THEORY OF ACID SECRETION.

In a recent paper the writer, along with S. Y. Wong (8), has brought forward evidence in support of the theory that hydrochloric acid is secreted by the oxyntic cells of the gastric mucosa as the chloride of a week base. This chloride, on reaching the lumen of the gut, is partially hydrolysed into base and acid; the cells of the gastric mucosa, being permeable to the basic ions and not to the chlorine ions, bring about a separation, the base being absorbed leaving the acid free in the stomach. As an example of such a substance, ammonium chloride was quoted for the following reasons. This salt is readily hydrolysed in solution; the mucous membrane is very permeable to the ammonium ion; the mucosa of the stomach has the highest ammonia content of any of the body tissues; in the stomach itself the ammonia content varies, that of the pyloric mucous membrane being greater than that of the cardia. Added to these reasons there is the fact that the effect of the addition of ammonium chloride to a test meal in a normal person has been shown to result in an increased formation of hydrochloric acid (8.).

The experimental method of arriving at this theory of acid secretion was as follows. Arabinose solution of know strength was added to a known voume of the test meal and each subsequent sample was analysed quantitatively for the pentose, using some of the original meal as the standard. By this means the dilution of the food in the stomach could be followed right through the whole of gastric digestion, or rather until the stomach contents became too dilute to detect the pentose accurately. On many occasions it was found that the dilution may remain the same for two successive samples, but that the acid curve over the same period may show a marked rise.

This could only be explained in two ways, either the acid was secreted in a very concentrated form or that it was secreted by some method whereby a volume of fluid was absorbed which was the same as the volume of fluid secreted. The first exlpanation was discarded owing to the fact that the arabinose method of measuring the dilution was accurate enough to detect the change in dilution caused by the addition of even very concentrated acid. We were therefore led to

* Some of the matter included in this article formed the basis of a paper read before the 8th Congress of the Far Eastern Association of Tropical Medicine, at Bangkok on December 10th, 1930.

adopt the hydrolytic theory given above, an example of such a reaction being represented by the following equation:

$$NH_4Cl + HOH = NH_4OH + HCl$$

From this equation it will be seen that the absorption of the ammonia on the right hand side of the reaction will result in the formation of acid, and if the molecular concentration of the absorbed ammonia be the same as that of the secreted chloride the volume of the fluid present before the ammonium chloride entered the system will remain unaltered. Thus the dilution of the stomach contents may remain the same, even though there be a marked rise in acid concentration.

This theory is by no means a new one. Claude Bernard was the first to deduce by animal experiments that the mucosa itself was alkaline in reaction and that it was only *after* secretion that the acid was formed. Foster was the first to suggest the hydrolytic theory and since then although much work has been done, the exact method of hydrochloric acid secretion has remained a matter of conjecture.

The situation is reviewed more fully in the writer's article mentioned above, and it remains here but to state that this additional evidence seems to weight the scales in favour of the hydrolytic theory. It is my desire in this paper to try and substantiate the claims of this theory still further, by showing how certain clinical states may be better explained by it than by the other theories of acid secretion.

Hyperchlorhydria and Hypochlorhydria.

It is a very common observation that in a series of fractional test meals on normal subjects, the free acid curves obtained vary within very large limits. So great is this variation that it is impossible to arrive at one average curve that would be of some clinical use, for some normal cases would diverge so greatly from it as to make it useless for purposes of comparison.

In 100 normal cases Bennett & Ryle (6) found such wide variations that, in making their chart, they used the limits of eighty per cent. of their cases as representing the area within which a curve should fall to be considered normal.

Apperly (4), has found in a similar examination of Australian students that the acid curves are even higher than those found in English students, and as far as I have proceeded with my group of Southern Chinese I find the acid values even higher still.

What is the explanation of these findings? Why of all the physiological constants (or averages) should this one be so very inconstant not only between different races in different parts of the world, but

also between members of the same race? Under the old acid secretion theory it would be explained by either a higher strength of acid secreted or lessened neutralization. If we believe in MacLean's theory (7) of the cause of the fall of the acidity, we should have to assume that in these cases the stomach had failed to substitute its chloride secretion for that of the acid.

