ST. LUKE'S HOSPITAL

Medical and Surgical Reports

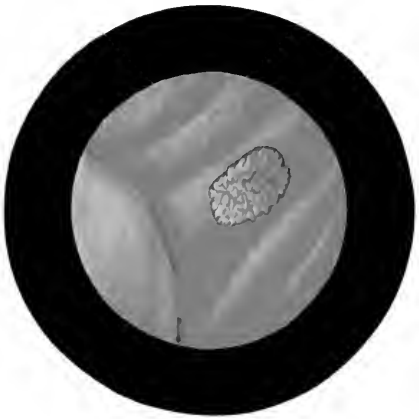
Volume III

1911

Columbia University
DEPARTMENT OF
PRACTICE OF MEDICINE

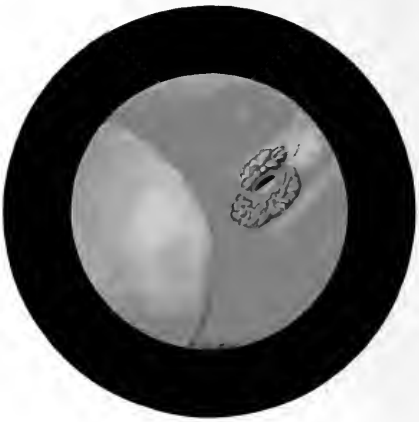
COLLEGE OF PHYSICIANS AND SURGEONS
437 WEST 59TH STREET, NEW YORK

I



Papilloma of Bladder Completely Covering the Left Ureteral Orifice

II



Same after One Application of the High Frequency Current

III

Same after Second Application of the High Frequency Current. (Close vision, prostate not seen in the field)

IV

Final Result, One Month after the first Application. (Close vision)

THE TREATMENT OF PAPILLOMA OF THE BLADDER WITH THE HIGH FREQUENCY CURRENT

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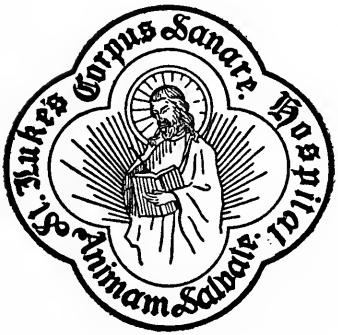

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Surgical Service

SURGICAL STATISTICS FOR 1911

ALIMENTARY SYSTEM INTESTINES	Operations	Cured	Improved	Unimproved	Died	Totals
Colitis	....	....	1	....	....	1
Colitis, mucous adhesions	....	....	1	....	1	2
Colitis (ulcerative), hemorrhages	1	....	....	1	....	1
Diverticulitis	....	1	....	....	....	1
Duodenal ulcer	3	3	....	....	....	3
Duodenal ulcer, peritonitis	3	....	....	....	3	3
Duodenal ulcer, volvulus	1	....	1	....	....	1
Enteritis, gastro-enteritis	....	1	....	....	....	1
Enteroptosis	1	....	1	2	....	3
Ileus	7	6	....	....	1	7
Ileus, band	4	1	2	....	1	4
Ileus, intestinal adhesions	1	1	....	....	....	1
Intestinal indigestion	1	1	1	....	....	2
Intussusception	5	3	....	....	2	5
Perisigmoiditis	1	1	....	....	....	1
Vicious circle	1	....	....	....	1	1
	29	18	7	3	9	37
HERNIA						
Hernia (femoral)	9	9	1	....	....	10
Hernia (femoral, incarcerated), ing. hernia	1	....	....	....	1	1
Hernia (femoral, strangulated))	1	2	....	....	....	2
Hernia (inguinal)	128	143	3	8	....	154
Hernia (inguinal sliding)	1	1	....	....	....	1
Hernia (inguinal strangulated)	4	3	....	....	1	4
Hernia (inguinal), œdema of lungs, broncho-pneu	1	....	....	....	1	1
Hernia (umbilical)	7	7	....	....	....	7
Hernia (ventral)	23	22	2	1	....	25
Hernia, (ventral strangulated)	3	1	....	....	2	3
Hernia (ventral), ileus, abscess of abdominal wall, general peritonitis	1	....	....	....	1	1
Hernia (umbilical), abscess of scrotum, croup	1	1	....	....	....	1
Hernia (inguinal), pleurisy with effusion	1	1	....	....	....	1
Hernia (inguinal), lobar pneumonia	1	1	....	....	....	1
Hernia (inguinal), phimosis, undescended testis	1	1	....	....	....	1
Hernia (omental)	1	1	....	....	....	1
	184	193	6	9	6	214
LIVER						
Abscess of liver	....	1	....	....	....	1
Abscess of liver, diffuse peritonitis	1	....	....	....	1	1
Abscess of liver, miliary tbc	2	....	....	....	2	2
Cirrhosis of liver, œdema of lungs	....	1	....	....	....	1
Cirrhosis of liver, endocarditis, ascites, œdema of lungs	....	....	....	....	1	1
Hepatitis (interstitial), displacem't of transverse colon	1	....	....	1	....	1
Jaundice (obstructive)	1	....	1	....	....	1
	5	2	1	1	4	8
BILE PASSAGES						
Cholecystitis	5	5	....	....	....	5
Cholecystitis (gangrenous), peritonitis	1	....	....	....	1	1
Cholecystitis, toxic insanity	....	....	....	1	....	1
Cholecystitis (suppurative)	1	1	....	....	....	1
Cholelithiasis	21	19	5	2	....	26
Cholelithiasis with adhesions	1	1	....	....	....	1
Cholelithiasis, cholangitis	1	....	1	....	....	1

ALIMENTARY SYSTEM—Continued	Op.	C.	Imp.	Un.	Died	Total
Bile Passages—Cont.						
Cholelithiasis, cholecystitis	10	7	1	....	2	10
Cholelithiasis, carcinoma of pancreas	1	....	....	....	1	1
Cholelithiasis, pleuro-pneumonia	2	2	....	....	....	2
Empyema of gall bladder	3	1	1	....	1	3
Hydrops, stone in common duct	1	1	....	....	....	1
	47	37	8	3	5	53
MOUTH, GUMS AND TEETH						
Alveolar abscess	3	3	1	....	....	4
Painful alveolar process	1	1	....	....	....	1
Suppurating root of tooth	1	1	....	....	....	1
	5	5	1	....	....	6
ŒSOPHAGUS						
Stricture of œsophagus	1	....	1	1	1	3
Stricture of œsophagus, gastric adhesions, gangrene of lung	1	....	....	....	1	1
	2	....	1	1	2	4
PANCREAS						
Pancreatitis (acute), cholecystitis	1	....	....	....	1	1
Pancreatitis (hemorrhagic), delirium tremens	....	....	....	....	1	1
	1	....	....	....	2	2
PERITONEUM						
Abscess of peritoneum	3	1	2	....	....	3
Adhesions	5	3	2	2	....	7
Peritonitis, cause unknown	2	....	....	....	2	2
	10	4	4	2	2	12
PHARYNX, TONSILS AND NASOPHARYNX						
Abscess (peritonsillar)	6	2	4	....	....	6
Adenoids	10	10	....	....	....	10
Hypertrophy of tonsils	25	24	....	1	....	25
Hyp. tonsils, facial paralysis	....	....	....	1	....	1
Hyp. tonsils, adenoids	97	97	....	....	....	97
Tonsillar hemorrhage	1	5	....	....	....	5
Tonsillitis (follicular)	....	2	....	....	....	2
	139	140	4	2	....	146
RECTUM						
Abscess (anal)	1	....	1	....	....	1
Atresia of anus	....	....	....	1	....	1
Fissure in ano	6	6	....	1	....	7
Fistula in ano	29	23	5	1	....	29
Fistula in ano, pul. tbc	2	1	1	....	....	2
Fistula (fecal)	7	7	....	....	....	7
Fistula (fecal), old appendicitis, peritonitis	1	....	....	....	1	1
Hemorrhoids	60	54	6	3	....	63
Hemorrhoids, with enlarged glands, neuritis	....	....	1	....	....	1
Ischio-rectal abscess	10	10	3	....	....	13
Ischio-rectal abscess, sub-ac. nephritis	1	1	....	....	....	1
Proctitis, ischio-rectal abscess	1	2	....	....	....	2
Prolapse of anus	2	1	1	1	....	3
Prolapse of rectum, erysipelas	1	....	1	....	....	1
Stricture of rectum	2	1	1	....	....	2
	123	106	20	7	1	134
STOMACH						
Gastritis (atrophic) cirrhosis of liver	....	....	1	....	....	1
Gastritis (chr.)	....	2	2	1	....	5
Gastritis (chr.), morphinism, neurasthenia	....	....	....	1	....	1
Gastritis (chr.), perforation	2	....	....	....	2	2
Indigestion	....	1	1	....	....	2
Ptosis, dilatation	1	1	1	....	....	2

ALIMENTARY SYSTEM—Continued	Op.	C.	Imp.	Un.	Died	Total
Stomach—Cont.						
Pyloric obstruction	3	2	1	....	....	3
Pyloric stenosis	2	1	1	....	....	2
Ulcer (gastric)	5	3	2	1	1	7
Ulcer (gastric), adhesive peritonitis	1	1	....	....	1	2
	14	11	9	3	4	27
VERMIFORM APPENDIX						
Appendicitis (acute)	65	61	3	....	1	65
Appendicitis (ac.), peritonitis	30	24	2	....	5	31
Appendicitis (ac.), peritoneal abscess	30	28	1	....	1	30
Appendicitis (ac.), peritoneal abs., pulmonary embolus	1	....	....	....	1	1
Appendicitis (ac.), mesenteric lymphadenitis	1	....	....	....	1	1
Appendicitis (ac. catarrhal)	1	1	....	....	....	1
Appendicitis (chronic catarrhal)	....	3	....	....	....	3
Appendicitis (chr.)	191	190	7	5	1	203
Appendicitis (chr.), mitral and aortic insuff	....	1	....	1	....	2
Appendicitis (chr.), peritoneal adhesions	3	3	....	....	....	3
Appendicitis (gangrenous)	7	7	....	....	....	7
Appendicitis (gangrenous), peritonitis	6	4	....	....	2	6
Appendicitis (perforative)	1	1	....	....	1	2
Appendicitis (sub-acute)	16	16	1	....	....	17
Appendicitis (sub-acute), gastritis	1	1	....	....	....	1
Appendicitis (sub-acute), ac. pneumonia	1	1	....	....	....	1
Appendicitis (sub-acute), thrombosed veins of thigh, pulmonary embolus	1	1	....	....	....	1
Appendicitis (relapsing), renal calculus	1	1	....	....	....	1
Appendicitis (relapsing), suppurative pneumonia	1	1	....	....	....	1
Atrophic appendicitis	1	1	....	....	....	1
Appendicular adhesions	1	1	....	....	....	1
	359	346	14	6	13	379
CARDIO-VASCULAR SYSTEM						
BLOOD						
Anemia	....	1	1	....	....	2
Anemia (pernicious.)	....	....	1	....	....	1
Hematuria	1	....	1	....	....	1
	1	1	3	....	....	4
ARTERIES						
Aneurysm of innominate	1	....	1	....	....	1
Aneurysm of thoracic aorta	....	....	....	....	1	1
Embolism of cerebral arteries	....	....	....	1	....	1
Endarteritis of foot	....	....	....	1	....	1
Thrombosis (cerebral)	1	1	1	....	....	2
Thrombosis (femoral)	....	1	....	....	....	1
Thrombosis (mesenteric)	2	....	1	....	1	2
	4	2	3	2	2	9
VEINS						
Phlebitis of arm	....	1	....	....	....	1
Phlebitis of femoral veins	....	....	1	....	....	1
Hemorrhage from rectal veins	....	....	1	....	....	1
Thrombo-phlebitis of thigh	1	1	....	....	....	1
Thrombo-phlebitis of leg	1	....	1	....	....	1
Varicose veins of leg	20	20	3	3	....	26
Varicose veins of leg, hemorrhages	....	1	....	....	....	1
	22	23	6	3	....	32
HEART						
Dilatation (acute)	....	....	....	....	1	1
Endocarditis (acute)	....	....	....	....	1	1
Endocarditis (septic)	1	....	....	....	1	1
	1	....	....	....	3	3

CARDIO-VASCULAR SYSTEM—Continued	Op.	C.	Imp.	Un.	Died	Total
LYMPH GLANDS						
Lymphadenitis (axillary)	5	6	....	1	....	7
Lymphadenitis (cervical)	16	10	6	....	....	16
Lymphadenitis (femoral)	1	1	....	....	....	1
Lymphadenitis (inguinal)	6	6	3	....	....	9
Lymphadenitis (suppurative, retroperitoneal)	1	1	....	....	....	1
Lymphangitis of arm	....	1	....	....	....	1
	29	25	9	1	....	35
CONNECTIVE TISSUE						
Abscess of arm	1	....	1	....	....	1
Abscess of chest wall	....	1	....	....	....	1
Abscess of abdominal wall	1	1	....	....	....	1
Abscess of breast	1	1	....	....	....	1
Abscess of neck	2	2	....	....	....	2
Abscess of popliteal space	1	1	....	....	....	1
Abscess of perineum	1	1	....	....	....	1
Abscess of thigh	8	7	2	....	....	9
Cellulitis of arm	8	6	3	....	....	9
Cellulitis of arm (gangrenous)	3	....	3	....	....	3
Cellulitis of broad ligament	1	1	....	....	....	1
Cellulitis of face	3	2	....	....	1	3
Cellulitis of foot	1	3	2	....	1	6
Cellulitis of hand	12	9	7	....	....	16
Cellulitis of leg	4	4	1	....	....	5
Cellulitis of parotid region	1	1	....	....	....	1
Cellulitis of penis	1	1	....	....	....	1
Cellulitis of scalp	2	2	....	....	....	2
Cellulitis of scalp, necrosis of maxilla	....	....	1	....	....	1
Bedsores	....	....	....	1	....	1
Hematoma of chest wall	1	1	....	....	....	1
Hematoma of scrotum	1	2	....	....	....	2
Hematoma of scrotum, hydrocele	....	....	....	1	....	1
Perineal inflammation following urethral stricture	1	....	1	....	....	1
Vesico-vaginal fistula	3	1	....	3	....	4
	56	47	21	5	2	75
DUCTLESS GLANDS						
Addison's disease	....	....	1	....	....	1
Goitre (simple)	8	6	3	....	....	9
Goitre (exophthalmic)	3	3	....	....	....	3
Hyperthyroidism	1	1	....	....	....	1
Parotid, cyst of	2	....	1	1	....	2
Parotiditis (acute)	1	1	....	....	....	1
	15	11	5	1	....	17
MUSCULAR SYSTEM						
Bursitis of popliteal space	1	1	....	....	....	1
Myosotis	....	1	....	....	....	....
Psoas abscess	1	....	1	....	....	1
	2	2	1	....	....	2
NERVOUS SYSTEM						
BRAIN						
Abscess of brain	2	....	....	....	2	2
Cyst of ventricle	1	....	....	....	1	1
Meningocele	....	....	....	1	....	1
Subdural hemorrhage	....	....	3	....	2	5
	3	....	3	1	5	9
DISEASES OF THE MIND						
Acute alcoholic mania	....	....	....	1	....	1
	....	....	....	1	....	1
NERVES						
Neuralgia (trifacial)	7	6	2	1	....	9

NERVOUS SYSTEM—Continued	Op.	C.	Imp.	Un.	Died	Total
Nerves—Cont.						
Neuralgia (trifacial), paralysis, eczema	1	....	1	....	....	1
Neuritis (brachial)	....	....	....	1	....	1
Neuritis (peripheral)	....	....	1	1	....	2
Neuritis (retro-bulbar)	....	....	2	....	....	2
	8	6	6	3	....	15
NERVOUS DISEASES OF UNKNOWN ORIGIN						
Hysteria	....	....	1	....	....	1
Nervous prostration	....	....	1	....	....	1
Neurasthenia (traumatic)	....	1	5	1	....	7
Spasmodic torticollis	....	....	....	1	....	1
Zoster, gangrenous hystericosus of forearm						
	....	1	8	2	....	11
SPINAL CORD						
Locomotor ataxia	....	....	1	....	....	1
Syringomyelia, paraplegia	....	....	....	1	....	1
Tabes dorsalis	....	....	1	....	....	1
	....	....	2	1	....	3
OSSEOUS SYSTEM						
Athetosis of arm	1	....	1	....	....	1
Coccygodynia	2	1	1	....	....	2
Hypertrophy of turbinate bone	1	1	....	....	....	1
Necrosis of vertebræ	....	....	1	....	....	1
Necrosis of maxilla	1	1	....	....	....	1
Necrosis of femur, Pott's Disease	1	1	....	....	....	1
Osteitis (rarefying)	....	....	....	....	1	1
Osteomyelitis of femur	5	1	3	1	1	6
Osteomyelitis of forehead	1	1	1	....	....	2
Osteomyelitis of humerus	....	....	1	....	....	1
Osteomyelitis of maxilla	2	....	2	....	....	2
Osteomyelitis of metacarpal bones of hand	2	....	2	....	....	2
Osteomyelitis of metatarsal bones of foot	3	1	2	....	....	3
Osteomyelitis of tibia	2	1	1	....	....	2
Periosteitis of phalanx of hand	3	....	3	....	....	3
Periosteitis of femur	....	1	....	....	....	1
Periosteitis of tibia	1	....	2	....	....	2
Periosteal abscess of maxilla	1	....	1	....	....	1
Rickets	....	1	....	....	....	1
Sequestrum of maxilla	1	....	1	....	....	1
	27	10	22	1	2	35
JOINTS						
Ankylosis of ankle	....	....	1	1	....	2
Ankylosis of hip and knee	1	1	....	....	....	1
Arthritis (chr.) of knee	1	1	1	....	....	2
Arthritis (suppurative) of ankle	1	1	....	....	....	1
Bunions	1	1	....	....	....	1
Hypertrophy of int. ligament of knee	1	1	....	....	....	1
Hypertrophy of synovial folds of knee	1	....	1	....	....	1
Osteo-arthritis of hip	1	....	1	....	....	1
Osteo-arthritis of knee and vertebræ	....	....	....	1	....	1
Synovitis of knee	1	1	2	....	....	3
	8	6	6	2	....	14
REPRODUCTIVE SYSTEM—FEMALE						
OVARY						
Atrophy of ovary	1	1	....	....	....	1
Abscess (tubo-ovarian.)	4	4	....	....	....	4
Cystic ovary	39	39	1	....	....	40
Cystic ovary (multilocular)	2	2	....	....	....	2
Cystic ovary, pregnancy	1	1	....	....	....	1
Oophoritis	9	9	1	....	....	10
Oophoritis (atrophic)	1	1	....	....	....	1

REPRODUCTIVE SYSTEM—FEMALE—Continued	Op.	C.	Imp.	Un.	Died	Total
Ovary—Cont.						
Parovarian cyst	2	2	....	....	....	2
Prolapse of ovary	1	1	....	....	....	1
	60	60	2	....	....	62
UTERINE TUBES						
Hematosalpinx	3	3	....	....	....	3
Hematosalpinx with twisted pedicle, pregnancy	1	1	....	....	....	1
Hydrosalpinx	3	3	....	....	....	3
Pyosalpinx	26	23	4	1	1	29
Pyosalpinx with abscess	2	1	....	....	1	2
Pyosalpinx with abscess, thrombosis of broad ligament and iliac veins	1	....	....	....	1	1
Salpingitis (acute)	4	6	3	....	....	9
Salpingitis (chronic)	46	45	11	5	1	62
Salpingitis (chr.) post-op. shock, peritonitis	1	....	....	....	1	1
Salpingitis (chr.), peritonitis	2	1	....	....	1	2
Salpingitis (perforative)	2	2	....	....	....	2
Salpingitis with pelvic abscess	1	1	....	....	....	1
Pyosalpinx, paralytic ileus, peritonitis	1	....	....	....	1	1
Salpingo-oophoritis	19	18	1	....	....	19
	112	104	19	6	7	136
UTERUS						
Abscess of broad ligament	1	1	....	....	....	1
Anteflexion	9	7	2	1	....	10
Cyst (intra-ligamentous)	4	4	....	....	....	4
Dysmenorrhea	4	4	....	....	....	4
Endocervicitis	1	1	....	....	....	1
Endometritis (chr.)	78	74	8	4	....	86
Endometritis (glandular)	1	1	....	....	....	1
Endometritis (hypertrophic)	1	1	....	....	....	1
Erosion of cervix	....	....	1	....	....	1
Menopause (artificial)	1	1	....	....	....	1
Metrorrhagia	1	2	....	....	....	2
Prolapse of uterus	21	18	2	1	1	22
Prolapse of uterus, rectocele, cystocele	1	1	....	....	....	1
Prolapse of uterus, laceration of cervix and perineum	2	2	....	....	....	2
Retroversion	53	53	....	....	....	53
Retroversion, pregnancy	1	1	....	....	....	1
Retroversion with adhesions	1	1	....	....	....	1
	180	172	13	6	1	192
PREGNANCY, ETC.						
Abortion (complete)	4	4	....	....	....	4
Abortion (incomplete)	21	19	2	1	....	22
Abortion (threatened)	2	4	1	....	....	5
Abortion (incomplete), pelvic abscess	1	1	....	....	....	1
Ectopic gestation	12	12	....	....	....	12
Ectopic gestation (ruptured)	8	8	....	....	....	8
Lithopedion	1	1	....	....	....	1
Pelvic abscess	12	14	....	1	1	16
Pelvic abscess, pyometra	1	....	....	....	1	1
Pelvic abscess, ileus	1	....	....	....	1	1
Pregnancy	....	1	2	2	....	5
Retained placenta	14	14	....	1	1	16
Toxemia of pregnancy	2	1	1	....	....	2
	79	79	6	5	4	94
VAGINA						
Atresia of vagina	1	1	....	....	....	1
Prolapse ant. vaginal wall	13	13	....	....	....	13
Prolapse post. vaginal wall	5	5	....	....	....	5
Vaginitis	1	1	....	....	....	1
	20	20	....	....	....	20
VULVA						
Abscess (vulvo-vaginal)	2	2	1	....	....	3

REPRODUCTIVE SYSTEM—FEMALE—Continued	Op.	C.	Imp.	Un.	Died	Total
Vulva—Cont.						
Abscess (Bartholin's gland)	1	1	....	....	....	1
Hooded clitoris	1	1	....	....	....	1
	3	4	1	....	....	5
REPRODUCTIVE SYSTEM—MALE						
MALE URETHRA						
Fistula of urethra	1	1	....	....	....	1
Stricture of urethra	10	9	3	1	1	14
Stricture of urethra, calculus, extravasation of urine	1	1	....	....	....	1
	12	11	3	1	1	16
PENIS						
Balanitis	1	2	....	....	....	2
Phimosis	15	15	....	....	....	15
Redundancy of prepuce	8	8	....	....	....	8
	24	25	....	....	....	25
PROSTATE						
Abscess of prostate	4	2	1	....	1	4
Congestion of prostate	....	....	1	....	....	1
Hypertrophy of prostate	8	4	2	....	4	10
Hypertrophy, orchitis, cystitis	1	1	....	....	....	1
	13	7	4	....	5	16
SPERMATIC CORD						
Hydrocele	9	9	....	....	....	9
Varicocele	20	20	1	2	....	23
	29	29	1	2	....	32
TESTICLE						
Epididymitis	1	1	....	....	....	1
	1	1	....	....	....	1
MAMMARY GLAND						
Abscess of breast	3	3	....	....	....	3
Cystic degeneration	1	1	....	....	....	1
Cystic mastitis	4	2	2	....	....	4
Mastitis (suppurative)	1	....	1	1	....	2
	9	6	3	1	....	10
RESPIRATORY SYSTEM						
LARYNX						
Stricture of larynx	....	....	....	1	....	1
	....	....	....	1	....	1
LUNGS						
Abscess of lung	....	....	1	....	....	1
	....	....	1	....	....	1
PLEURÆ						
Pleurisy (chr.)	....	....	....	1	....	1
Pleurisy with effusion	....	....	1	....	....	1
Pleurisy (suppurative)	13	10	5	....	1	16
Pleurisy (supp.), catarrhal croup, ac. bronchitis	1	....	....	....	1	1
	14	10	6	1	2	19
NASAL CAVITY						
Atrophic rhinitis	....	....	1	....	....	1
Atrophic rhinitis, adenoids	....	....	1	....	....	1
Deviation of nasal septum	11	11	1	1	....	13

RESPIRATORY SYSTEM—Continued	Op.	C.	Imp.	Un.	Died	Total
Nasal Cavity—Cont.						
Epistaxis	....	1	....	....	....	1
Frontal sinusitis	....	....	2	....	....	2
	11	12	5	1	....	18
SENSE ORGANS						
ORGAN OF HEARING						
Mastoiditis	10	8	1	1	....	10
Mastoiditis, meningitis, otitis media	1	....	....	....	1	1
Mastoiditis, thrombosis of lateral sinus	1	1	....	....	....	1
Mastoiditis (suppurative), catarrhal jaundice, lobar pneumonia, septic arthritis of elbow	1	....	1	....	....	1
Otitis media	5	6	1	1	....	8
Otitis media (purulent)	....	....	1	....	....	1
	18	15	4	2	1	22
ORGAN OF VISION						
Cataract	9	5	6	....	....	11
Chalazion	....	1	....	....	....	1
Conjunctivitis	....	....	1	....	....	1
Exotropion	....	....	1	....	....	1
Glaucoma	5	1	4	....	....	5
Iritis, dacryocystitis	1	....	2	....	....	2
Ophthalmitis	1	1	....	....	....	1
Panophthalmitis	1	....	1	....	....	1
Rupture of cornea	1	1	....	....	....	1
Strabismus	1	1	....	....	....	1
Traumatic conjunctival hemorrhage	....	....	1	....	....	1
	19	10	16	....	....	26
TEGUMENTARY SYSTEM						
Carbuncle of lip	1	....	....	....	1	1
Carbuncle of neck	4	4	....	....	....	4
Cicatrix (painful)	2	1	1	....	....	2
Furunculosis	2	1	1	....	....	2
Ingrowing toe-nail	1	....	1	....	....	1
Pilonidal cyst	2	1	1	....	....	2
Purpura hemorrhagica	....	....	1	....	....	1
Sebaceous cyst of head	3	3	....	....	....	3
Ulcer of foot	1	....	1	....	....	1
Ulcer of neck	....	....	....	1	....	1
Ulcer (perforative) of foot	2	....	3	....	....	3
Ulcers (varicose) of leg	5	4	3	....	....	7
	23	14	12	1	1	28
URINARY SYSTEM						
KIDNEY						
Abscess of kidney	1	1	....	....	....	1
Hydronephrosis	3	2	2	....	....	4
Nephritis (chr. interstitial)	....	1	....	....	2	3
Nephritis (chr. interstitial), ascites	1	....	2	....	....	2
Nephrolithiasis	9	9	1	1	....	11
Nephroptosis	1	1	4	....	....	5
Movable kidney	4	3	1	....	....	4
Movable kidney, pyelonephritis	....	....	1	....	....	1
Perinephritic cyst	1	1	....	....	....	1
Pyelitis	....	1	1	....	....	2
Pyelonephritis	1	2	1	....	....	3
Pyonephrosis, pregnancy	1	....	....	....	1	1
Renal colic	1	1	1	....	....	2
Renal colic, hemorrhagic cystitis	1	1	....	....	....	1
Traumatic nephritis	....	....	1	....	....	1
	24	23	15	1	3	42
URINARY BLADDER						
Calculus in bladder	2	1	1	1	1	4
Calculus, tabes dorsalis	1	....	1	....	....	1

URINARY SYSTEM—Continued	Op.	C.	Imp.	Un.	Died	Total
Urinary Bladder—Cont.						
Cystitis (hemorrhagic)	....	....	1	....	....	1
Cystitis, cystocele	1	....	1	....	....	1
Cystitis, tumor of bladder	....	1	1	....	....	2
Incontinence of urine and feces	....	....	....	1	....	1
Retention of urine	1	1	1	....	2	4
Ulcer of bladder	1	1	....	....	....	1
	6	4	6	2	3	15
DISEASES DUE TO ANIMAL PARASITES						
Oxyuris vermicularis infection of colon	....	....	....	1	....	1
Tænia saginata	....	....	....	1	....	1
	....	....	....	2	....	2
CONGENITAL MALFORMATIONS						
Adhesions of lip and jaw	1	....	1	....	....	1
Branchial genetic cyst	....	....	....	1	....	1
Cleft palate	1	....	2	....	....	2
Contraction of foot	....	....	1	....	....	1
Hare lip	5	2	4	....	....	6
Double uterus	1	1	....	....	....	1
Hydrocephalus	2	....	....	....	2	2
Infantile uterus	4	3	1	....	....	4
Infantile uterus, retroversion	2	2	....	....	....	2
Undescended testicle	2	2	....	....	....	2
	18	10	9	1	2	22
DEFORMITIES						
Deformities of ears	1	1	1	....	....	2
Deformities of feet	....	....	1	....	....	1
Deformities of nose	2	1	1	....	....	2
Cicatricial contractures	3	1	2	....	....	3
Muscle-bound feet	1	....	1	....	....	1
Genu valgum	....	....	....	1	....	1
Talipes equinus	2	1	1	....	....	2
	9	4	7	1	....	12
LOCAL INJURIES						
Burns of chest, neck and back	....	....	1	....	....	1
Burns of face	....	1	....	....	....	1
Burns of shoulders and arms	....	1	....	....	....	1
Burns (extensive)	1	1	2	....	1	4
Burns (chemical) of leg, convulsions	....	1	....	....	....	1
Concussion of brain	....	1	4	....	....	5
Contusions of back	....	2	1	....	....	3
Contusion of elbow region	....	....	1	....	....	1
Contusion of face	....	....	1	....	....	1
Contusion of foot	....	1	....	....	....	1
Contusion of leg	....	....	1	....	....	1
Dislocation of astragalus	1	1	....	....	....	1
Dislocation of elbow, paralysis agitans	1	....	1	....	....	1
Dislocation of meniscus	4	4	....	....	....	4
Dislocation of shoulder	1	2	1	....	....	3
Displacement of transverse colon	2	2	....	....	....	2
Division of tendons of hand	1	1	....	....	....	1
Division of tendons of wrist	1	1	....	....	....	1
Foreign body in abdomen	1	1	....	....	....	1
Foreign body in finger	1	1	....	....	....	1
Foreign body in hand	1	1	....	....	....	1
Foreign body in knee	1	1	....	....	....	1
Foreign body in leg	2	2	....	....	....	2
Foreign body in œsophagus	2	2	1	....	....	3
Foreign body in pleural cavity	1	1	....	....	....	1
Foreign body in peritoneal cavity	1	1	....	....	....	1
Fracture of clavicle	....	1	1	....	....	2
Fracture of femur	2	3	2	2	1	8
Fracture of femur and tibia	1	....	1	....	....	1
Fracture of femur, pernicious anemia	....	....	....	....	1	1
Fracture of fibula	....	3	1	....	....	4

LOCAL INJURIES—Continued	Op.	C.	Imp.	Un.	Died	Total
Local Injuries—Cont.						
Fracture of fibula (Potts')	5	7	3	3	....	13
Fracture of humerus	2	3	2	1	....	6
Fracture of jaw	3	2	2	1	....	5
Fracture of neck of femur, pneumonia	....	....	2	....	....	2
Fracture of os calcis	1	1	....	....	....	1
Fracture of olecranon	....	....	1	....	....	1
Fracture of patella	2	1	3	....	....	4
Fracture of phalanx	2	....	2	....	....	2
Fracture of radius	3	3	1	....	....	4
Fracture of radius (Colles')	....	1	2	....	....	3
Fracture of radius and ulna	1	1	1	....	....	2
Fracture of ribs	....	2	1	....	....	3
Fracture of skull	5	6	5	....	2	13
Fracture of tibia	2	1	....	1	....	2
Fracture of tibia and fibula	2	1	1	....	....	2
Fracture of tibia (ununited)	1	1	....	....	....	1
Fracture of vertebræ	....	....	....	1	....	1
Fracture of vertebræ, alcoholism	1	....	....	....	1	1
Gangrene of foot	2	1	2	....	....	3
Gangrene (diabetic) of foot	3	....	2	1	....	3
Gangrene of foot, arterio-sclerosis	1	....	1	....	....	1
Gangrene of foot, nephritis	3	1	1	1	....	3
Gangrene (dry) of foot, nephritis	1	1	....	....	....	1
Gangrene (wet) of foot	1	1	....	....	....	1
Heat prostration	....	....	1	....	....	1
Laceration of cervix uterl	25	25	....	1	....	26
Laceration of perineum	46	45	3	2	....	50
Perforation of ileum	1	1	....	....	....	1
Rupture erector spinal muscle	....	....	1	....	....	1
Rupture of ligament of knee	....	....	1	....	....	1
Sinus of abdominal wall	2	1	2	....	....	3
Sinus of leg	2	....	2	....	....	2
Sinus of neck	1	1	....	....	....	1
Sinus of sacro-coccygeal region	1	....	1	....	....	1
Sinus of thigh	1	1	1	....	....	2
Sinus, perirectal	1	1	....	....	....	1
Sprain of ankle	....	....	1	....	....	1
Wound (gunshot) of face	1	1	....	....	....	1
Wound (incised) of neck	....	....	1	....	....	1
Wound (lacerated) of neck	2	1	1	....	....	2
Wound (lacerated) of hand	4	2	3	....	....	5
Wound (lacerated) of scalp	....	1	1	....	....	2
Wound (lacerated) of scrotum	1	1	....	....	....	1
Wound (incised) of abdomen	....	....	....	1	....	1
	154	151	70	15	6	242
DISEASES DUE TO MICRO-ORGANISMS						
Erysipelas	....	4	....	....	....	4
Gonococcus epididymitis	....	1	....	....	....	1
Gonococcus salpingitis	....	2	1	....	....	3
Gonococcus uterus and tubes	1	....	1	....	....	1
Gonococcus urethritis	1	1	....	....	....	1
Malaria	....	1	1	....	....	2
Pertussis	....	....	....	1	....	1
Rheumatism (ac. articular)	....	....	1	....	....	1
Scarlet fever	....	....	1	2	....	3
Syphilis (primary)	....	....	1	....	....	1
Syphilis (secondary)	....	3	1	....	....	4
Syphilis (tertiary)	....	....	1	....	....	1
Syph. adenitis, axillary and inguinal	....	....	1	....	....	1
Syph. gumma of scalp	....	....	1	....	....	1
Syph. gumma of liver	2	1	....	1	....	2
Syph. osteitis of femur	1	1	1	1	....	3
Syph. osteitis of tibia	1	....	1	....	....	1
Syph. fistula in ano	....	....	1	....	....	1
Tbc. abscess of shouider	1	....	1	....	....	1
Tbc. of bladder	2	....	....	2	....	2
Tbc. of bladder, nephritis	2	....	1	....	1	2
Tbc. of elbow	1	1	....	1	....	2
Tbc. of eyelid	1	....	1	....	....	1
Tbc. of epididymis, orchitis	2	2	1	....	....	3