According to the hydrolytic theory the explanation lies in the fact that the variable factor is the permeability of the gastric mucosa to the ammonium ion, the more permeable it is the more will the ammonia be absorbed and the more acid will be formed. Ryle (9) has stated that "neither hyperchlorhydria nor achlorhydria can in themselves be regarded as pathological findings," but if these states are due to deficient, faulty and absent secretion, it is hard to reconcile them with this statement of Ryle's. If however we accept the hydrolytic theory these states become merely the reflection of the varying permeability of the gastric mucous membrane cells to ammonia, and then we can accept Ryle's clinical observation knowing how many and various are the factors which can alter the amount of a salt passing through a membrane. We know that the absorptive power of the renal tubule cells varies, and there is no reason why we should be surprised if the absorption of the ammonia in the stomach should also vary. Under the same reasoning hypochlorhydria depends on the fact that the cells are less permeable to the ammonia, and therefore less free acid is left in the stomach.

If this theory be true, one should expect the effect of the addition of ammonium chloride to the meal given to an hyperchlorhydric, to be greater than the effect in the case of an hypochlorhydric. During my investigations on the effect of ammonium chloride added to test meals I have obtained quite a number of examples proving this point, and here one might mention the variation in the effect noticed clinically after the administration of ammonium chloride to patients. Some can stand it well while others complain that it gives them "indigestion," generally of the acid dyspeptic type. The hyperchlorhydrics comprise this latter class, for even a small amount of ammonium chloride in their medicine increases the acid formation, causing the symptoms of dyspepsia of which they complain; the same amount of ammonium chloride given to hypochlorhydrics has a very small effect on the acid secretion and hence does not give rise to acid dyspepsia.

Peptic Ulcers.

Whatever theory one wishes to accept for the causation of peptic ulcers, one is bound to admit that the acid of the gastric juice must play a more or less important role in their causation and persistance. If once the mechanism whereby the mucous membrane protects itself against the action of the acid is interfered with, the result is a condition

favourable to the formation of an ulcer. Under the hydrolytic theory, the ammonia content of the epithelial cells becomes of prime importance in this protective mechanism, for should the acid gain access through the cell membrane it will be immediately neutralized by the ammonia and the harmless ammonium chloride reformed in the cells. The moment however that any substance arrives on the scene which can interfere with this protective mechanism, the HCl would be free to attack the stomach lining. Any substance that could bind the ammonia would bring about this state of affairs and one such substance is CO_2 gas.

In certain conditions, especially when there is a high acid content, free carbon dioxide gas may be evolved during the neutralization of the duodenal carbonate by the gastric acid. This evolution of carbon dioxide gas may take place in the *pyloric portion of the stomach or in the first part of the duodenum*, according as to whether the acid passes on to the duodenum or whether the duodenal contents regurgitate into the stomach. In both cases CO_2 gas can only be given off if there is an excess of acid.

This gas, as soon as it comes into contact with the gastric mucosa, is absorbed readily, for the cells are very permeable to CO_2. Immediately on entry it meets and combines with the ammonia which is there in plenty during gastric secretion, and the acid resisting powers of that part of the stomach lining are thereby lessened. One would thus expect to find the *site of peptic ulcers limited to the areas where CO_2 gas may be produced* and where high strengths of acid may have access. As will be seen from the above reasoning, such places are to be found in the fundus of the stomach, in the region of the pylorus, and also around a gastro-jejunal stoma. The high acidity does not of itself cause the ulcer, but causes the formation of a gas which in turn interferes with the normal protective mechanism of the mucosa, thus exposing these cells to the attack of the high acid. It may be mentioned here that in the gas bubble seen in the cardia of the stomach in most cases of X-ray, there may be a large percentage of CO_2. But we must not expect to find any ulcer occuring in that region because there is no high concentration of free acid. This is additional evidence in favour of the theory that both CO_2 gas and strong acid are necessary for the production of the ulcer.

The case of the gastro-jejunal ulcer is of additional interest because it has long been known that the cause of such ulcers does not lie alone in the fact that there is a high acidity, nor does it depend only on the tissues at that site, for it is a common observation that immediately the stoma is closed or the proper continuity of the intestinal tract is restored, the ulcer disappears of its own accord. In such a case the tissues at the site are still the same, there is still as much acid bathing

the cells, but what has changed is that there is no longer any gaseous CO_2 reaching the cells to fix the protecting ammonia. Here we also have the explanation of the fact that trauma of the mucous membrane does not necessarily result in a persistent ulcer; if there be no excess of CO_2 gas the protective mechanism is still intact and no persistent ulcer will result. If there be an excess of CO_2 gas present, there will be a likelihood of the traumatic ulcer remaining unhelaed and becoming a true peptic ulcer.

Achlorhydria.