DISEASES DUE TO MICRO-ORGANISMS—Continued	Op.	C.	Imp.	Un.	Died	Total
Diseases Due to Micro-Organisms—Cont.						
Tbc. of finger	1	....	1	....	....	1
Tbc. of foot	1	1	....	....	....	1
Tbc. of glands of neck	38	31	7	....	....	38
Tbc. of hand	1	....	1	....	....	1
Tbc. of kidney	4	4	1	1	....	6
Tbc. of knee	5	2	4	....	....	6
Tbc. of lungs	....	....	5	1	....	6
Tbc. of peritoneum	2	1	1	1	....	3
Tbc. of prostate and bladder	1	....	1	....	....	1
Tbc. of rib	3	2	1	....	....	3
Tbc. of spine	1	....	....	1	....	1
Tbc. of testicle	2	1	1	....	....	2
Tbc. of uterine tubes, pulmonary tbc	....	....	....	1	....	1
Tbc. of uterus and broad ligament	2	1	1	....	....	2
Tbc. costal cartilage pectoralis major	1	....	1	....	....	1
Tbc. fecal fistula	1	....	1	....	....	1
Tbc. lumbar abscess	1	....	1	....	....	1
Tbc. ovarian cyst	1	1	....	....	....	1
Tbc. peritonitis, thrombosis saphenous vein, prolapse of vagina	1	1	....	....	....	1
Tbc. keratitis	....	....	1	....	....	1
Tbc. salpingitis, peritonitis, fecal fistula	1	....	....	....	1	1
	82	62	45	13	2	122
LOCAL INFECTIONS						
Infection of hand and arm	1	....	1	1	....	2
Infection of herniotomy wound	1	1	....	....	....	1
Stitch abscess	5	5	....	....	....	5
	7	6	1	1	....	8
NEOPLASMS						
Adenoma of breast	4	4	....	....	....	4
Adenoma of endometrium	1	1	....	....	....	1
Adeno-carcinoma of colon	2	2	....	....	....	2
Adeno-carcinoma of rectum	....	....	1	2	....	3
Adeno-carcinoma of uterus	1	....	1	....	....	1
Adeno-fibroma of breast	4	4	1	....	....	5
Adeno-fibroma of uterus	1	....	....	1	....	1
Angioma of neck	1	....	1	....	....	1
Carcinoma of abdominal wall	1	....	2	....	....	2
Carcinoma of antrum	1	....	1	....	....	1
Carcinoma of bile duct	1	....	....	1	1	2
Carcinoma of bladder	4	3	1	....	2	6
Carcinoma of breast	40	21	17	4	2	44
Carcinoma of cervix uteri	4	1	1	1	1	4
Carcinoma of chest wall	1	1	....	....	....	1
Carcinoma of face and cheek	5	3	2	....	....	5
Carcinoma of glands (inguinal)	1	1	....	....	....	1
Carcinoma of intestines	9	1	5	3	5	14
Carcinoma of liver	1	....	....	1	....	1
Carcinoma of lungs and pleura	....	....	....	....	1	1
Carcinoma of neck	5	3	2	2	....	7
Carcinoma of œsophagus	3	1	....	1	1	3
Carcinoma of orbit	1	1	....	1	....	2
Carcinoma of ovary	8	2	3	1	2	8
Carcinoma of parotid gland	2	1	1	....	....	2
Carcinoma of pancreas	2	....	1	....	1	2
Carcinoma of rectum	9	2	6	2	1	11
Carcinoma of rectum, fibroma uteri, pulmonary thrombosis	1	....	....	....	1	1
Carcinoma of tonsil	2	1	1	....	....	2
Carcinoma of stomach	9	....	3	2	5	10
Carcinoma of tongue	1	....	1	....	....	1
Carcinoma of thorax, ribs, axillary glands, fracture of femur	....	....	....	....	1	1
Carcinoma of uterus	5	1	2	5	2	10
Carcinoma of vagina	1	....	....	....	1	1
Carcinoma of vulva	1	1	....	....	....	1
Cyst-adenoma of breast	2	2	....	....	....	2
Cyst-adenoma of ovary	3	1	1	1	....	3

NEOPLASMS—Continued	Op.	C.	Imp.	Un.	Died	Total
Neoplasms—Cont.						
Cyst-adenoma of neck	1	1	....	....	....	1
Dermoid cyst of ovary	5	5	....	....	1	6
Dermoid cyst of chest wall	1	1	....	....	....	1
Epithelioma of face	4	3	1	....	....	4
Epithelioma of forehead	1	....	1	....	....	1
Epithelioma of neck	2	....	2	....	....	2
Epithelioma of nose	3	3	....	....	....	3
Epithelioma of lip	5	5	....	....	....	5
Epithelioma of orbit	3	3	....	....	....	3
Epithelioma of maxilla	1	1	....	....	....	1
Epithelioma of tongue	1	....	1	....	....	1
Epithelioma of tonsil	1	....	1	....	....	1
Epithelioma of toe	1	1	....	....	....	1
Epithelioma of vagina	1	....	....	1	....	1
Epithelioma of vulva	1	....	1	....	....	1
Epulis	1	1	....	....	....	1
Exostosis of hard palate	1	1	....	....	....	1
Exostosis of os calcis	1	1	....	....	....	1
Fibromyoma of uterus	82	79	1	1	1	82
Fibromyoma of uterus, pregnancy	1	....	....	1	....	1
Fibroma of omentum	1	1	....	....	....	1
Fibro-sarcoma of femur	1	....	1	....	....	1
Glioma of ulna nerve	1	1	....	....	....	1
Lipoma of abdominal wall	1	1	....	....	....	1
Lipoma of buttock	1	1	....	....	....	1
Lipoma of chest wall	1	1	....	....	....	1
Lipoma of chest and arms	1	....	1	....	....	1
Lipoma of neck	2	3	....	1	....	4
Lipoma of shoulders	4	4	....	....	....	4
Lipoma of thigh	1	1	....	....	....	1
Lymphangioma of neck	1	....	1	....	....	1
Lympho-sarcoma of neck	1	....	....	....	1	1
Hemangioma of hand	1	1	....	....	....	1
Myxo-sarcoma of thigh	1	1	....	....	....	1
New growth of patella	1	....	1	....	....	1
Neuro-fibroma-lipomata (multiple)	1	....	1	....	....	1
Papilloma of bladder	1	1	1	....	....	2
Papilloma of larynx	1	....	1	....	....	1
Papilloma of toe	1	1	....	....	....	1
Papilloma of ovary, pregnancy	1	1	....	....	....	1
Polyp of rectum	1	1	....	....	....	1
Polyp of uterus	4	4	2	....	....	6
Sarcoma of bladder	1	....	....	....	1	1
Sarcoma of abdominal wall	1	....	1	....	....	1
Sarcoma of leg	1	1	....	....	....	1
Sarcoma of maxilla	3	1	3	....	....	4
Sarcoma of mediastinum, aneurysm of aorta	1	....	....	1	....	1
Sarcoma of testis	1	1	....	....	....	1
Sarcoma of neck	1	....	1	....	....	1
Sarcoma of sacrum	1	....	1	....	....	1
Sarcoma of sheath of thigh muscle	....	....	....	1	....	1
Sarcoma of tibia	1	1	....	....	....	1
Tumor of abdomen	1	....	....	1	....	1
Tumor of breast	4	4	....	1	....	5
Tumor of face	....	....	....	1	....	1
Tumor of intestines	....	....	....	1	....	1
Tumor of neck	....	....	....	1	....	1
Tumor of parotid	....	....	1	....	....	1
Tumor of prostate	....	....	....	1	....	1
Tumor of rectum	....	....	....	....	1	1
Teratoma of abdomen	....	....	1	....	....	1
Teratoma of testicle	1	1	....	....	....	1
	264	194	79	40	32	345
INTOXICATIONS						
Auto-intoxication	....	....	1	....	....	1
Diabetes mellitus	....	....	1	....	....	1
Diabetes, ulcers, nephritis	....	....	1	....	....	1
Gout	....	1	....	....	....	1
Morphinism	....	....	1	....	....	1
	....	1	4	....	....	5

	Op.	C.	Imp.	Un.	Died	Total
MISCELLANEOUS						
Donor in transfusion	....	2	....	....	....	2
Diagnosis not made	1	1	4	2	....	7
For observation	....	1	2	....	....	3
No pathological condition	2	3	4	9	....	16
	3	7	10	11	....	28
SUMMARY						
Alimentary System	919	872	75	37	48	1032
Cardio-vascular System	57	51	21	6	5	83
Connective Tissue	56	47	21	5	2	75
Ductless Glands	15	11	5	1	....	17
Muscular System	2	2	1	....	....	2
Nervous System	11	7	19	8	5	39
Osseous System	35	16	28	3	2	49
Reproductive System	533	512	49	20	18	599
(Mammary Gland)	9	6	3	1	....	10
Respiratory System	25	22	12	3	2	39
Sense Organs	37	25	20	2	1	48
Tegumentary System	23	14	12	1	1	28
Urinary System	30	27	21	3	6	57
Animal Parasites	....	....	....	2	....	2
Congenital Malformations	18	10	9	1	2	22
Deformities	9	4	7	1	....	12
Local Injuries	154	151	70	15	6	242
Micro-organic Diseases	89	68	46	14	2	130
Neoplasms	264	194	79	40	22	345
Intoxications	....	1	4	....	....	5
Miscellaneous	3	7	10	11	....	28
Total	2189	2047	512	174	122	2854

ALIMENTARY SYSTEM

INTESTINES

Cecostomy	1
Colostomy	7
Entero-colostomy	1
Entero-enterostomy	1
Enterostomy	8
Enterorrhaphy	2
Ileo-colostomy	4
Ileo-colectomy	3
Intestinal anastomosis	4
Jejunostomy	1
Proctoscopy	2
Resection of intestines	8

HERNIA

Femoral hernia repair	8
Inguinal hernia repair	107
Omental hernia repair	2
Umbilical hernia repair	8
Ventral hernia repair	20

LIVER AND BILE PASSAGES

Cholecystenterostomy	2
Cholecystectomy	17
Cholecystostomy	12
Cholecystotomy	8
Choledochotomy	1
Cholelithotomy	3
Duodenorrhaphy	1
Duodenostomy	1
Incision for abscess of liver	1

MOUTH, TONGUE AND TEETH

Extraction of tooth	1
Incision of alveolar abscess	3
Partial glossectomy	2

ŒSOPHAGUS

Dilatation of œsophagus	1
Œsophagotomy	2

PERITONEUM, OMENTUM AND RETRO-PERITONEAL TISSUES

Cellotomy	4
Closure of perforation	1
Division of adhesions	16
Exploratory celiotomy	35

PHARYNX, TONSILS AND NASOPHARYNX

Adenoidectomy	9
Adenoidectomy and tonsillectomy	78
Incision for peritonsillar abscess	3
Tonsillectomy	18

RECTUM, ANUS AND PERI-RECTAL TISSUES

Clamp and cautery	36
Dilatation of sphincter ani	9
Excision of fistula in ano	2
Excision of mucous membrane of rectum	4
Incision of fistula in ano	21
Incision of ischio-rectal abscess	7
Incision of peri-rectal abscess	1
Ligation of hemorrhoids	7
Proctectomy	1
Proctoscopy	2
Dissection of fistulous tract	2

STOMACH

Gastrectomy (partial)	1
Gastro-enterostomy	12
Gastropexy	2

VERMIFORM APPENDIX

Appendicectomy	219
Appendicectomy with drain	69
Appendicostomy	2
Appendipexy	1
Drainage of appendicular abscess	4

CARDIO-VASCULAR SYYSTEM

ARTERIES

Ligation of artery	2

VEINS

Ligation of vein	8
Phlebectomy	32

LYMPH GLANDS

Incision	4
Lymphadenectomy	44
Lymphadenectomy (tbc. cervical)	17

CONNECTIVE TISSUE

Excision of carbuncle	3
Excision of scar	5
Incision for abscess	40
Incision for cellulitis	33
Repair of fistula	6
Repair of scar	5
Repair of sinus	5

DUCTLESS GLANDS

Excision of goitre	1
Formation of fistula from parotid duct	1
Incision of parotid duct	1
Thyroidectomy	8

MUSCULAR SYSTEM

Excision of bursæ	2
Excision of ligaments	1
Excision of semi-lunar cartilage of knee	1
Myectomy	1
Tendon transplantation	3
Tenoplasty	1
Tenotomy	1

NERVOUS SYSTEM

BRAIN

Decompression	3
Drainage of abscess	2
Elevation of depressed fragments	4
Exploratory craniotomy	3
Subdural drainage	3

NERVES

Neurectomy	7

SPINAL CORD

Laminectomy	3

OSSEOUS SYSTEM

BONES

Ostectomy	8
Osteotomy	12
Osteotomy with drain	7
Reduction of fracture (closed)	1
Reduction (open) of fracture	4
Resection of knee	1
Resection of carpal bones	1

JOINTS

Arthrectomy	3
Arthrodesis	1
Arthrotomy	1
Excision of meniscus	2
Excision of synovial folds	1
Removal of foreign body	2

REPRODUCTIVE SYSTEM

OVARY

Excision of cyst	5
Incision for cyst	2
Oophorectomy	37
Plastic on ovary	3
Shortening ligament of ovary	1

UTERINE TUBE

Salpingectomy	44
Salpingectomy with drain	4
Salpingo-oophorectomy	85

UTERUS

Amputation of cervix uteri	2
Curettage	116
Excision of intraligamentous cyst	2
Hysterectomy (complete)	4
Hysterectomy (partial)	4
Hysterectomy (supravaginal)	64
Hysteropexy (round ligament)	25
Hysteropexy (ventral)	32
Myomectomy	6
Tracheloplasty	11
Trachelorrhaphy	12

VAGINA AND PELVIC FLOOR

Colpoplasty	5
Colporrhaphy	15
Colpotomy	21
Excision of cyst	2
Incision of cyst	1
Perineoplasty	26
Perineorrhaphy	24
Plastic repair of abscess	1

URETHRA

Urethrotomy	9

PENIS

Circumcision	18
Incision of scrotum for abscess	1
Meatotomy	1

PROSTATE

Prostatectomy (perineal)	6
Prostatectomy (suprapubic)	4

TESTICLES

Incision for orchitis	1
Orchidectomy	4
Transplantation of testicle	4

SPERMATIC CORD

Bottle operation for hydrocele	1
Excision of hydrocele sac	6
Eversion for hydrocele	1
Inversion for hydrocele	1

RESPIRATORY SYSTEM

LARYNX, BRONCHI AND TRACHEA

Tracheotomy	2

LUNGS AND PLEURÆ

Costatectomy	14
Decortication	1
Thoracotomy	19
Thoracostomy	3

NASAL CAVITY

Opening of lateral sinus	1
Plugging of nares	1
Submucous resection	9

ORGAN OF HEARING

Mastoidectomy (partial)	8
Mastoidectomy (radical)	4
Mastoidotomy	4
Paracentesis	4

ORGAN OF VISION

Curettment for tbc. of eyelid	1
Dilatation for cataract	1
Discission of cataract	2
Enucleation of eyeball	3
Excision of cataract	4
Excision of eyeball	2
Needling for cataract	1
Removal of lens	1

TEGUMENTARY SYSTEM

Excision of carbuncle	1
Excision of sebaceous cyst	3
Incision for furuncle	2
Onychectomy	1
Removal of foreign body	2
Skin graft	7

URINARY SYSTEM

KIDNEYS

Decapsulation	1
Nephrectomy	10
Nephropexy	3
Nephrolithotomy	7
Nephrotomy	4
Ureterectomy	2
Ureterotomy	1

BLADDER

Cystectomy	1
Cystorrhaphy	1
Cystoscopy	11
Cystostomy	1
Cystotomy	4

DEFORMITIES AND CONGENITAL MALFORMATIONS

Division of double uterus	1
Excision of scar	2
Plastic repair on cleft palate	1
Plastic repair on hare lip	3
Plastic repair on nose	1
Pozzi operation for infantile uterus	6

INJURIES

Opening of sinus	2
Removal of foreign body	6
Suture of wound	6
Wiring of jaw and teeth	1

DISEASES DUE TO MICRO-ORGANISMS

Incision of local infection	1

NEOPLASMS

Cauterization	6
Excision	81
Plastic on regions involved	16

AMPUTATIONS

Amputation through mid-forearm	1
Amputation of finger	4
Amputation at hip	1
Amputation through metacarpals	1
Amputation through upper thigh	1
Amputation through lower thigh	3
Amputation at knee	1
Amputation through middle leg	1
Amputation through metatarsus	1
Amputation of toe	11
Disarticulation at knee	1

MISCELLANEOUS

Radium treatment	18

ESOPHAGEAL STRICTURES.

ROBERT ABBE, M.D.

It is but just to a novel surgical procedure, that after a sufficient number of years' trial the results should be checked up and a fair record of its established value should be made.

By a fortunate observation, in 1892, in St. Luke's Hospital, while endeavoring to dilate a very tight resisting stricture of the lower esophagus, I found that a Billroth bougie (that is, a gum-elastic bougie, tipped with a metal conical point, in which a string was fastened for traction) was wedged so tightly that no reasonably safe pulling would bring it through the stricture. By accident of the moment, I happened to have another heavy braided silk thread alongside of it, passing from the open stomach wound to an opening in the upper esophagus, which I had made.

When the stricture resistance absolutely prevented the bougie being pulled through, a simultaneous pull on the parallel string moved the bougie unexpectedly forward. At once I saw that a back and forth, or sawing motion, of the independent string, wore away the resisting fibrous stricture while it was put on the stretch by the dilating end of the bougie. Larger and larger bougies at once followed as the string completed the rasping or safe cutting of the stricture, and the esophagus was enlarged to its full caliber in a practically bloodless manner.

An entirely new procedure was thus added to the armentarium of the surgeon in dealing with this hitherto inoperable disease of the esophagus. I say inoperable because, although numerous cutting instruments had been devised to divide these tough strictures, they were uniformly condemned by surgical authorities as dangerous to use, because the thin-walled esophagus lies parallel to, and in contact with the aorta and vena cava.

This happy experience, first published in the *Medical Record,* February 25, 1893, was accepted and adopted by surgeons generally, and has been incorporated in most surgical works as safe and efficient.

Without reviewing the large number of published and unpublished cases, I will speak only of my subsequent experience in our hospital.

The good results are lasting if properly followed.

The first case was of a young woman who had swallowed pure ammonia, with consequent inflamed esophagus and stricture. She was reduced to a desperate state when I did the above successful operation.

During the subsequent year a full-sized bougie was passed to the stomach; at first, twice a week, then once a week, then monthly. During the years following, she passed it herself, several times yearly, until, after 10 years, she gave it up, as there was no tendency to recurrence. When I saw her, more than 15 years later, she was in perfect health, and I could detect no stricture even with a bougie à boule. That particular patient had a stricture of no great length, perhaps a half inch, though very tight, admitting merely a thread, following a whalebone filliform passed up from the opened stomach.