Achlorhydria may be either false or true. The most common cause of false achlorhydria is the presence of mucus. It is very common to find an apparent achlorhydric produce a practically normal acid curve after gastric lavage (Ryle) (11). The function of mucus may be taken as twofold, (*a*) to assist the passage of food along the intestinal tract and (*b*) to lessen the irritant action of some foreign substance introduced into the stomach. The former is its action under normal circumstances, the latter under abnormal circumstances. It is accompanied by a false achlorhydria, or an hypochlorhydria. If we agree that its secretion in such cases is primarily to protect the mucous membrane from irritation, it will do so not only by diluting the stomach contents but also by preventing such contents from coming into contact with the surface of the cells. Such an action will automatically prevent the absorption of the ammonia and therefore result in a low acidity.

The true achlorhydrias of Addison's anæmia and primary achlorhydria may be considered together. So constantly is achlorhydria found in Addison's anæmia that it is now generally agreed that this disease should not be diagnosed in its absence. Yet it is generally admitted that unless the case be one of very long standing the oxyntic cells are always to be found present and more or less normal in appearance. If the function of these cells is to secrete hydrochloric acid, then in such a case these cells must have ceased functioning years before death, and one can hardly understand why they should not have atrophied long since. Under the hydrolytic theory, the persistance of these cells is no longer a mystery, for they continue to secrete their chloride, but either the chloride is not secreted as an hydrolysable salt or else the mucous membrane has lost its power of absorbing the ammonium portion. In either case the result will be an absence of hydrochloric acid together with the persistance of the oxyntic cells. It must be emphasised here that this is not an attempt to explain the achlorhydria of Addisson's anæmia; it does not do so; it merely shifts the explanation one step further back, but it does offer a solution to a hitherto unexplained finding, nafely *the persistance of the oxyntic cells after years of absence of the secretion of HCl.*

Nor yet has a satisfactory explanation been forthcoming for the achlorhydria of carcinoma ventriculi. The explanation given by Ryle (10) is that it is due to " neutralization by the alkaline and albuminous exudates from the surface of a malignant growth." I do not know on what experimental evidence this statement is founded, but it has always appeared to me to be an inadequate explanation. Is there any reason to suppose that the product of carcinomatous cells should be more alkaline than normal cells? Rather one might reasonable expect that the product of such cells might be less alkaline than normal, so much greater is the cellular activity there. If the exudate from a single ulcer fails to neutralise the gastric contents, why should that of carcinomatous cells be expected to do so? In leather bottle stomach there may be no ulcerating surface at all, and therefore no exudate, and yet the finding of achlorhydria is just as constant there. The explanation may much more reasonably be expected to lie in the fact that the growth affects the surface tension of the mucosa cells with the result that such surface tension phenomena as absorption of ammonia are greatly interfered with. A much less common, but none the less interesting, cause of achlorhydria has been the subject of comment by David Smith in a recent article on " Organic Gastric Syphilis " (12). In dealing with his series of six cases of gummatous tumour formation in the stomach he states " Achlorhydria was demonstrated in five out of the six cases. *The lesion in the case which showed free hydrochloric acid was a gummatous mass causing pyloric obstruction.* This is in agreement with the findings of Downes and Le Wald. *In this case there was almost complete retention of the gastric contents, and it was in marked contrast to the other cases, where the gastric contents left the stomach rapidly.* There was no excess of mucus in any of the cases. Blood and lactic acid were absent; the analysis depends to a great extent on the situation and type of the lesion, and may be influenced by the general debility and anæmia due to the syphilitic infection, for syphilitic anæmia generally shows an achlorhydria. Some authors contend that the syphilitic virus in addition to the localized lesion may produce a general gastritis which is more responsible for the achlorhydria than is the lesion itself. This is rather negatived in the cases here recorded by the fact that no excess mucus was demonstrated. Crohn *thinks it is unlikely that any particular form of gastritis exists in these cases to a degree sufficient to cause an atrophy of the mucous membrane and a resultant anacidity or achylia gastrica.* Section of the mucosa away from the actual gummatous area fails to show any unusual form of gastritis. *The cells and membrana propria are not materially altered.*" (The italics are mine). If the achlorhydria is caused by the syphilitic condition of the stomach mucosa per se, why should it not be a constant finding? The key to the problem

lies in the fact that " The lesion in the case which showed free hydrochloric acid was a gummatous mass *causing pyloric obstruction*. In this case there was almost complete retention of the gastric contents, and it was in marked contrast to the other cases, where the gastric contents left the stomach rapidly." If gummata of the stomach produce achlorhydria by affecting the absorption of the basic ion from the hydrolysed chloride, as long as there is no pyloric obstruction, absorption being greatly reduced, no acid will be formed, but as soon as there is obstruction, the stomach contents will be held back for such a length of time that a small though definite amount of absorption can take place with the resulting liberation of free acid. If this case had been examined earlier before obstruction supervened, it too would have exhibited achlorhydria, and it is only after the tumour begins to cause obstruction that free acid again appears in the gastric contents.