Many cases which I have since operated on have uniformly shown long stretches of the esophagus (often one-third or one-half), showing tight, fibrous, solid remnants with the canal almost closed.

Two of these are beautifully shown in the pictures, Figs 1 and 2.

Another case, of which either of these pictures would be representative, was brought to me from Philadelphia, 6 or 7 years ago, and furnishes a fair illustration of what we may expect in the final outcome of such bad cases. The child was emaciated to a skeleton, and the best that could be offered to the parents by two of our most eminent surgeons, by other surgical methods, was, to create a gastrostomy opening and thus feed the child for the rest of its life. I first created such an opening and fed the child until it was strong and hearty. Eight weeks later I did the string cutting esophagotomy.

Dilatation was kept up for many weeks at first with anæsthesia for safety. Then, as the child bore it well, by easy passage of bougie.

The family physician persisted, for 2 or 3 years, patiently and conscientiously, to pass the bougie, and the child ate everything, as other children. He writes me now that she has grown to be a fine, robust girl, and has a normal acting esophagus.

It may be said of all these cases that they are caused by swallowing caustic or burning fluids. I have never seen or heard of a stricture following the long retention of foreign bodies in the esophagus, such as tooth-plates, toys, coins, etc., which necessarily make an ulcerated area after a few weeks. I judge nature is competent to dilate such narrowings by the ordinary bolus of food in

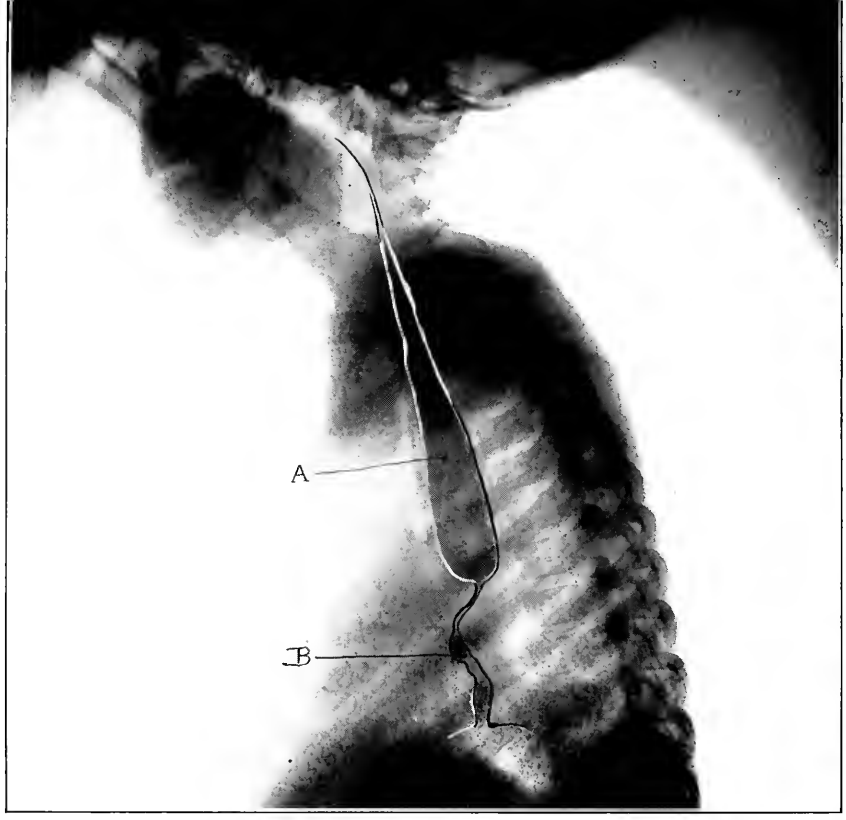

Fig. 1.—X-ray and bismuth picture, showing dilatation (A) of esophagus above stricture (B). It also shows the track of the minute lumen in the strictured portion. This picture was taken after partial dilatation, whereas Fig. 2 (the next case) was taken before any interference of an operative nature. Both of these pictures have been outlined with black and white to facilitate the tracing of the strictured portion.

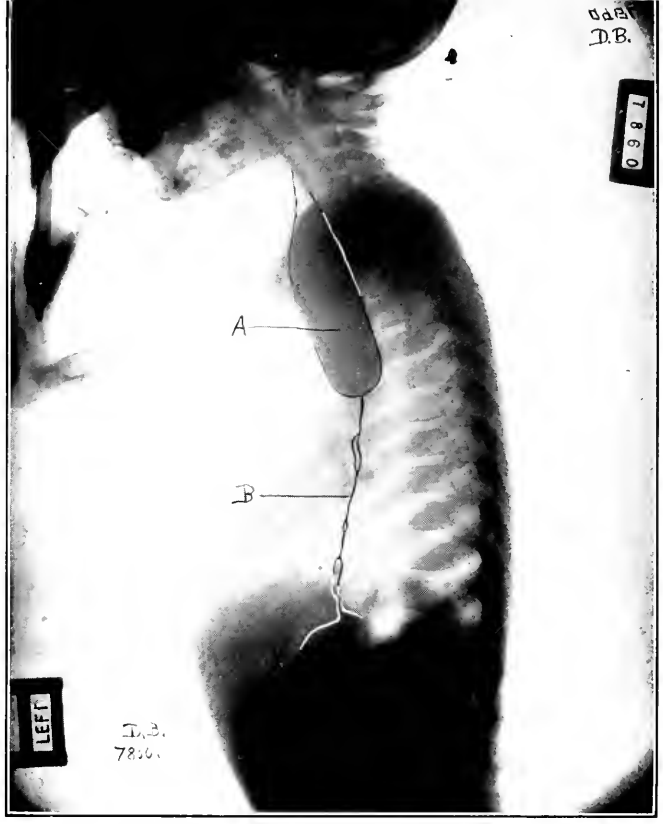

Fig. 2.—X-ray and bismuth picture showing dilatation (A) of esophagus above stricture (B).

deglutition. It is the destructive type of inflammation similar to the urethral infective type, which destroys the epithelial lining and replaces the mucous and muscular coats by fibrous tissue, which we have to deal with.

It may be asked—how can one expect ever to restore such a tube? It is a fair question, and can only be answered by saying it is never restored to normal. That is, the muscular coat cannot be replaced.

Nevertheless, a perfectly competent and practically useful tube is created by carving a channel through the fibrous mass—and keeping it open—until it has been lined by flat epithelium, through Nature's kindly and wonderful laws of repair, and until the contractile tendency of the formed tissue has ceased, as it does after months or years, according to the amount present.

The same law of stenosis goes on precisely as in urethral strictures, unless dilatation is kept up at longer and longer intervals. The occasional passage of a bougie is a very small penalty to pay for a perfectly restored swallowing apparatus.

Taken altogether, we can truly say that the annals of St. Luke's Hospital may be credited with the demonstration of a successful, safe and bloodless method of dealing with a bad surgical condition for which no other method is adapted. One may say that some strictures can be dilated, without cutting. That is true. And those should always be dilated. But the majority are absolutely undilatable after they have become indurated by time, and to these, fortunately, this method offers complete cure. The surgeon, however, must be sure to follow up his patient if the result is to be permanent. That happy issue is now demonstrated by this report of 20 years' use.

PAPILLOMA OF THE VOCAL CORDS.

ROBERT ABBE, M.D.

Warty vegetations on the vocal cords are the most obstinate of all surgical conditions in recurrence after removal—and most destructive to voice and breathing. It is said they sometimes change to cancer by irritation of the basal cells—but many do not.

One of the most extraordinary illustrations of this persistence of type is shown in a woman of 60, who was first treated by intralaryngeal excision by Dr. Elsberg, the pioneer laryngologist of America—who, more than 45 years ago, began to excise masses of these growths, and continued to do so 2 or 3 times yearly, during his life. He was succeeded by Dr. Lincoln, and later by Dr. Culbert, all experts, who, in order to give her breathing space, cleared away all visible growth every 6 months. Dr. Elsberg published her case (Trans. Am. Med. Assn., 1865) and Dr. Culbert reported upon it 40 years afterward (see "The Laryngoscope," St. Louis, September, 1904), giving pictures of the original masses as illustrated by Elsberg.

This case is one of four in which I have been called upon to use radium, and with the same effect, as shown by each case.

Dr. Culbert held a device between the cords containing 20 mg. radium, for one-half hour. Three months later he reported almost all growth had gone from one side—quicker than by any removal with instruments which he had ever done. One year later he examined and reports it "to be the cleanest he had ever seen it. One-third of the inside of the larynx is entirely free from papilloma." One year later she was breathing even better, without further treatment, and growths were smaller.

She then showed senile spinal paresis, and died.

Two other cases, presenting great difficulties, but with fair demonstration of the specific action in curing them that radium always shows in curing warts elsewhere, will be briefly mentioned before narrating the most brilliant result of a fourth case, herewith illustrated.

The first is that of a woman, voiceless and with stridulous breath-

Fig. 1.

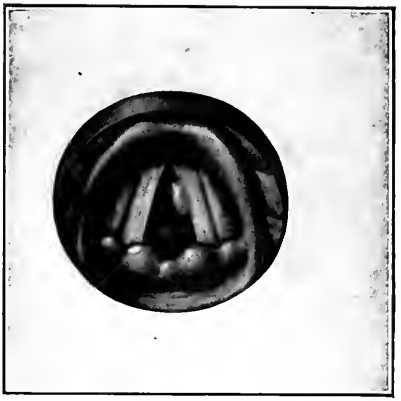

Fig. 2.

Fig. 3.

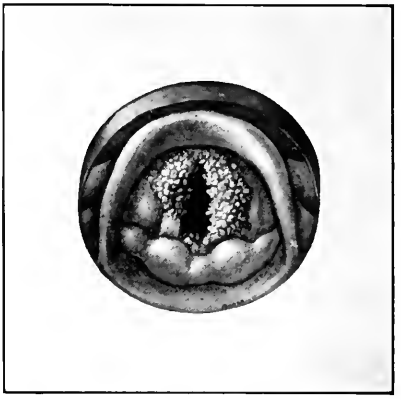

Fig. 4.

Fig. 5.

Fig. 6.

ing, from whom Dr. Josiah L. Barton had many times excised the papillomatous mass. To give her relief at first, I did a laryngotomy, and after excising the growths, applied monochloracetic acid to the base. Recurrence took place. Radium was then intra-laryngeally applied.

The result has been disappearance, and, later, small recurrence. The patient regained her voice well. The slight recurrence has given her no annoyance for a year past, and she has not come to the city, as she is entirely satisfied with her present condition—without further treatment.

The second case is of a young child, whose recurrences filled the larynx, and the laryngologist had made a permanent tracheotomy not only with no relief, but an extension had followed downward in the trachea itself, so that a mass had grown on the posterior face of the trachea, opposite the opening.

The child was so intolerant of laryngeal application of radium that I placed her in St. Luke's, and under ether, made a thorough use of strong radium, held in place one-half hour. The result was a diminution in the disease, but it required a second application, after 6 months, to further control its growth. At the present time, there is still a visible mass, about one-third of the original, hidden below the vocal cords, and a very small remnant in the tracheal wound. There may be one additional treatment required to cure it, but it seems at present that the final cure by radium will be accomplished. An interval of many months is usually the best manner of administration, inasmuch as the good effect always progresses that long before one can judge whether a sufficient dosage has been given.

The fourth case is a delightful demonstration of the cure of papilloma laryngis by radium:

A girl of 17 years had an unusually sweet singing voice, which she noticed became hoarse in July, 1910. She applied to Dr. Culbert in September following, who successfully removed a small tumor of the left vocal cord—which Dr. Ewing pronounced "fibroma" (Fig. 1). A rapid recurrence (Fig. 2), looking now like papilloma, was removed, but not examined. Again a rapid recurrence, looking now larger and more dusky, rather like a sarcoma than either papilloma or carcinoma. It occupied the central half of the cord, and overflowed into the ventricle. It was difficult to control the anæsthesia of this patient's larynx so as to make an adequate radium application, though it was carefully tried. The growth progressed and now seemed typi-

cally papillomatous—obstructing respiration (the voice was gone entirely) (Fig. 3). By the following June it occupied most of the left and much of the right vocal cord. The pictures accompanying show its varied stages, the condition immediately before operation being shown in Fig. 4.

On June 14th, I decided to make a thorough radium application under anæsthesia. Through a tracheotomy wound, I passed a wire up to the mouth and drew into the trachea a tube containing 100 milligrammes of pure radium, which I was able to suspend with accuracy between the vocal cords.

This I kept *in situ* half an hour, while ether was given through the tracheal tube. Nothing else was done except to allow the tube to remain a few days in the trachea, for safety. The wound healed at once on its removal.

Three months afterward (Fig. 5) the patient talked and sang perfectly.

Examination of the larynx showed and continues to show an apparently normal condition (Fig. 6), with clean, white vocal cord. The singing voice is restored completely, and is as sweet as ever.

This perfect condition remains after one year.

RUPTURE OF THE KIDNEY IN CHILDREN.*

CHARLES L. GIBSON, M.D.

Ruptures or other subcutaneous injuries are very uncommon in children, only 22 cases being reported in Watson's[1] tables. My experience comprises 4 cases of complete rupture in children from 8 to 12 years old, and a consideration of the conditions found furnishes some interesting features.

Case 1.—Barbara S., age 10. Admitted to St. Luke's Hospital Aug. 25, 1902. Two weeks ago was kicked by a horse on the right side of the body; unconscious for a while. Next morning urine contained some blood; none seen since. Some swelling of the right side developed, with a considerable amount of pain. Has had no chills, but there have been fever and sweating.

Physical examination showed a bright, healthy child, with a visible swelling of the right lumbar region. No superficial discoloration. The swelling was elastic, insensitive to pressure, flat on percussion.

Urine.—Acid 1018, no albumen.

Operation.—Right lumbar incision showed the swelling to be a large retroperitoneal accumulation of normal appearing urine. The kidney was ruptured in two, the lower pole entirely separated from the upper three-fourths of the viscus. Nephrectomy; good recovery. Discharged Oct. 3.

Case 2.—These details are as exact as I can furnish them from memory, the record being lost.

Boy, about 10, admitted to the Hudson Street Hospital, probably in the summer of 1907; run-over injury; abdominal symptoms; median laparotomy by a colleague; negative findings. Seen by me several days later; diagnosis of rupture of left kidney. Lumbar incision revealed complete tear of left kidney. Nephrectomy; good recovery.

Case 3.—James L., 12. Admitted November, 1909, to the Hudson Street Hospital. While running across the street, an automobile struck him in the left side, knocking him down. Scalp wound, requiring two stitches. Brought to the hospital by the guilty automobile. Soon began to complain of great pain and tenderness over the left kidney region.

Physical Examination: Tenderness and rigidity in left hypochondrium,

*Read before the Section of Surgery of the New York State Medical Society, April 17, 1912.

[1]Watson and Cunningham, Genito Urinary Diseases, vol. ii.

also some slight discoloration. Skin and mucous membranes of good color. Shortly after admission passed blood-tinged urine. Hemoglobin color index 70 per cent.

Operation: About eight hours after injury; left lumbar incision. Complete rupture of kidney in two pieces. Nephrectomy; drain; good recovery. Highest temperature, 100½° F. Discharged in three weeks.

Case 4.—M. S., girl, aged 8, admitted to St. Luke's Hospital July 26, 1910, complaining of pain in the "stomach." Two days before she had fallen a distance of four and a half feet, landing on the ground on the right side. Went home; complained of pain in her stomach, which has continued ever since. Bowels regular. No trouble with urination; no blood in the urine; has vomited twice.

Physical Examination: Negative, except for the abdomen, which shows general rigidity, with tenderness on the lower right side. Temperature, 102° F. Blood count: Leucocytoses, 2,500; polynuclears, 88 per cent. No urine record. Probable diagnosis, appendicitis. Immediate operation. Intermuscular incision. On separating the muscles a considerable amount of fluid blood evacuated. On opening the peritoneum a similar fluid escaped from the pelvis; the cœcal wall was the site of a considerable ecchymosis. Appendix normal (removed). The wound was dilated retroperitoneally to allow of a sponge being pushed up into the lumbar region; it returned bloody, but without evacuating any fluid. Injury to the kidney seemed probable; it could be palpated quite readily, but no obvious abnormality being detected (intra-capsular rupture), it was decided to await further developments.

The child recovered well, and seemed relieved. The urine the next day (17th) was: Neutral 1,034, very faint trace albumen, a few hyaline casts; July 20, acid 1,014, very faint trace albumen, a few leucocytes; July 21, acid 1,020, albumen 10 per cent, many red blood cells. In view of this last urine report, exploration was undertaken. Right lumbar incision. The true capsule was found intact, but distended with blood, and raised from the kidney. On opening it, the kidney was found broken completely in two, the lower smaller fragment showing beginning necrosis. Nephrectomy; drain. Perfect recovery. Discharged Aug. 9.

The case is interesting, showing a complete rupture resulting from a relatively slight trauma, leaving no mark on the body and producing absolutely no shock, the masking of kidney symptoms by the bruising of the lower abdominal muscles and the colon, the absence of any urinary symptoms till five days after injury, and also that the kidney may be divided completely in two without appreciable solution of continuity of its capsule. Four complete ruptures of the kidney in children under 12, occurring in the practice of one surgeon, seems unusual, in view of the small number of such cases on record. It is possible that these cases are really not so rare and may be overlooked, with disastrous results, by those who hesitate to interfere in dubious cases. The similarity of the lesions is interesting, being exactly alike

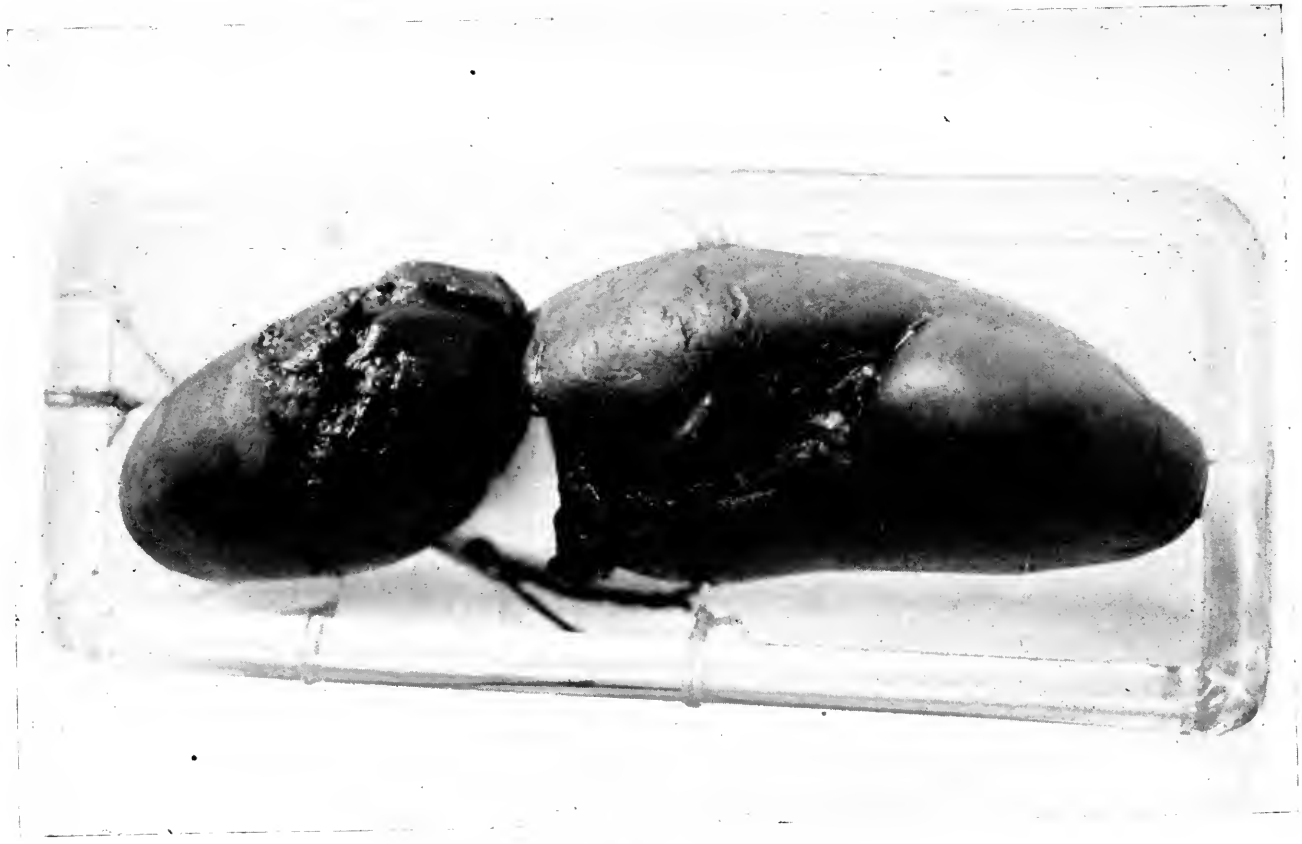

Fig. 1. (Case IV).—Ruptured kidney.

in all 4 cases—complete division of the viscus in 2 parts, the lower one being the lesser. In one instance the capsule remained untorn.

The fact that the kidney lesions were the same with the different kinds of violence seems to confirm the theory of "bursting" by hydraulic pressure. Also the line of rupture—vertical to the long axis at about the junction of the two lower thirds would seem to indicate that we had here an instance of a definite line of least resistance such as I have not seen indicated in any of the treatises on the subject.

Although the lesion in all these cases was severe, the symptoms, on the whole, were mild, and in several ways deficient. Nephrectomy was necessary in every instance, and successful; no other operation would have been permissible. Three of the children have been under observation and remained well.

As regards the etiology of such severe injuries, it is obvious that children are relatively little exposed to the various forms of trauma commonly encountered by active men (96 per cent of all cases). Most modern observations seem to corroborate Küttner's view, that the kidney being a semi-fluid body, bursts along the line of least resistance according to the law of hydraulics. Direct pressure from the lower ribs can also explain it. It is less easy, however, to understand the effects of indirect violence as from a fall on the feet. A point, however, to be borne in mind, illustrated in two of my cases, is that the severest form of damage may result from an injury unaccompanied by marks of external violence on the surface of the body in the kidney region or anywhere else. Possibly in some children a persistence of the infantile ptosis[2] may persist, leaving more of the surface unprotected by the thoracic bulwark. The particular vulnerability in childhood has also been ascribed to the minimum deposit of perinephric fat and the greater tension of the overlying peritoneum.

The extent of the lesion naturally runs the gamut from the mildest of superficial bruises to the complete rupture observed in my 4 cases—to the tearing away of the kidney from its vascular pedicle or the ureter or complete pulpifying from extraordinary crushes. In the less extensive injuries it is of practical importance whether the tear involves or extends into the pelvis—whether larger vascular trunks are destroyed, with resulting dangerous hemorrhage or jeopardizing the future vitality of portions of the organ—whether the injury is

[2]Aglave, Bulletin de la Soc. d'Anatomie de Paris, 1910, p. 595.

subcapsular, and finally, whether there is a coexistent tear of the peritoneum or injury of the contiguous viscera. Unfortunately, few if any of these lesions can be diagnosticated with certainty as regards their extent, particularly at a period when early interference may be all-important. A consideration of the nature of the violence is helpful. Injuries resulting from direct violence will probably produce a rupture of the kidney alone by "bursting" violence. Gross, direct violence, such as "run over" accidents, are more likely to result in complex lesions. The intensity of the violence is, however, not a trustworthy guide, as shown by Case I, where a complete rupture resulted from the kick of a horse that left no mark on the skin. It must also be borne in mind that a pathological kidney may rupture from the most trivial accident (Watson's case of the woman whose hydronephrotic kidney ruptured from muscular action—washing windows).

The loss of blood resulting from any of these injuries naturally varies. Generally speaking, it is rarely sufficient to endanger life quickly; it is, rather, the constant and recurring hemorrhage that is most to be dreaded. Even with extensive rents of the kidney, the integrity of the capsule tends, by tension, to check extraordinary bleeding.

As regards diagnosis, it may be stated broadly that a diagnosis of some degree of injury to the kidney presents little difficulty. Statistics give a history of hematuria in 80 per cent of the cases, and certainly, with painstaking microscopic urinary examinations, this figure would be increased. It will not ordinarily be difficult to exclude lesions of other portions of the urinary tract, *e.g.*, of the bladder, practically always complicated by a fracture of the pelvis. The history or evidence of an injury which may implicate the kidney will generally be elicited, pain, tenderness and eventually more or less pronounced signs of the extravasation of blood or urine, or both, in the marked cases, will accentuate the diagnosis and also indicate the side involved. For unusual cases and conditions, the cystoscope or ureter catheter may be used; but as a routine, these are uncalled for, as well as unwise, and in children can scarcely ever be used, and if requiring anesthesia, had better be replaced by a harmless and more satisfying exploratory and therapeutic lumbar incision.

What is most difficult is to determine the extent of the lesion, and particularly as regards the conditions which most urgently call for interference. The initial symptoms, with the exception of the degree

of shock and hemorrhage, do not present any features which sharply indicate the severity of the damage—it is rather on the development and sequence of secondary manifestation that we have to rely, or, perhaps, waste valuable time.

Very severe injuries or very mild ones may be usually diagnosed with readiness, especially with a definite knowledge and appreciation of the nature of the causative violence. For instance, a child is run over by a heavy wagon, as reported by a competent witness—there are extensive marks on the body, there is abundant and early, perhaps immediate, hematuria, there is marked shock. Given these conditions, there should be a severe laceration of the kidney and perhaps of other contiguous organs, possibly entailing a laceration of the peritoneum overlying the kidney. These complicating conditions may not always be obvious at the outset, although these marked and dangerous symptoms will manifest themselves later—too late, probably, to remedy them.

On the other hand, a lad may be hit a severe blow in boxing—the so-called "kidney blow"—feels a good deal of pain, may be temporarily dizzy or sick at his stomach, sooner or later the urine is tinged with blood. Such a history and such findings indicate a trifling condition requiring no active treatment.

It is, however, the cases of moderate severity or of incomplete symptoms that are the most difficult to judge. The degree of initial shock is alone no criterion; it may be intense, certainly, for a short time, with only a trifling injury; it may be insignificant or wanting, with the severest damage. The degree of hemorrhage is also misleading; a small vessel may bleed savagely for a while, and if the bulk of the hemorrhage finds a ready escape down the ureter we shall have an alarming picture for a perhaps trifling condition. On the other hand, mechanical obstacles—rupture of the pelvis or ureter (or blocking), clotting or absence of considerable hemorrhage from the kidney, may result in little hematuria even in the presence of the severest damage.

Absence of visible marks of external violence is no criterion, for complete rupture may occur despite this negative evidence (Cases I and IV).

The significance of a swelling in the flank varies a good deal. If considerable and early, it usually means extensive damage. Some of it may be due to the trauma to the abdominal wall, some to the bulk of the extravasated blood, some to the reaction of irritated intestines

inhibiting peristalsis, or to an actual lesion of the gut, or later, to a peritonitis due to extravasation of urine, or an infection of the retroperitoneal tissues or from associated injuries.

The amount of urine collecting in the tissues will depend on whether the injury involves a rupture of (a) the capsule, (b) pelvis, (c) ureter, and whether the urine can accumulate in a well-defined space, or whether opportunity is offered for extravasation into the tissues or the peritoneum. Tuffier has shown from animal experiments, and clinical observations have corroborated that the lacerated renal surface *per se* allows little or no urine to escape.

Later swellings may be due to secondary infections. A considerable and increasing, well-defined (colon pushed forward) swelling with remission of acute symptoms and absence of inflammatory signs would indicate the retroperitoneal accumulation of a well walled-off collection of urine whose escape down the ureter is shut off—exploratory puncture (if deemed wise) will prove the condition.

It is obvious that we are not able to diagnosticate accurately the extent of many of these lesions. We know also that many such injuries, while not rapidly producing death, may do so eventually on account of the many complications that may arise. My feeling is that we should not hesitate in dubious cases to complete our diagnosis by an early exploratory lumbar incision, which will also fill a useful and probably necessary therapeutic rôle. Not many years ago we thought ourselves competent to differentiate the several forms of appendicitis —few surgeons to-day care to take such a risk, and prefer to replace doubt with certainty, and I believe that the varying possibilities for harm of a kidney lesion furnish a reasonable analogy.

As regards prognosis, statistical data of large series of cases have been collected to show results both of the condition and the value of the various forms of treatment, but it is doubtful if the older figures have much value to-day.

Suter,[3] in 1905, found in a study of 701 subcutaneous injuries of the kidney:

	Per cent
Total mortality	18.6
" " of 131 treated by nephrectomy	16.7
" " " 143 conservative operations	14.6
" " " 427 treated expectantly	20.6

If these figures are of any value at all, certainly an expectant

[3]Suter, Beit, zur klin. Chirurgie, Band 47.

treatment which has a mortality of over 20 per cent does not make a very impressive showing. With modern technique, generalization of skilled operators, efficient means of combating shock, etc., to refrain from operation satisfied with a mortality of 20 per cent, cannot be accepted as progress. Watson showed, in a series of 99 cases of operation in which the condition of the kidney called only for minor procedure, there were only 7 deaths, the cause of death being found, generally, to conditions independent of the operation proper (injury of the other kidney, peritonitis). Watson has formulated the indication for treatment as follows:

Cases suitable for expectant treatment:

1. The milder forms of the injury.
2. The cases in which there is reason to believe that both kidneys have been injured, the signs being external evidence of injury on both sides, tumor in both loins, and anuria.
3. Cases in which there are injuries of other parts of the body of such grave character as to make futile any operative treatment of the renal lesion.

Cases demanding operative treatment:

1. All in which there is evidence of progressive hemorrhage, *e.g.*, increasing pallor, pulse of declining strength and increasing rapidity, sighing respiration, and, locally, a tumor in the loin which is increasing in size; or an increasing amount of free fluid in the peritoneal cavity in the cases complicated by intra-abdominal injuries.
2. Hematuria which persists for a long time, even though the quantity of blood is at no one time large; hematuria in which there is a large amount of blood, even though it has not lasted long; hematuria which recurs after having ceased; sudden cessation of a previously profuse hematuria, and, if there is no reason to believe that both kidneys are injured.
4. Cases in which there is evidence of intra- or perirenal suppuration, or of peritoneal infection.

My own feeling would be that we should refrain from immediate operation in (a) all milder cases, presenting no one symptom of any severity, and giving a history of injury which is presumably of no great violence; (b) cases of generalized injury with a very bad general condition, and absence of urgent kidney symptoms.

For the latter class I would urge an exploratory operation with an appreciable increase of any or all symptoms at an early date. Operation in some form, then, is indicated for all milder cases that show a tendency to increase their symptoms and for all other cases, barring those falling in class B. My attitude in the border-line cases would be, when in doubt operate, believing that by such a routine

measure we will not let some seemingly mild case slip through our fingers. As regards the time of operation, in general, one should operate as early as possible, but if the main symptom is not that of an increasing anemia (repeated examinations of the hemoglobin), one might well occasionally give the patient a few hours to pull himself together, though such a delay should not be entertained if we have associated intraperitoneal injuries calling for prompt relief.

As a rule, the incision should give an extra-peritoneal approach by the lumbar route—it is the most direct, avoids infecting the peritoneum, and does not require handling and blocking off of protruding intestines. Moreover, it will provide the safe and efficient drainage demanded in most of these conditions. An anterior incision should be reserved for injuries which presumably involve the intraperitoneal organs—even in these cases a supplementary lumbar incision for drainage may be indicated, particularly if a nephrectomy is not performed.

Nephrectomy should be reserved for the cases in which the integrity of the kidney cannot be preserved, and it is obvious that hemorrhage cannot be effectually stopped or prevented otherwise, or the outflow of the urine into the ureter cannot be efficiently restored. In the event of doubt arising, regarding the integrity of the other kidney, nephrectomy may be deferred until sufficient information is obtained. Meanwhile, the injured kidney should be attended to, peritoneum if torn, sutured or packed, laceration sewn if advisable, the pelvis drained and the whole or part of the wound packed and drained efficiently. Where nephrectomy is not required suture or packing with drainage will suffice. How much more efficient suture rather than packing a lacerated area will prove, is to me an open question. I think not much time should be lost in performing it and it should perhaps be reserved for cases in which packing may less efficiently check bleeding. The main indication is to provide free drainage, which will minimize the disastrous secondary effects of injury and extravasation.

This paper is written to call attention to the fact that rupture of the kidney in children is probably commoner than generally estimated. That the lesion is frequently severe, consisting of a complete division of the kidney into unequal halves. That shock and other symptoms may be slight and out of proportion to the gravity of the lesion. That operative interference should be more freely employed and gives good results.

THE SURGICAL TREATMENT OF COLITIS.*

CHARLES L. GIBSON, M.D.

My interest in the surgical treatment of colitis dates back to 1900, when I devised a line of treatment intended to replace the only means recognized then as efficient, namely, artificial anus. This method of mine is the one most generally used to-day; but its origin and usefulness has been considerably obscured by the introduction of a modification in the technique of my original operation by Weir, substituting for my valvular cæcostomy appendicostomy. I hope to be pardoned if I make this paper the subject of a review of the development of the more modern treatment.

As regards the value of the artificial anus, I had been very skeptical, in the brief years it flourished, whether the cure was not worse than the disease. Moreover, the evidence advanced of its curative value was oftentimes unconvincing, and it was natural that the relief obtained should only be partial unless a complete artificial anus was made, absolutely eliminating the fecal current from reacting the colon.

If a complete artificial anus were made, its eventual repair required a severe operation with a high mortality.

I set out deliberately to devise a form of operative treatment that should be the antithesis of the artificial anus, allowing of no escape of fecal contents.

I felt that if the principles of ordinary surgical drainage and cleanliness could be applied to the large intestine, we would have gained considerably in facilitating the healing of the ulcerated surfaces. That result I thought could be brought about by devising a means of frequently flushing the large intestine, greatly diluting its irritating contents and removing them from prolonged contact with the ulcerations. So if we could give the patient an opening in the bowel for access to its contents and yet prevent their egress, the problem would be solved.

The Kader form of gastrostomy had then come to be considerably

*Read before the International Surgical Association at Brussels, September, 1911.

employed, and all I had to do was to use the same technique in the cæcum which I did. At the outset I believed that by making a suitable incision (intermuscular) we should have a small and easily controlled wound, confinement to bed for its healing 10 days or less, the patient could then receive ambulant treatment or administer it himself by introducing the tube several times a day and flushing out the bowel with various appropriate solutions. During the intervals neither tube nor dressing need be worn, and the closure of the wound would be automatic as the discontinuance of the passage of the tube for a few days would allow of the valve action to become permanent.

All these theoretical requirements were found in general to be feasible in practice; but owing to the introduction of appendicostomy two years later, the origin of the method was lost sight of. I was a long time in getting an opportunity to perform this operation myself; but two of my kind friends, to whom I described this procedure, were good enough to make a trial of it at my suggestion.

Dr. P. R. Bolton performed it in 1900, reporting the case in the *Medical Record* for March 16, 1901, and in November, 1901, Dr. F. H. Markoe also performed it at my suggestion. My first case was performed later in 1901. The method was described in a paper[1] read by me March 5, 1902, in Boston, but publication was delayed till September.

Dr. Weir, in April, 1902, did my operation at my suggestion. The same day he had a second case, and having had some difficulty with my technique (tube was pulled out after being put in place), decided to use the lumen of the appendix as the channel. He lost no time in getting into print, so that, when my article appeared, appendicostomy had already been claimed as the proper treatment for colitis, and is generally so used. I think, whatever its merits from the technical standpoint, that the modern treatment owes its origin distinctly to me. That is, if I had not shown Dr. Weir how to do a valvular colostomy, he never would have thought of treating colitis except by the formation of an artificial anus.