Smith adds " The cells and membrana propria are not materially altered." It would be interesting in the light of what has already been said with regard to the persistance of oxyntic cells in Addison's anæmia patients, to ascertain whether the persistance of these cells is also inferred by the above quoted sentence. In such cases as these it would be of great interest to measure not only the acids but the total chloride as well.

As a result of the consideration of all these clinical states, it will be seen that the important factor in regulating gastric acidity (and therefore an important factor in the diseases of the stomach) is the permeability of the gastric mucous membrane to ammonia, and the factors which regulate its absorption. What these factors are in detail one cannot as yet say; but in general the basic ion after absorption must accumulate in the gastric mucous membrane cells, and as it increases in amount there, it must automatically tend to diminish further absorption. It is this banking up in the tissues as well as the mass action of the free acid itself which the author believes to be responsible for the fall in the acid curves towards the end of a meal (8). The base however cannot stay in these cells permanently and must be transported by the blood, during the period following digestion, to the liver. It is obvious therefore that if the power of the blood to deal with the base is in any way interfered with there will be a resulting fall in gastric acidity. In this connection it is interesting to see that Frank Artz (5) has recently found that free hydrohcloric acid in pregnant women was diminished as compared with non-pregnant women. *In spite of the achlorhydria, the total amount of chlorides was normal.* He explains this finding by supposing regurgitation from the duodenum to be the cause. It is much more likely that in the early stages of pregnancy the extra work of carrying excretory products caused by the presence of a rapidly growing embryo, affects the chemistry of the blood in

such a way that its power of transporting the gastric base is diminished. Later on in pregnancy one would expect the compensating powers of the body to rectify this state and gradually therefore acidity should return to normal. This is what Artz actually found for he states that the low acid values were more pronounced in the early months.

It is hoped that this question of the relation of gastric acidity to the blood will be dealt with in more detail in a further paper in the near future. But evidence of its importance is not lacking when one reads the papers of Apperly on " The Possible Influence of Climate on the Incidence of Peptic Ulcer in Australia " (1), and Apperly & Semmens on " A Nomogram Showing The Relation of Gastric Activity and Acidity to the Blood " (3) and " The Relation of Gastric Function to the Chemical Composition of the Blood " (2). In the latter article the authors state " Although we are convinced that blood chemistry and gastric function run parallel, there still remains the problem of showing that the former controls the latter." I suggest that the hydrolytic theory of acid secretion not only shows that the blood chemistry does control gastric function, but also reveals the mechanism by which this control is attained.

It must be admitted that a great deal of this is pure theory, but nevertheless it must also be admitted that this would not be the first theory in the realm of science which depended greatly for its proof on the fact that it supplied rational explanations for definite scientific findings otherwise unexplained.

SUMMARY.

1. A short description of the Hydrolytic Theory of hydrochloric acid secretion is given.
2. The occurrence of hyperchlorhydria and hypochlorhydria is explained on the grounds of this theory.
3. The dyspeptic effect of ammonium chloride on some patients is explained.
4. Evidence in favour of a theory of the formation of peptic ulcers depending on carbon dioxide gas formation and the absorption of ammonia is put forward.
5. Achlorhydrias both true and false are considered in the light of this theory, and an explanation for the persistance of the oxyntic cells in the gastric mucosa of patients suffering from Addisson's anæmia is given.
6. The achlorhydrias of carcinoma ventriculi, of gumma of the stomach and of hypochlorhydrias of early pregnancy are considered.

7. The importance of the factors governing the absorption of ammonia in the stomach is pointed out, special attention being drawn to the parrallelism between blood chemistry and gastric acidity.

REFERENCES.

(1) Apperly, F. L.—" Medical Journal of Australia," June 14th, 1930.

(2) Apperly, F. L. & Semmens, K.—" Medical Journal of Australia," September 10th, 1927.

(3) Apperly, F. L. & Semmens, K.—" Medical Journal of Australia," August 10th, 1929.

(4) Apperly, F. L. & Semmens, K.—" Medical Journal of Australia," August 25th, 1928.

(5) Artz, Frank.—" American Journal of Obstetrics & Gynæcology," September, 1930; (Abstract in Medical Journal of Australia February 28th, 1931, p. 267).

(6) Bennett, T. I. & Ryle, J. A.—Guy's Hospital Reports LXXI, 286, 1921.

(7) MacLean, H. & Griffiths, W. J.—" Journal of Physiology," LXV, 63, 1928.

(8) Ride, L. T. & Wong, S. Y.—This Journal, 1931 Vol. 10, p. 65.

(9) Ryle, J. A.—" Gastric Function in Health & Disease," Oxford Medical Publications, 1926, p. 35.

(10) Ryle, J. A.—Ibid p. 87.

(11) Ryle, J. A.—Ibid p. 111.

(12) Smith, David.—" British Medical Journal," November 8th, 1930, p. 773.

Editorial

FAR EASTERN ASSOCIATION OF TROPICAL MEDICINE.

The Eighth Congress of the Far Eastern Association of Tropical Medicine was held in Bangkok, Siam, from the 7th to the 20th December, 1930, under the patronage of His Royal Highness, Prince Paribatra of Nagor Svarga, Minister of the Interior. The Congress was attended by 160 members and 290 Associate Members, China, Japan, Hong Kong, United States of America, Philippine Is., Borneo, Java, F.M.S., India and Burma being amongst the eastern countries sending representatives. The Delegates from Hong Kong were Dr. W. B. A. Moore, Dr. A. V. Greaves, representing the Government, and Prof. W. I. Gerrard and Prof. L. T. Ride representing the University. Unfortunately Prof. Gerrard ultimately found it impossible to make the trip. Amongst the other delegates were three of our own graduates, two from Siam itself and one from Penang, while one of the delegates from Borneo was an old member of the Hong Kong College of Medicine and a member of our own medical faculty in its earliest days.

The Scientific Sessions were all held during the first week, and in addition to each afternoon was allotted either an At Home or an excursion, and each evening was taken up by either a reception or a command performance of native dancing or plays, in fact so lavish and overwhelming was the hospitality of our Siamese hosts that many chose the quiet of the return journey home in preference to visiting various places of medical interest in Siam, to which trips were arranged for the second week of the Congress.

As may be expected when one realises that most of the leprosy, malaria and plague experts of the Far East were gathered there, the papers and the discussions on these subjects were of a high order and of exceptional interest. It was on this account that the amount of time allotted for discussion after each paper often seemed disappointingly small, and without belittling the papers in any way, one must admit that the greatest advantage of a good paper is obtained when it is followed by relevant discussion in which competent listeners have time to develop their sides of the questions under consideration. This of course is the ever present problem confronting people responsible for such conferences. One cannot help feeling that although less people may be satisfied, more good would ultimately accrue from future conferences if more time were devoted to the scientific sessions or if a large number of the papers were taken as read and only the more important discussed.

A paper on the Yellow Fever problem in the Far East by Professor E. P. Snijders, Professor of Tropical Medicine, University of Amsterdam, justly attracted great attention to this problem which has assumed such importance of late years. Dr. Snijders made the startling and disquieting statement that certain species of monkeys and mosquitoes in

the Dutch East Indies were just as susceptible to the virus of yellow fever as those in the endemic areas. In cleverly developing his theme, he made only too apparent the awful results which might follow the importation of the virus to the Far East, even though this importation be under the strict supervision of experimental laboritories. In view of this the Congress at its General Meeting held on Saturday, 13th December unanimously passed the following resolution, proposed by Major-General Graham and seconded by Dr. van Lonkhuiyzen,

> " that in view of the increasing dangers of introduction of the disease (Yellow Fever) into suitable areas of the Far East, stringent measures must be adopted against its importation either through air traffic from infected or suspected areas or through introduction of the virus for experimental or other purposes "
>
> and
>
> " that such measures may mean the interdiction of air traffic from certain areas until adequate protective measures are framed by the Office International d'Hygiene Publique, and are in operation, and prohibition by law under severe penalty of the importation or possession of the Yellow Fever Virus."

Dr. L. Anigstein, Professor of Parisitology in the University of Warsaw, who is now working in the Institute of Medical Research, Kualà Lumpur, gave a paper embodying the results of his studies on Tropical Typhus in Malaya, while Professor C. D. de Langen, Professor of Internal Medicine, Batavia, diverted attention to other directions by a stimulating paper on ' Le Champ Electrique de l'Atmosphere et le Proces d'Acclimatement dans les Pays Chauds.' Three papers were contributed from Hong Kong, one by Dr. Greaves, " Pellagra in Relation to Food Supply " and the others by Prof. Ride, one on Blood Grouping Technique and the other on Fractional Test Meals.