I cheerfully recognize the merits of appendicostomy. It is a little simpler for a person without much surgical skill to perform, and therefore safer. The appendix may, however, not be of a suitable size or position (retrocæcal) to lend itself properly to the procedure, and the

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[1]The Creation of an Artificial Valvular Fistula for the Treatment of Chronic Colitis (Boston Medical and Surgical Journal, Sept. 25, 1902).

patient has to wear constantly a dressing, which is not the case with my technique.

So there still remains some sphere of usefulness to the original operation, and I repeat its original description, as given in the *Boston Medical and Surgical Journal,* September 25, 1902.

The technique is as follows: A small incision—preferably the McBurney intermuscular—is made over the caput coli. If desirable, the anæsthetic can be discontinued as soon as the peritoneum is opened. Nitrous oxide gas anæsthesia might be used. With an intelligent and self-controlled patient local anæsthesia might suffice. Should there be any difficulty in bringing the colon to the surface, I see no positive disadvantage in utilizing the lower ileum. Two Lembert sutures, half an inch apart, are inserted, and the cæcum opened between them. A soft catheter, about 30° F., is introduced so that it projects well into the bowel, and the original sutures tightly tied. The wall of the gut is further infolded around the tube in two superimposed layers. The ends of the superficial layer are used to suture and hold the cæcum to the musculo-aponeurotic structures. The tube may also be secured in place by passing a finer catgut stitch through its wall. The abdominal wound is closed at the angles, or packed. It will be safer not to begin irrigation before 3 or 4 days. The tube may be withdrawn in a week or 10 days, being introduced only when necessary for the irrigation, and withdrawn so soon as it has served its purpose. If our ideal has been attained, there will be no leakage, even when the colon is visibly distended. Treatment should be persisted in till a cure is obtained. Closure of the fistula occurs spontaneously with the discontinuance of the daily passage of the catheter.

It seems to me unwise, if not impossible, to attempt at present to formulate any indications for the employment of this measure. From what has been related, it is fair to say that certain forms of colitis can be cured by it. It may be objected that such cases and the ones here described are of the milder variety that would yield to the orthodox treatment. Personally, it seems that the results have been more direct, progressive and prompt than are attained by the non-operative measures.

On the other hand, I do not cherish any illusions regarding certain forms of ulceration, such as the tubercular, that may be properly considered as incurable, especially when accompanied with similar or more extensive changes in the small intestine. Actual experience only can determine whether by frequent cleansing of these ulcerating

surfaces and by neutralization of the products of decomposition we can somewhat ameliorate the symptoms, and if to an extent that warrants actual interference.

With regard to the therapeutic agents that may prove of value when so locally applied, I can only indicate those ordinarily employed. For the present I shall rely principally on the mechanical cleansing by flushing the bowel with an appropriate bland solution, such as the normal saline. It may either be used as a continued irrigation, escaping through the rectal tube, or the colon may be filled to moderate distention, say 3 quarts, and subsequently evacuated. The frequency should be established by the tolerance of the bowel and the urgency of the symptoms. At the beginning, if well borne, I should prefer to repeat the irrigation at regular intervals of 8 or 12 hours, possibly oftener. Agents destined to exert a direct influence on the ulcerating surfaces will naturally act better after the preliminary cleansing. They should be introduced separately from the saline, or after it has been evacuated. The bowel should be flushed with plain water prior to the use of substances such as $AgNO_3$, which combine with the saline.

The required therapeutic agents will also vary somewhat with the nature of the colitis. Gradually increasing strengths of quinin and methylene-blue have been recommended for the amœbic form. Nitrate of silver in strengths increased from 1-20,000 will, I think, prove the best single remedial and stimulating agent. The whole gamut of the milder non-poisonous antiseptics, especially of the naphthol group, may be tried, as well as the ordinary astringents. Small doses of iodoform in emulsion might be tentatively tried in the tubercular form. Glutol, a non-irritating derivative of formalin, which acts so admirably in ordinary suppurations, might also be employed. The patient should be on an appropriate, chiefly proteid, diet.

My own experience is very small, but gratifying. Six cases. One tubercular case (unsuitable) was not improved. Four cases were cured. In one subsequently operated upon by another surgeon for another condition, marked healing of many of the ulcerated areas was found. One patient almost moribund was operated upon with local anæsthesia very satisfactorily and was completely restored to health.

None of these cases was of the amœbic variety, which I believe is hard to cure by this or any other means, and are liable to undergo relapse sometimes after long intervals of freedom from symptoms.

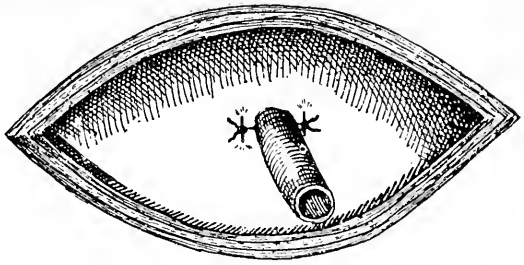

Fig. 1.—Tube introduced and held in place by the suture placed on either side.

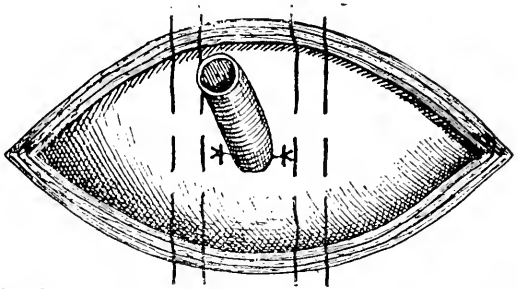

Fig. 2.—The first row of inversion sutures in place but not tied.

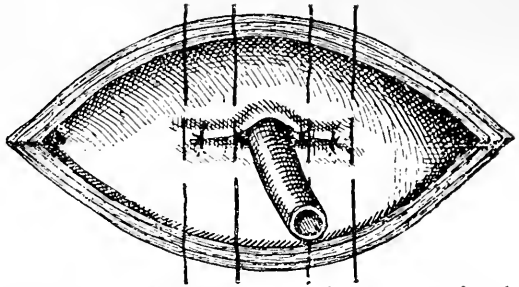

Fig. 3.—The first row of inversion sutures has been tied, the second row is in place but not tied.

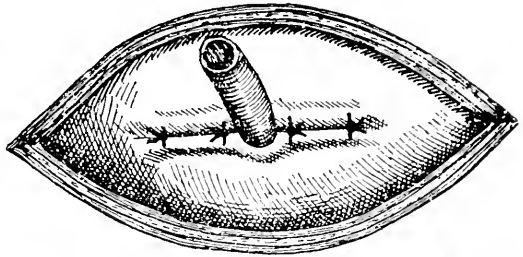

Fig. 4.—Operation completed. The tube is infolded in a furrow of the cæcum.

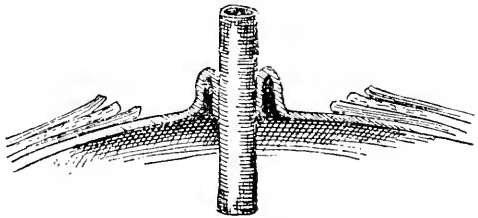

Fig. 5.—Showing the formation of the valve as seen from within the lumen of the bowel.

FECAL CONCRETION IN THE FALLOPIAN TUBE.

WALTON MARTIN, M.D.

On March 8, 1911, a Swedish girl, 20 years old, unmarried, was admitted to the hospital. She had been ill for 2 weeks. During that time she had had severe sharp pain in the lower right quadrant of the abdomen. The pain had not been constant, but had occurred at intervals. She had felt ill, and had had fever. There had been no disturbance with bowel or bladder.

On examination, there was well marked rigidity on both sides of the lower abdomen, but it was more marked on the right side. The patient looked ill. The temperature was 101°, the pulse 142.

The diagnosis of appendicitis was made, and operation was done as soon as the patient could be prepared. On opening the peritoneum, there was a gush of foul-smelling pus. The appendix had partly sloughed away and only the proximal end could be found. This was removed and a drainage tube introduced.

The patient made a slow but satisfactory recovery and left the hospital 5 weeks later, with a normal temperature. There was still, however, a discharging sinus at the site of the incision. The discharge was purulent and foul-smelling, but not fecal. A probe could readily be passed for several inches along a fistulous tract.

The patient returned to her work, but reported at the hospital from time to time, and on August 7, 1911, 4 months after her first operation, she was again admitted, as she still had the discharging abdominal sinus. This sinus seemed to have changed little since she had left the hospital. From time to time it had discharged small amounts of very foul pus, and she had had, at times, considerable pain in her side. It was evidently not a fecal fistula, and the persistence of the sinus was supposed to be due to the failure to remove the distal portion of the appendix. It was supposed that the presence of this distal portion was causing the trouble. An operation was advised.

On August 8, 1911, an incision, circumscribing the old scar, was

made, and the fistulous tract carefully dissected out. The tract led downward and inward between loops of intestine, until it reached a dark purple, tubular mass about the size of the index finger; from the end of this structure pus was exuding through a pin-point opening. Followed mesially this structure became narrower and finally joined the uterus. It was obviously the uterine tube. It was removed, and the abdominal wall closed. The wound healed satisfactorily, and the patient left the hospital at the end of 3 weeks.

The specimen removed was tubular and 8 cm. long. It measured 0.5 cm. at the uterine end, and 2 cm. at the distal end. On cutting it open, a fecal concretion, about 1 cm. in length, was seen in the lumen of the thickened distal portion. It was identical in appearance with a fecal concretion such as is usually seen in the appendix. There was pus in this portion of the tube; it had a foul, fecal odor. The fimbriæ at the outer end of the tube were turned in, so that the end of the tube looked club-shaped, as in the ordinary pyosalpinx. Microscopic examination showed the walls of the Fallopian tube thickened and infiltrated with round cells.

The concretion had evidently been freed during the attack of appendicitis by the sloughing away of the appendix, and had been taken up by the Fallopian tube, where it had found lodgment for 4 months. The irritation of the concretion in the tube caused the constant escape of pus through the end of the tube into the abdominal sinus.

I have been unable to find the record of a similar case.

EXTENSIVE EPITHELIOMA OF THE CHEEK WITH SECONDARY INVOLVEMENT OF THE GENIAL GLANDS.

H. H. M. Lyle, M.D.

Although the genial or facial glands were not mentioned by the majority of the older writers (Richet, Bouchard, Sappey, etc.), Mascagni described them in 1787, distinguishing the supra-maxillary and buccinator groups. Boyer, Jacob and Cruveilhier also mention them. In 1887, Poncet called attention to the clinical significance of these glands; his work was further extended by his pupils, Vigier (1892), Albertin (1895). This clinical work stimulated an interest in the subject and brought out researches by Princetau (1899), Cappette-Lapléne (1899), Buchbinder (1899), Küttner, Trendel, Thévenot (1900).

The glands are found in 65 per cent of the cases. According to Cunéo and Poirer, they can be divided into three sets. An inferior or supra-maxillary group, situated on the external surface of the inferior maxilla, close to the facial vessels. The middle or buccinator group (Molar of Testut and Jacob) are situated on the external surface of the buccinator, in front of the anterior border of the masseter; they are in close relation to Stenson's duct. The superior or molar group when present, are found along the ascending branches of the facial, one in the supra-orbital region, a second in naso-genial fold, and a third on the malar bone.

Trendel has collected 25 cases of secondary cancerous involvement of these glands; cases are also reported by V. Bruns, Küttner and others.

In the light of these facts the following case occurring on the service of Dr. Gibson, is of interest:

The patient, a man, 52 years old, was admitted to St. Luke's Hospital September, 1910. He had a tumor of the left cheek, of 11 weeks' duration. Ten days previously his physician had incised the tumor, but no pus was found.

On examination, there was a large, indurated swelling occupying the left

cheek and involving the angle of the mouth. In the center of the mass there was a discharging sinus. The buccal surface showed a cauliflower-like growth, which was not attached to the jaw. The sub-maxillary lymphatics were enlarged and hard. A section of the growth removed for examination showed it to be a squamous-celled carcinoma.

Operation.—A wide excision of the growth, including the angle of the mouth, was made, and the defect closed by a modification of the Dowd operation, plus a flap taken from the neck. Primary union resulted. Ten days previously a block dissection of the neck and sub-maxillary and submental regions had been done.

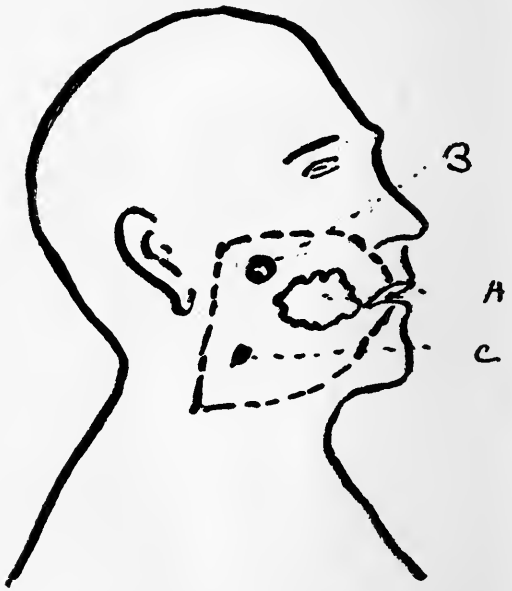

Fig. 1.—A, original growth. B, recurrence in Buccinator group of genial glands. C, recurrence in the inferior group. The dotted outline indicates the amount of skin removed.

Four months later, when the patient returned for observation, an examination showed that the mucoperiosteum of the jaw had been involved, and there were two distinct hard nodules on the cheek, one just below the center of the malar bone; the second an inch lower down and posterior to the angle of the mouth. There was no cervical involvement.

At a second operation, virtually the whole cheek below the malar bone was removed, along with the fascia and fat covering the masseter. In this fascia, the buccinator and supra-maxillary groups of the genial

Fig. 2.—The result, after the removal of a large portion of left cheek, the angle and floor of the mouth, and half the body of the lower jaw.

glands were found to be involved. The left half of the body of the jaw, with the involved mucous membrane of the floor of the mouth, was removed. Cutaneous flaps were obtained from the neck and the right side of the chin. Considerable difficulty was experienced, owing to the cicatricial tissue left from the previous operation. The buccal lining of these flaps was obtained by splitting the mucous membrane of the tongue and turning it upward, after the method suggested by Sonnenburg.

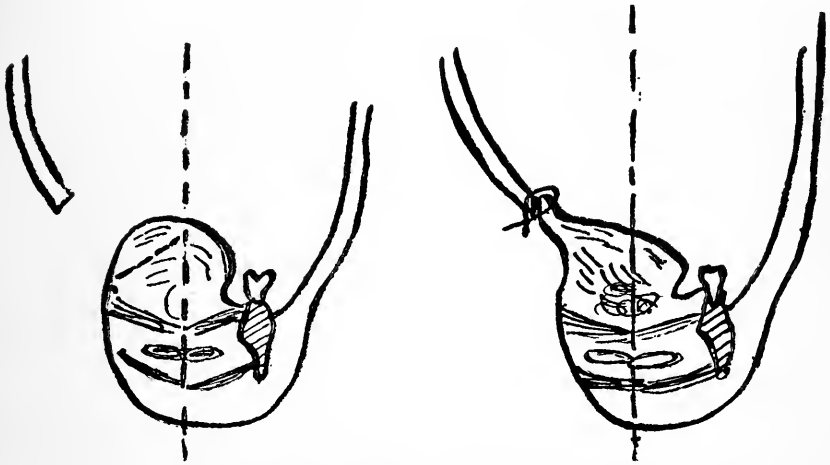

Fig. 3.—Sagittal section through the mouth, after removal of a large portion of the cheek and half the lower jaw, showing the method of closing the defect by flap from the tongue.

Three months later a secondary plastic operation was performed to widen the cavity of the mouth.

The points of interest in the case are: (1) the involvement of the genial glands, failure to recognize and remove these at the time of the primary operation having been one of the causes of the recurrence; (2) the use of a flap of mucous membrane from the tongue to line the cheek.

THE BOTTLE OPERATION FOR HYDROCELE OF THE TUNICA VAGINALIS—TEN CASES—THREE FAILURES.

H. H. M. Lyle, M.D.

In order to get the true value of any surgical procedure it is necessary to report the failures as well as the successes. In Keen's Surgery, volume IV, page 607, in describing the "Bottle Operation," Bevan quotes E. Wyllys Andrews as follows: "I recommend it without reserve and do not hesitate to urge that it supersede the older operations."

The following is a brief account of our experience with the "Bottle Operation." In a series of 10 cases we have had 3 failures. The first of these failures could not be justly charged to the operation. The patient had a chronic cardiac condition, which might have been a factor in the passive congestion of the reversed sac. In the second case, the surgical indications for this operation were apparently perfect. The failure was a complete surprise, and led us to think that it was due to some error in technic. As the patient refused further operative treatment, the cause of the increased size of the sac and testicle remained problematic. In the third case, with the previous failures freshly in mind, the operation was carefully and deliberately carried out. The hydrocele tumor disappeared, but its place was gradually taken by a tumor composed of thickened sac and testis. This secondary condition was little or no better than the primary. Two months later the reversed sac was excised by Dr. Douglas. The sac wall, which measured one-half inch thick, was composed of edematous connective tissue.

The patient was shown before the New York Surgical Society, November 8, 1911. The discussion of the case brought out the fact that this operation had failed in the hands of other surgeons. Dr. A. V. Moschcowitz said that he had abandoned the operation on account of failures, and said there was also a good theoretical reason why this operation should not be done. In some cases the testis are fastened to the bottom of the scrotum by the remains of Hunter's

ligament. To perform this operation properly, in such cases, the ligament has to be cut. If this has to be done, it is just as easy, or easier to cut the sac away, that is, to perform Von Bergmann's operation.

The object in reporting these failures is to emphasize the fact that even in selected cases, "the bottle operation" may give as unreliable operative results as the allied operations of Doyen, Jaboulay and Winklemann.