Great as the advantages of such conferences are from the purely scientific point of view, it may be justly claimed that their greatest benefit lies in the fact that workers on similar problems are given opportunities of coming into personal contact with one another; full advantage of these opportunities was taken by the various University representatives present, and a number of meetings was held which should result in closer co-operation on the various research problems being attacked in these different institutions as well as the adoption of a more unified system of medical education in the Far East.

Of the many other lessons to be learnt from such a meeting, we shall only mention one other and that is the very wise system followed by the Medical Schools in Holland of sending out to their colonies, for a short period, some of their most capable professors so that they may study tropical problems in their natural settings. This practice must be of inestimable value to medical science both in the homeland and in the colony, and such an interchange between a medical school in England and ours here would, if properly managed, result in nothing

but the greatest mutual benefit. Our medical school has many needs it cannot be doubted, but it is a moot point whether the satisfaction of any of these needs would result in greater benefit than that bestowed by the adoption of the plan mentioned above. It has often been held up as a reproach that we in Hong Kong have no memorial to Manson. What more fitting memorial could be instituted than a Manson Memorial Professorship, which would be held by an eminent professor from home once every four and a half years while our own professor of medicine was away on leave.

Such a scheme woud need large funds, but surely not beyond the realms of possibilities when one considers the immense number of Manson's admirers both here and at home who would be only too pleased to contribute. One can hear it whispered "What a shock the Memorial Professor would get when he arrived." Agreed. But eminent men are generally capable of returning shocks with interest where they are most deserved, and perhaps even we might be judged as 'deserving.'

THE LATE PROFESSOR C. Y. WANG.

One of the penalties of age is that it brings us into ever closer touch with the realities of life, and of death. Our University is fast coming of age, but it must be viewed as untimely that it should be so soon bereft of the services and councils of its first Professor of Pathology in the prime of his life and usefulness. Professor Wang passed away on the 14th December, 1930; we print elsewhere in this number an appreciation of his life and his work, and here we should just like to place on official record our profound regret at the passing of so distinguished a member of our society, and to offer our sincerest sympathies to his wife and family in their irreperable loss. In so doing we cannot do better than quote the words spoken by the Vice-Chancellor at the grave-side—

> "WANG CHUNG YIK.—We give back your body to the earth, to the earth whence we all came and to which we are all returning. You gave up your life to the science and art of Medicine in one of its most vital aspects. You saved others, yourself you could not save. In the plenitude of your life and vigour a deadly disease got hold of you and in spite of the serenity of your courage and patience and of your grim struggle to live for the sake of those you loved, you passed yesterday, in the hush of the hours that prelude the dawn, from pain to peace. The laws of Nature are inexorable, and perhaps you who always accounted it your highest privilege to be Nature's servant and interpreter would not have it otherwise.
>
> You have left behind you the abiding memory of a blameless life of unostentations devotion. This is your gift to your family, your University and your Country. It is no mean one—Fare you well."

Review of Books

" Fellowship Examination Papers for the Diplomas of the Royal College of Surgeons: Edinburgh 1927—1930. (E. & S. Livingston, 16-17 Teviot Place, Edinburgh). 2/6 nett.

This small book of 42 pages not only gives all the papers set during the 4 years, 1927 to 1930 inclusive, for the Single License Examination and the Fellowship Examination, but also those set in Optional Subjects which include Anatomy, Dental Surgery & Pathology, Gynæcology, Laryngology, Otology, Rhinology, Obstetric Surgery, Ophthalmology, Surgical Pathology and Operative Surgery.

For those contemplating post graduate work in Europe, this book gives an accurate idea of the standard and scope of these examinations, as well as according the ordinary student excellent samples of test papers for his own use.

" Catechism Series ": Chemistry (Inorganic & Organic) Part II, Pathology Part V, Medicine Part II. (E. & S. Livingstone, 16-17 Teviot Place, Edinburgh. Price 1/6 per Part.

The Catechism Series is too well known to need description. It is not designed to replace text books, but as an adjunct to a student's own summary, these books are a very valuable help in revision, ensuring that no important part of the subject is left untouched.

Chemistry Part II deals with many of the heavy metals, such as Zinc, Tin, Copper, Silver, Iron, Lead, etc. and with Organic Chemistry, the latter being done very well.

Pathology Part V contains 79 pages and includes diseases of lungs, alimentary canal, liver, kidney, nervous system and joints. While Medicine Part II gives concise answers to questions on signs, symptoms, treatment, etc. of disorders of nutrition, kidneys, blood and ductless glands.

" Diseases of Women ": (4th Edition) by Ten Teachers. Edited by Comyns Berkely, H. Russel Andrews, J. S. Fairbairn.

This, " the fourth edition " has undergone general revision and the subject matter has been re-arranged with distinct advantage.

The chapter on Anatomy and Physiology of the female reproductive organs has been brought up to date and some new figures have been added. The steps in the operative treatment of Prolapse are briefly described, but the description is not quite clear, we think that this subject is deserving of more attention than it has received.

A two stage operation is inadvisable and a vaginal Ventro Suspension would meet all cases done at one sitting.

The failure to mention Diathermy in the treatment of subacute and chronic Salpingitis of Gonococcal origin should be rectified.

The material contained in the volume comprises some of the most important in the whole of Gynæcology, and real advances have been made from the old edition.

The book merits the greatest respect.

D. K. P.

"*Modern Infant Feeling*": by Bernard Meyers, C.M.G., M.D. (Edin.), M.R.C.P.

The book under review is one of the series of modern treatment and the author from his wide experience has kept well abreast of the modern advances in infant feeding.

Infant feeding has always been difficult to the student of Medicine and this book is recommended to all students and practitioners.

The opening chapter gives an interesting account of anatomy and physiology of the intestinal tract.

The capacity of the child's stomach at birth is gives as 7 grms.: in the 2nd week the capacity is about 90 grms. and thereafter till the 1st year the physiological capacity increases from 20 to 25 c.c. per month.

The value of breast feeding is emphasised; it is nature's food for the new-born and should be given by all mothers: if not for 9 months, for varying shorter periods to ensure some benefit to the child.

The treatment of Constipation in children has always been a difficulty both to the doctor and mother.

The author suggests that the habit of having an action at regular interval should be inculcated from early infancy.

Suppositories and oiled catheters should not be necessary if proper attention is made to feeding and proper habit.

There is an appendix in which is briefly described, blood transfusion in children giving the indications, compatibility, and technique.

The author is to be congratulated in producing a useful and clearly written book in a small space.

"*Enzymes*": By J. B. S. Haldane. (Messrs. Longman Green & Co., Ltd., 39, Paternoster Row, London E.C.4. Price 14/- Net.)

The "Monographs on Biochemistry" has added yet another good book to its already imposing series. The immense amount of work that the writing of this book entailed cannot be gauged by merely one reading. Each chapter reveals fresh evidence of the remarkable grasp the writer has on this involved and intricate part of physiology.

In a short review such as this it is impossible to discuss all the matter set out in the book, but for any physiological departmental library the monograph is indispensable. One is thankful to see the inclusion of the modern conception of carbohydrate structural formulæ, but one should have been more pleased had one found a few paragraphs on reliable methods of quantitative estimations of the enzymes more commonly used, especially such as pepsin, trysin, etc.

"*Medical Jurisprudence and Toxicology*": By J. Glaister. (Messrs. E. & S. Livingstone, Edinburgh. 5th Edition: 954 p. 152 illustrations 7 plates, 30/- net.)

It is very seldom said that a medical text book is as interesting as a novel, but of this book it can be truthfully stated that it is much more readable than a large percentage of modern novels. In this edition the author has had the assistance of John Glaister, Jr. who, as well as being Professor of Forensic Medicine at Cairo, is a Barrister-at-Law in the Inner Temple.

The book begins with a clear exposition of that much misunderstood body—The General Medical Council, and owing to the fact that the modern practitioner is coming more and more into contact with legal matters, detailed coensideration of the Workmen's Compensation Acts, National Health Insurance Acts and Dangerous Drugs Acts is given.

The chapter dealing with Medical Evidence is very important and the advice given to medical men who are under the strain of cross examination is very sound. Eight pages are devoted to Professional Secrecy and Privilege.

The explanation of Henry's Cabinet for Classifying Finger Prints on p. 92 is rather hard to follow, but that is of no great importance to the ordinary medical man. The examination of blood stains, hair, etc. is gone into with the minutest detail.

The authors raise some very interesting points in the section on Post-mortem Delivery. They state "Since the existence of the child ***in utero*** is held to depend upon the existence of the mother, the question may be pertinently put, Does the very fact of the live birth

of the child involve the fact of the existance of life in the mother?" Surely "life in the mother" and "life of the mother" are two very different things. For example, some time after death from a fatal accident grafts of the pancreas may be removed from a body and transplanted into a diabetic. No one in such a case would hesitate to say the donor was dead and yet if the grafts took, "life" must have still been present "in" the donor's body.