INTRADURAL SECTION OF THE SIXTH, SEVENTH, EIGHTH AND FIRST DORSAL POSTERIOR NERVE ROOTS FOR INTRACTABLE BRACHIAL NEURALGIA—FAILURE TO RELIEVE THE PAIN—LATER SECTION OF THE CORRESPONDING ANTERIOR ROOTS WITH NO RELIEF.

H. H. M. Lyle, M.D.

The patient, an engineer, 43 years old, was referred to the service of Dr. C. L. Gibson by Dr. W. Bastedo, with a diagnosis of Intractable Brachial Neuralgia. Three months previously to admission he was violently injured by a lever striking him on left side of his neck at the level of his jaw. He was unconscious for 24 hours. The left arm was completely paralyzed, and he suffered intense pain in the neck and arm. An exploratory incision showed that the roots of the cervical plexus were torn. The arm was amputated. At the present time he complains of an intense neuralgic pain in his missing hand and arm.

Physical Examination.—Spare man, aged 43, looks haggard and worn. Heart, lungs and abdomen normal. Left pupil contracted and undilatable. There is a vertical scar, 2½ inches long, at the anterior border of the left sterno-mastoid. The left arm has been disarticulated at the shoulder. The resultant scar is freely movable and the stump is not sensitive to pressure. The bony parts appear to be normal except for a slight prominence of the left clavicle and upper ribs; there is slight lateral curvature of the spine. As the arm was missing, no notes regarding anæsthesia are available. The patient was examined by Dr. Pierce Bailey, who recommended an unilateral intraspinal division of the left 7th and 8th cervical and 1st dorsal posterior nerve roots. X-ray examination of spine is negative.

Operation, December 1, 1910—Unilateral laminectomy with section of 6th, 7th, 8th cervical and 1st dorsal posterior nerve roots, by Dr. Lyle.

With the patient in the ventral position, an incision 4½ inches long was made in the cervical region, the muscles separated and a

hemisection of the laminæ made, according to Taylor's method. The dura was then opened, the posterior roots of the last three cervical and the first dorsal were identified, hooked up and cut. The posterior root of the sixth cervical had been torn away from the cord. The dura was sutured with a fine catgut and the wound closed.

There was a moderate post-operative reaction. The patient remained free from pain for 5 days, then he began to complain of pain in the thumb, hand and arm; this pain gradually increased, and at the end of two months was, if anything, more intense than before the operation.

As an explanation of the failure of posterior root section to cure certain cases, it has been stated that there are additional sensory paths in the anterior roots, and in such cases, these possible sensory paths must be cut in order to obtain a cure. With this point in view, Dr. Taylor decided to section the anterior roots. As the arm was already gone, this appeared to be an ideal case to try it in. Six months after the original operation, Dr. Taylor cut the anterior roots. This operation has failed to relieve the pain.

The points of interest in this case are the tearing away from the cord of the posterior root of the sixth cervical, and the persistence of the intense pain in the hand and arm after a complete section of both the posterior and anterior nerve roots of the 6th, 7th, 8th cervical and 1st dorsal.

GUMMA OF THE LIVER AS A SEQUEL TO YAWS.

H. H. M. Lyle, M.D.

Patient, male, age 49 years, native of West Indies (Grenada).

Family History.—Father and one uncle died of carcinoma of the stomach.

Past History.—The patient had had gonorrhœa 15 years ago, and yaws (frambesia), 39 years ago. No history of syphilis. Has complained of gastric trouble for 30 years, at various times has been treated for gastritis, ulcer and carcinoma.

Present History.—Patient complains of a constant pain in stomach; this comes on an hour after eating, and persists until the following meal, or until relief is obtained by vomiting. The constant pain has kept him awake at night. Lately he has noticed a fulness in the upper epigastrium. Is weak, and has lost 40 pounds in weight. The blood examination is negative; the analysis of the stomach contents shows a hyperacidity.

Physical Examination.—The patient is greatly emaciated. No glandular involvement can be made out. There are several old scars on the arms and legs; these are said to be results of yaws. In the midline of the abdomen, 1 inch below the ensiform cartilage, there is a smooth, hard mass, which apparently lies below the edge of the liver. The spleen is not enlarged. A preoperative diagnosis of carcinoma of the stomach, starting from an old ulcer, was made.

Operation by Dr. Lyle.

A smooth, round tumor, the size of a mandarin orange, was found on the anterior surface of the left lobe of the liver. The liver was enlarged and congested. The stomach, pancreas, and spleen normal, the mesenteric glands are not enlarged. As an extended search failed to reveal any other lesion, a diagnosis of gumma of the liver was made and the abdomen closed.

Two Wassermann tests were made, the first was negative, the second doubtful. After an intravenous injection of salvarsan, a third Wassermann test was made, which was positive. Under specific treatment, the tumor has disappeared and the liver has grown smaller.

The interest in this case rests largely on the possible relationship between yaws and syphilis. The patient and two other members of

his family were isolated in a hospital given over to the treatment of yaws. Under these circumstances, it is reasonable to assume that the diagnosis of yaws was correct.

It has been said that an attack of yaws gives an immunity to syphilis. If this is the case, the patient must have had syphilis before yaws.

CHRONIC PERISIGMOIDITIS WITH PARTIAL VOLVULUS.

H. H. M. Lyle, M.D.

The patient, male, 50 years old, has for a considerable time suffered from attacks of pain and distention in the left iliac region. These symptoms have been distinctly localized. The attacks have been preceded by severe frontal headaches, and no relief could be obtained until the distention was reduced by free bowel movements. Two days prior to his admission to the hospital, he had a severe headache, which compelled him to give up his work. He took a cathartic, which set up violent peristalsis, resulting in 10 or 12 movements of the bowels. These were at first fecal, but soon became mucoid and bloody. Following this, the patient was seized with a violent pain in the iliac region, and he vomited several times. With the onset of the pain, the movements of the bowels ceased abruptly. No gas was passed.

Physical examination: The patient presents the picture of an acute intestinal obstruction. There is moderate general distention of the abdomen, with marked local distention and tenderness in the left iliac region. The patient's temperature is 100, pulse 120. There is moderate leucocytosis, with no relative increase in the polymorphonuclears. After lavage and repeated enemata, the distention was greatly reduced, and some gas passed per rectum. As the symptoms were apparently improving, the operation was deferred, the patient meanwhile being kept under close observation. With the reduction of the distention a distinct mass was made out, which gave the impression of a thickened intestine. The most probable diagnosis seemed to be an obstruction from a new growth.

Operation by Dr. Lyle.

The sigmoid was found to be covered with broad, veil-like adhesions; the lower edge of this veil was tough and fibrous, and it extended from the parietal peritoneum across the sigmoid. Around this, as an axis, the sigmoid had partially revolved. The band was freed, and the sigmoid straightened out. The presence of the veil-like adhesions showed an attempt on the part of Nature to limit the greatly dilated and movable sigmoid.

PERFORATION OF A SIMPLE ULCER OF THE COLON—OPERATION.

H. H. M. Lyle, M.D.

The caput coli and the lower portion of the ascending colon can be looked upon as the stomach of the large intestine. The functions of this segment correspond both embryologically and anatomically to those of the true stomach. With these facts in mind, it is not strange that similar pathological conditions may occur in this region.

The occurrence of a simple ulcer of the colon has been known for a long time. Cruveilhier (1830-32), in speaking of gastric ulcer, wrote, "these considerations apply perfectly to the small and large intestine." Unfortunately, Cruveilhier's clinical examples were not well chosen. The discussion aroused by Cruveilhier led to the reporting of a case by Marchesseaux (in 1837) and a second by Roger (1838). Lebert (1855-61) stated that simple chronic ulcers are analogous in all points to those of the stomach; the latter occurring the more frequently, had received the most attention. In 1897, Combes reported 2 cases.

In all these observations not much attention was paid to the clinical aspect of the subject until 1902, when Quénu and Duval published a paper entitled "L'Ulcère Simple du Gros Intestin." In this monograph the authors attempted to rescue the simple ulcer from the pathological chaos of the large intestine. The paper is a clinical study of the pathogenesis, the symptoms, diagnosis, etc., of this little-known affection. It is based on personal observations and a study of the 31 cases reported in the literature. They state that the simple ulcer of the colon has all the pathological characteristics which distinguish a "round ulcer" of the stomach from other gastric ulcers; it is a simple ulcer in the group of colon ulcerations. Of the 31 reported cases, 13 occurred in the cæcum and ascending colon; in 20 cases the ulcers were multiple; 23 cases perforated.

The following case occurred in the service of Dr. C. L. Gibson, and was operated on by Dr. Lyle, October, 1909.

Surgical Number 79,130.—The patient, a married woman, 26 years old, was admitted to the hospital with a diagnosis of acute gangrenous appendicitis. The patient has been a sufferer from indigestion and chronic constipation for years. In her search for relief she has made the rounds of the clinics. The obscurity of her symptoms has led to the different diagnoses of gall stones, kidney stones, ulcer of the stomach, appendicitis, etc. Lately she has complained of a dull, aching pain in the region of McBurney's point; this pain is worse after a full meal and after exercise. Occasionally she has had attacks of alternating constipation and diarrhœa. No blood has been passed. There is no history of jaundice, typhoid, tuberculosis, or lues. She has lost considerable weight and strength.

Two weeks ago she was seized with a sharp, rumbling, colicky pain in right iliac fossa. There was considerable tenderness and distention on the right side. The patient was nauseated, but did not vomit. The pain was accompanied by a chill and a severe headache. After an hour the pain subsided, and became intermittent in character, lasting for an hour or so and then disappearing. This condition persisted for three days. Just before admission she was seized with violent cramplike pain in the right iliac fossa; she was nauseated, and vomited. The pain was accompanied by a severe chill, a sense of weakness, and intense tenderness just above the crest of the ileum. After an hour the pain subsided somewhat and became intermittent in character. Her bowels are constipated. There is an increased frequency of urination, but no burning.

Surgical condition: Woman, small frame, poorly nourished; heart and lungs normal, abdomen slightly distended, no general rigidity, no cutaneous hyperesthesia. In the right iliac fossa there is a tender mass about the size of a lemon. Vaginal and rectal examinations negative. On admission, patient had a subnormal temperature, but just before going to the operating room it rose to 100, pulse 92, respiration 26.

Blood examination: Leucocytes 25,000, polynuclear 86, lymphocytes 14.

A diagnosis of acute appendicitis, with abscess, was made, and patient sent to the operating room.

Operation by Dr. Lyle, October 23, 1909.

The abdomen was opened by an intermuscular incision over the mass; on reaching the peritoneum a large abscess containing fecal matter was encountered, the cavity was evacuated and a search for the appendix was instituted. The appendix was found without the mass and apparently had nothing to do with it. On the inner wall of abscess cavity there was a moderate-sized perforation of the ascending colon; the immediate edges of the perforation were formed of necrotic mucous membrane. The perforation was found to be in the center of an oval, indurated ulcer of the external wall of the ascending colon. The greatest length of the ulcer (2½) lay in the long axis of the gut. There was marked involvement of the surrounding lymphatic glands. The immediate edges of the ulcer were trimmed

away and saved for microscopical examination. The ulcer was then closed with a double Lembert suture as the induration precluded the use of an exulcerating purse-string suture. Contrary to expectation, the indurated fibrous tissue offered an excellent hold for the suture material and made the closure very simple. As the possibility of a lymphatic infection from the appendix could not be excluded, the appendix was removed. After inserting a rubber dam drain, the wound was closed. The patient made an uninterrupted recovery and was discharged from the hospital in 21 days. There was no fecal leakage at any time after the operation. The microscopical examination showed an edematous mucous membrane with ulceration, no evidences of carcinoma or tuberculosis, the appendix was normal.

The resemblance between this condition and that of a perforating gastric ulcer was so striking that one could almost have believed that one was dealing with a typical round ulcer. The condition in no way resembled the usual ulcerative processes encountered in this region; there were no diverticulæ or fecal concretions.

The patient disappeared from view until March, 1912, when she returned with a ventral hernia in the scar of the former operation. The hernial repair was performed by Dr. Gibson; and at this operation an excellent opportunity was offered to study the condition of the colon.

The second operation for a ventral hernia occurring in the scar of the above operation was performed by Dr. Gibson, in March, 1912. The colon appeared to be perfectly normal in all respects, and the only means of identifying the site of the old ulcer was the presence of a few membranous adhesions.

A SERIES OF CASES OF SURGERY OF THE SMALL INTESTINE.

W. Scott Schley, M.D.

The following cases of resection of the intestine, and of obstruction without resection, occurred upon the 1st Surgical Division in the service of Dr. Abbe. They are a part only of the small intestine work, and represent chiefly emergency conditions operated by the writer. There are several interesting types of obstructive conditions. The fairly numerous cases of strangulated herniæ, inguinal, femoral and umbilical, have not been included. All of these patients have recovered, all have been seen at intervals since leaving the hospital (but one), and all remain in good condition. The conditions cited below illustrate a variety of troubles, and the operative findings have been shown by schematic drawings.

Case 1.—M. F., a small boy of 12 years, was admitted March 11, 1908. Two and one-half hours before entrance, while attempting to climb a wall, he pulled down a large stone, and falling backward the rock came down upon his abdomen. He was carried home and suffered severe pain from the moment of being struck. He vomited brownish fluid resembling "blood and dirt." Urination normal after accident; no blood in urine.

On admission he was greatly shocked, pale and with cold extremities. There were contusions and abrasions of the face, but none of the abdomen, which was flat, not distended, but generally rigid, especially the upper half. Maximum point of tenderness in epigastrium just to right of median line. There was dulness in the flanks, which seemed distinctly to change with change of position. The house surgeon noted that the area of dulness seemed to have increased in the short time the patient was in the ward before operation. It was considered a case of ruptured liver or intestine. Operation: Straight incision through right rectus muscle. Abdomen found to contain a large amount of bile with considerable blood. Liver and gall-bladder and bile-passages found undamaged. Jejunum found torn completely across three inches from duodeno-jejunal junction. Ends of gut trimmed and immediately united with small Murphy button, reinforced with peritoneal stitch. Recovery uneventful; button passed before leaving hospital.

Case 2.—S. McC., a woman of 37 years, was admitted to the hospital, May 20, 1910. Fifteen years before she had had the ovaries removed, and two years later the uterus. Ten years later, following a year of constipation

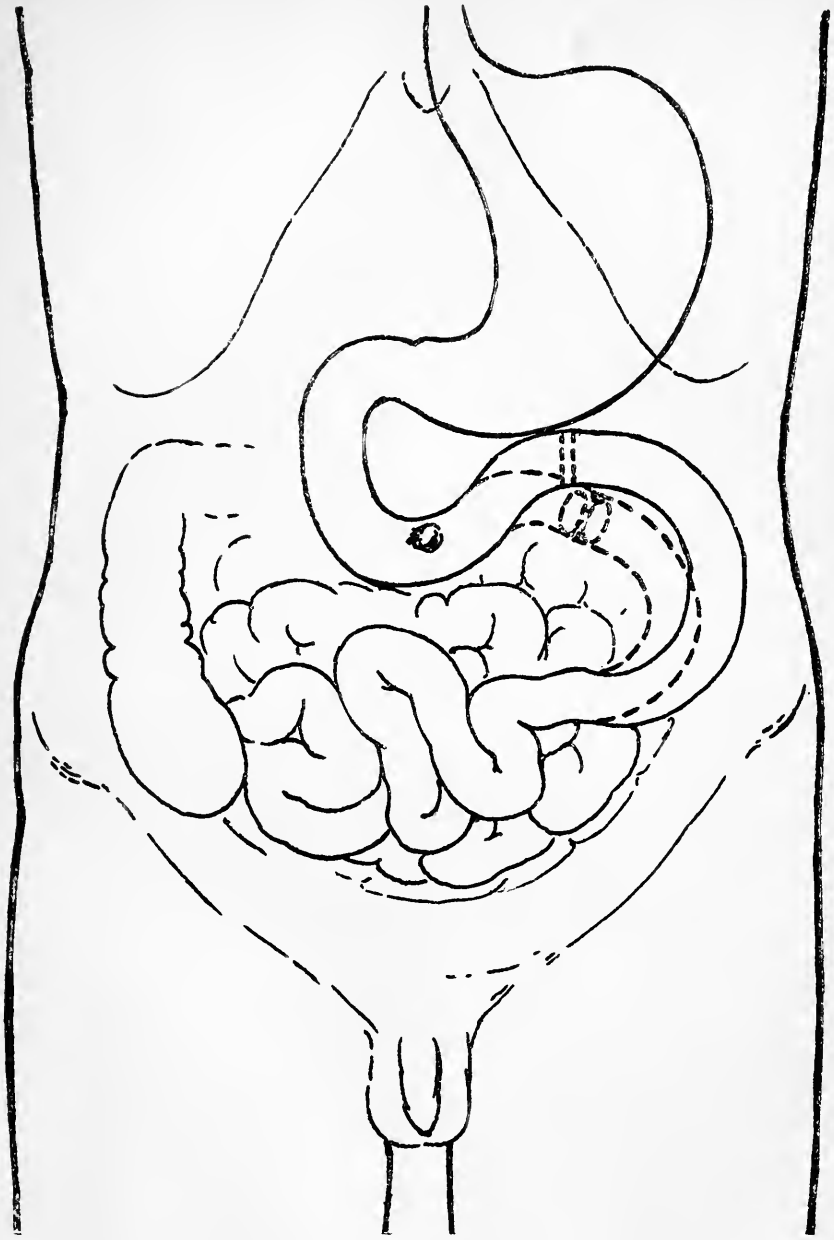

Fig. 1 (Case 1).—Rupture of jejunum at transverse double lines. Second dotted lines show continuity restored with button.

with periods of vomiting and difficult micturition, she was operated upon again and adhesions were said to be the cause of the trouble. After this operation a "lump" appeared in the scar, and grew for six months, when it was excised. It recurred, and was again removed, a year and a half ago. For the third time it has appeared and gradually increased in size. Posture, she declares, has some effect upon the size. At times it is painful.