If one might make suggestions, and one does it with all humility in the light of such a good book, perhaps the Ascheim-Zondek Hormone Test for Pregnancy might have been mentioned as well as Abderhalden's, and also perhaps attention might have been drawn to the use, in Germany at any rate, of Blood Groups in the question of Parentage.

An estensive index and a legal index increase greatly the utility of the volume.

"*Affections of the Eye in General Practice*": By R. Lindsay Rea. (Messrs. H. K. Lewis & Co., Ltd., London. 10/6 net).

This book is too well known to our readers to need any detailed introduction to them, for extracts from it have been running through our magazine for the last year or so. In book form, however, one gets the advantage of seven coloured plates and thirty-three illustrations as well as the index and seven pages of therapeutic formulæ.

The book should form a great stand-by to general practitioners especially in this part of the world where eye disease is so common, and where each doctor has often to be his own consultant specialist.

"*Diseases of Children*": By Bruce Williamson. (E. & S. Livingstone, Edinburgh, 10/6 net).

This is a small book of 290 pages, and the author may be congratulated on having accomplished what he says in the preface he intended to do, namely," to provide a handbook which will not only see the student through his undergraduate course, but will later help him in the application of his knowledge in practice." It is doubtful, however, whether such a book as this, despite the inclusion of the more rare conditions, can be expected to cater for "the needs of the post graduate who seeks further academic honours in a membership in one of the colleges of physicians."

On first looking into the book one is favourably impressed by the manner in which it is set out, and that impression is retained after careful reading.

The chapter on the heart is well done; a clear and concise explanation of cardiac physiology fills a gap which is far too obvious in most books.

Perhaps the author might have mentioned loss of weight amongst the other signs or Congenital Pyloric Hypertrophy, and for the benefit of the above mentioned post graduates, it would have been better not to have mixed two schools of thought by using both terms "achalasia" and "spasm" when dealing with Pyloric Obstruction. Also it is doubtful whether a correct impression of the digestion of fats is given by saying that the "bile salts convert the fatty acids into soaps."

The chapters on Dietetics and Infant Feeding are good and the Formulary should be of great use to the student; in fact the book is what it claims to be, a good handbook for students.

"*An Introduction to Human Experimental Physiology*": By F. W. Lamb. (Messrs. Longmans Green & Co., Ltd., 39, Paternoster Row, London E.C.4. 12/6 net).

It is not an easy task to produce a book on Experimental Physiology (or theoretical too for that matter) which is suitable for a class of students some taking a science course and some a medical course. There is so much of the more mathematical and scientific side of this subject which is of merely passing interest to the clinician, that it is a waste of time stressing it in classes of medical students.

Although at times one feels that Dr. Lamb's book is a bit outside the realm of the medical student, it is a book which should be present in all University libraries. Its two most striking features are its completeness and the fact that it is up to date in every department e.g. Behring's Venule is fully described in the methods of obtaining blood for examination; Arneth's count is described in the section on blood cells, the various classifications of Blood Groups are given (but it would be our opinion a distinct advance to adopt the League of Nations Nomenclature in this connection). In fact when one tries to pick out the good points for mention one is unable to decide what not to mention; but for medical students with whom we are mostly concerned here, the best chapters are those on blood and circulation.

As the author claims in his preface, the references at the end of each section are of inestimable value to the scientific students.

Acknowledgments

Acta Psychiatrica Et Neurologica—Denmark.

Arquivos de Clinica Medica—Porto—Portugul.

Peiping Union Medical College Hospital—Annual Report.

Lingnan Science Journal—Canton.

Japanese Journal of Experimental Medicine.

The Moukden Medical College Journal.

Boletin de La Universidad Nacional de La Plata.

Revista Medico-Chirurgocala—Roumanie.

The Birmingham Medical Review.

King's College Hospital Gazette.

St. Bartholomew's Hospital Journal.

The Medical Times, London.

University College Medicine Gazette.

The Hospital.

Okayama-Igakkai-Zasshi.

New York Academy of Medicine Bulletin.

Journal of Bone and Joint Surgery.

Med. College of Virginia Bulletin.

Manchester University Medical School Gazette.

Queen's Medical Magazine.

University of Durham Medical College Gazette.

St. Mary's Hospital Gazette.

Journa of B.M.A. Ceylon.

Chiba Igakukwai Zasshi.

Mitteilungen Uber Allgemeine Pathologie Und Pathologische Anatomie.

Aeta Universitatis Asiae Media.

Porto Rico Journal of Public Heath & Trop Medicine.

Medical Journal of Australia.