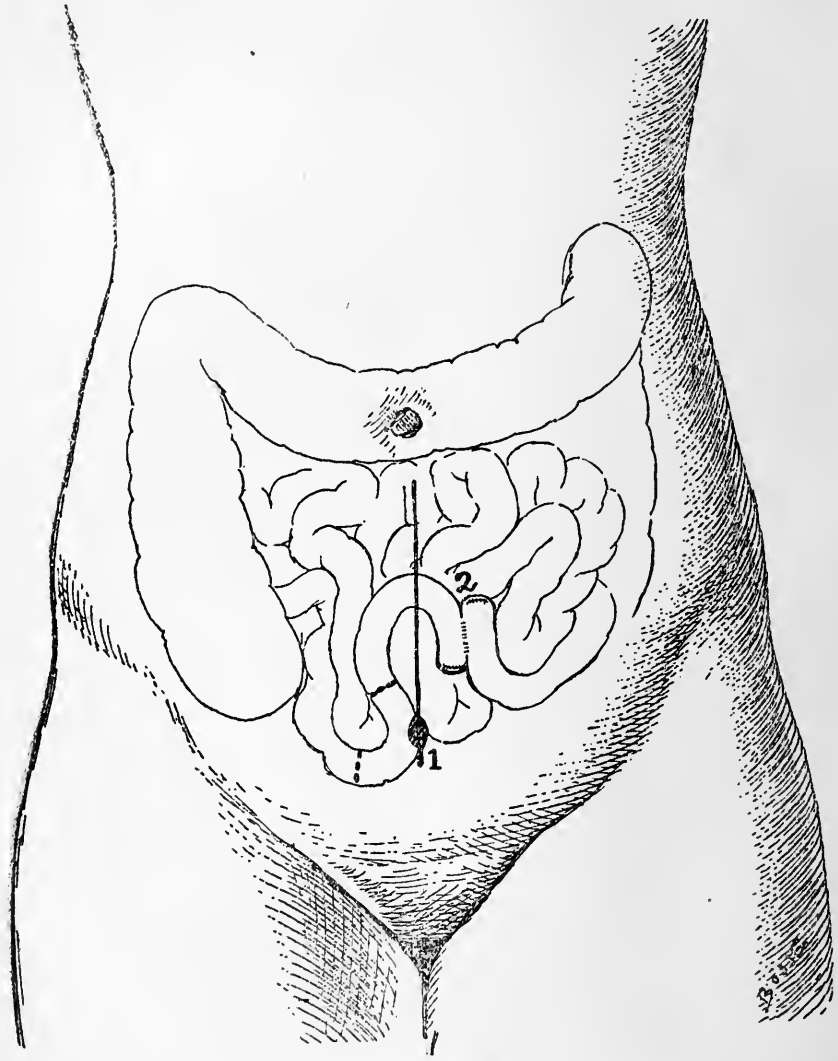

Fig. 2 (Case 2).—Point 1 shows mass in abdominal scar adherent to gut. Dotted lines, limit of resection. Point 2, lateral anastomosis.

She presented a mass the size of a golf ball in the abdominal wall near lower margin of previous laparotomy scar. The skin was involved and the center had an ulcerated area. Probe passed down the center an inch. No discharge. The mass did not appear to be tender and was attached to the tissues of the abdominal wall. Examined vaginally, no added information could be obtained.

Operation: Old scar, including growth, excised. The mass was found densely adherent to a loop of small gut. Malignancy was suggested from the history of recurrences, and it was thought wiser to resect the adherent portion of gut. Four inches of gut were excised and the ends brought together by lateral anastomosis (Fig. 2).

Microscopic examination of the tissue showed chronic inflammation only. There was no history of a fecal fistula, wound suppuration, nor was there an old stitch. The muscular tissues were not invaded.

Case 3.—F. F., a woman of 40 years, was admitted to the hospital November 9, 1910. This patient came seeking relief for a large ventral hernia, resulting from an operation performed two years before for ovarian tumor.

She presented a long scar to the right side of the mid-line, broad and very thin. Skin and thinned-out scar tissue, to which the gut was densely adherent and through which the convolutions were visible and palpable, alone formed the abdominal wall at that point.

Operation: Old scar excised. Even with the greatest care a loop of the very thin-walled gut, densely adherent to the cicatrix, was opened. The remainder of the adherent intestine was separated with difficulty, often leaving a mass of scar tissue on the bowel wall. The opened knuckle of gut was excised and the ends brought together by lateral anastomosis. Recovery was uneventful; highest temperature following operation, 100 1-5°.

Case 4.—E. H., a woman 42 years of age, entered the hospital April, 1910. She had a discharging small intestine fistula in a scar in the mid-line of the abdomen. In November, 1909, I had operated in the country upon this patient for intestinal obstruction of the most urgent sort. She was then seven months pregnant and had been taken six days before with the acute pain, vomiting and abdominal cramps of that condition. When seen, her condition was desperate, and a hasty operation resulted in freeing a loop of ileum from a band just below the pelvic brim on the right side. From the length of time the gut had been shut off, it was gangrenous at the point of constriction. Three inches were excised and the ends joined with Murphy button reinforced with peritoneal stitch. During convalescence, two weeks later, the wound opened and discharge from the small gut took place. The button could be felt in the gut and was removed by the attending physician through the wound. Several months later she came to the city for the closure of the intestinal fistula. At this operation it was found that the button had passed down several feet from the original site of resection and had lodged in an angle of bowel that had become attached to the median incision. It had then ulcerated its way through. The intestine was freed, again resected and the ends closed by end to end suture. This patient has been seen recently and is in perfect health.

Case 5.—M. D., a small girl of six years, was admitted December 7, 1909. She had been taken sick one week before with abdominal pain and vomiting. From the onset of pain, the vomiting had been frequent and irrespective of attempts to take nourishment. Bowels said to have moved well day after beginning of attack and two days before entrance. No blood or unusual

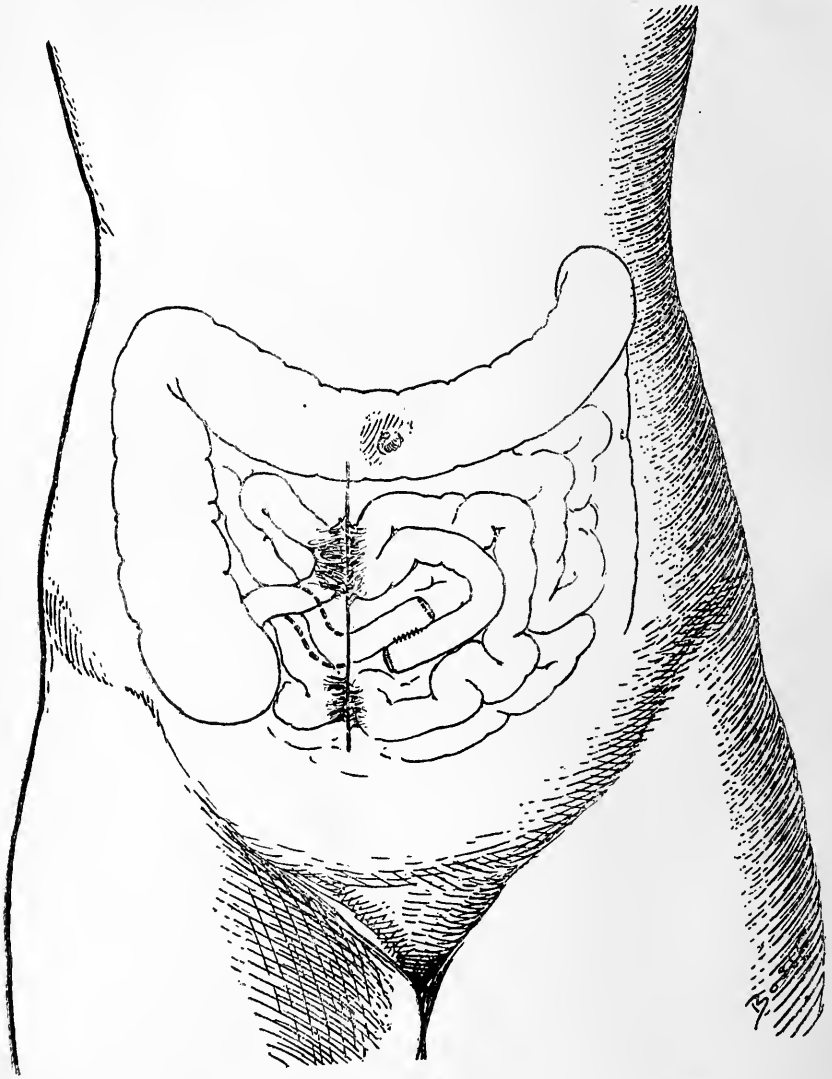

Fig. 3 (Case 3).—Intestine densely adherent along whole extent of abdominal cicatrix. Portion of gut resected with cicatrix and united by lateral anastomosis.

conditions were noted by the parents. The abdomen was not rigid, moderately distended and with general tenderness. Signs of fluid within the peritoneum. Right rectus more rigid than left. Rather more tenderness over right lower quadrant and with greater muscular spasm. A mass occu-

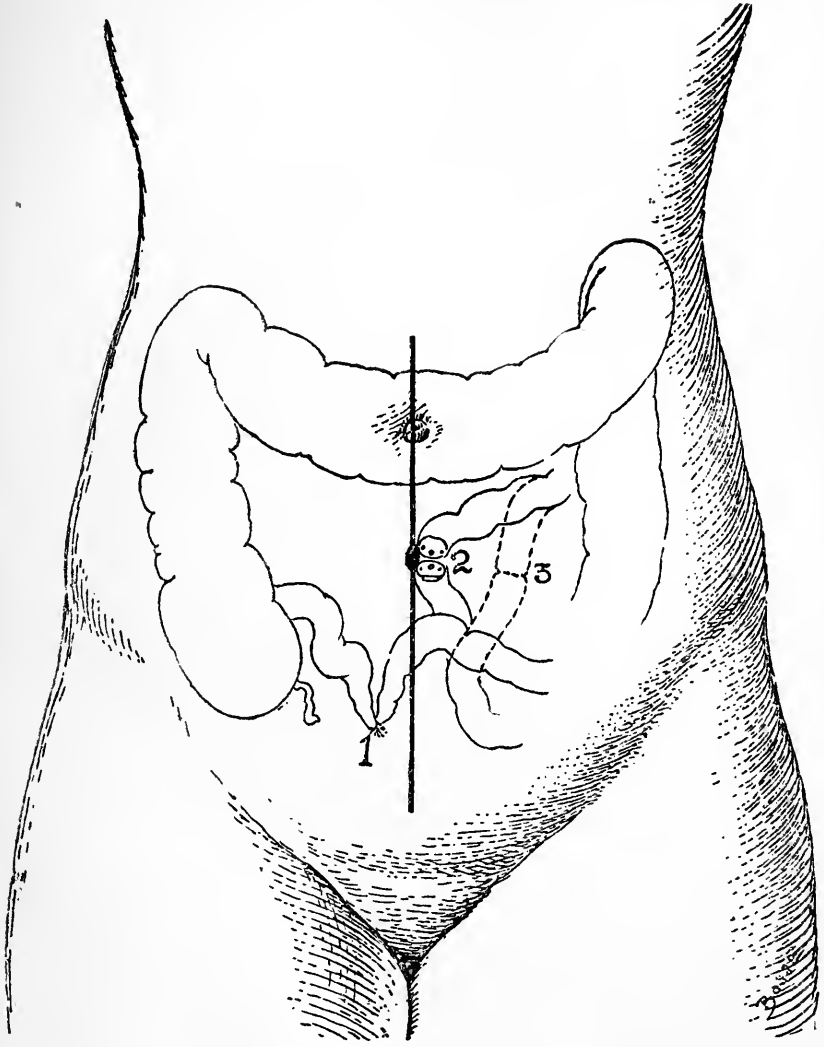

Fig. 4 (Case 4).—Point 1, site of original obstruction. Point 2 (should have been descending colon), where button ulcerated through gut and established a fistula. Point 3, where colon was resected and joined by end-to-end anastomosis with suture.

pied the region of the caput and extended for four or five inches along the line of the ascending colon. Rectal examination revealed nothing.

Operation: Intermuscular incision over caput extended by opening the rectus sheath. Condition found to be ileo-colic intussusception with gangrene of the small gut. It was so rotten that it was difficult to reduce. The mesentery was black with thrombosed vessels. Over 22 inches of intestine were excised and the small gut anastomosed with the caput at the ileo-cæcal junction by button reinforced with peritoneal stitch. Button passed on 7th day. Convalescence stormy and prolonged. Child now in excellent health.

Case 6.—G. K., a man, 42 years of age, was admitted first to the Medical Service of Dr. Janeway, Dec. 23d, 1910. An abdominal condition of gravity was certain, but an exact diagnosis could not be made. He had been taken sick 24 hours before entrance and several hours after a meal, with a sudden sharp pain across the upper abdomen. This pain was continuous and frequently radiated to the lower abdomen in a stab-like manner. He vomited once several hours after the beginning of the attack. Bowels have not moved since the attack, nor has he passed flatus. Blood count and differential count both high. Examination revealed only a moderately distended abdomen with general rigidity. Tenderness to pressure was not marked and seemed somewhat greater over the upper half. Some fluid accumulation. He had the appearance of suffering and of one acutely ill. Transferred to Surgical Division.

Operation: Median incision below umbilicus. Large amount of blood-stained serum. No odor. Intestine moderately distended and with slight vascular engorgement. Twenty-two inches of bowel were found black red from occlusion of mesenteric veins. On section, the arteries of the mesentery bled freely, but the veins were thrombosed. The diseased gut and liberal healthy margins were removed. Ends joined by button reinforced with peritoneal stitch.

This patient did well, but failed to pass the button before leaving the hospital. He returned a month ago for another condition, and the radiograph showed that he had passed it in the meantime.

Case 7.—E. S., a man of 27 years, was admitted April 11, 1911. He had been operated upon five months before at the hospital for an acute appendicitis with abscess. He was drained for some time and made a good recovery. After being home for some weeks, he began to have occasional attacks of colicky pain associated with a sluggish condition of the bowels. The morning of his admission to the hospital, for the second time he had been taken with a sudden and very severe pain about the umbilical region accompanied with nausea and vomiting. On entrance his appearance was typical of intestinal obstruction, and the demand for interference immediate.

Operation: The abdomen was opened in the median line.

The adhesions in the right iliac region were very dense and also on the right side of the pelvis. In the greatly distended condition of the small gut and the mass of adhesions, the particular point of obstruction could not be found. An enterostomy was done, taking as low a point in the ileum as possible. After drainage of the bowel for several weeks, his condition was

so greatly improved that the operation for relief of the cause of the obstruction could be undertaken with more leisure and deliberation. The area of the colostomy wound containing the gut was excised and the intestine lightly clamped off. With considerable difficulty, the small gut was freed from adhesions and bands throughout and traced down to the cæcum and the large gut from that point to the rectum. On account of the damaged condition of the peritoneal coat from the old inflammatory process, the anastomosis was done by invagination (see Fig. 8), after the method described last year by

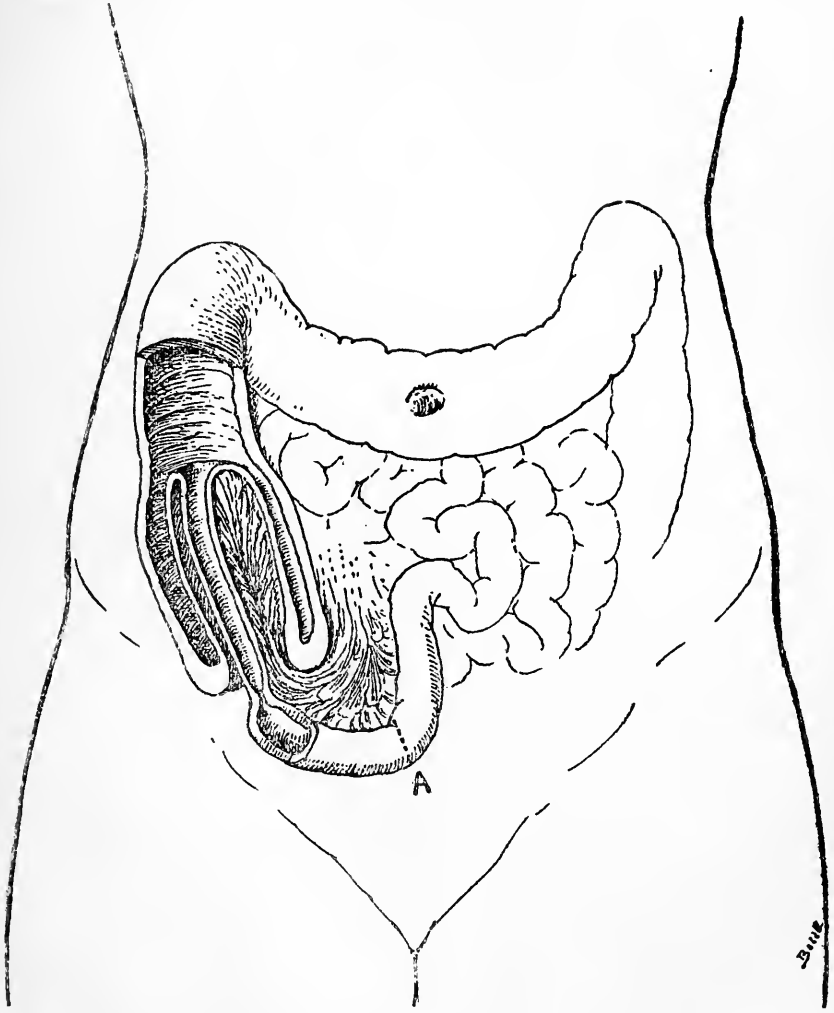

Fig. 5 (Case 5).—Gangrenous intussusception. Twenty-two inches of ileum resected.

Dr. Gibson in the report, rather than by the more usual one of end to end or lateral anastomosis. This man has been seen within a fortnight and is well.

Case 8.—M. H., a woman 22 years of age, was admitted January 6, 1911. She had been operated upon a year before for an appendicitis with abscess

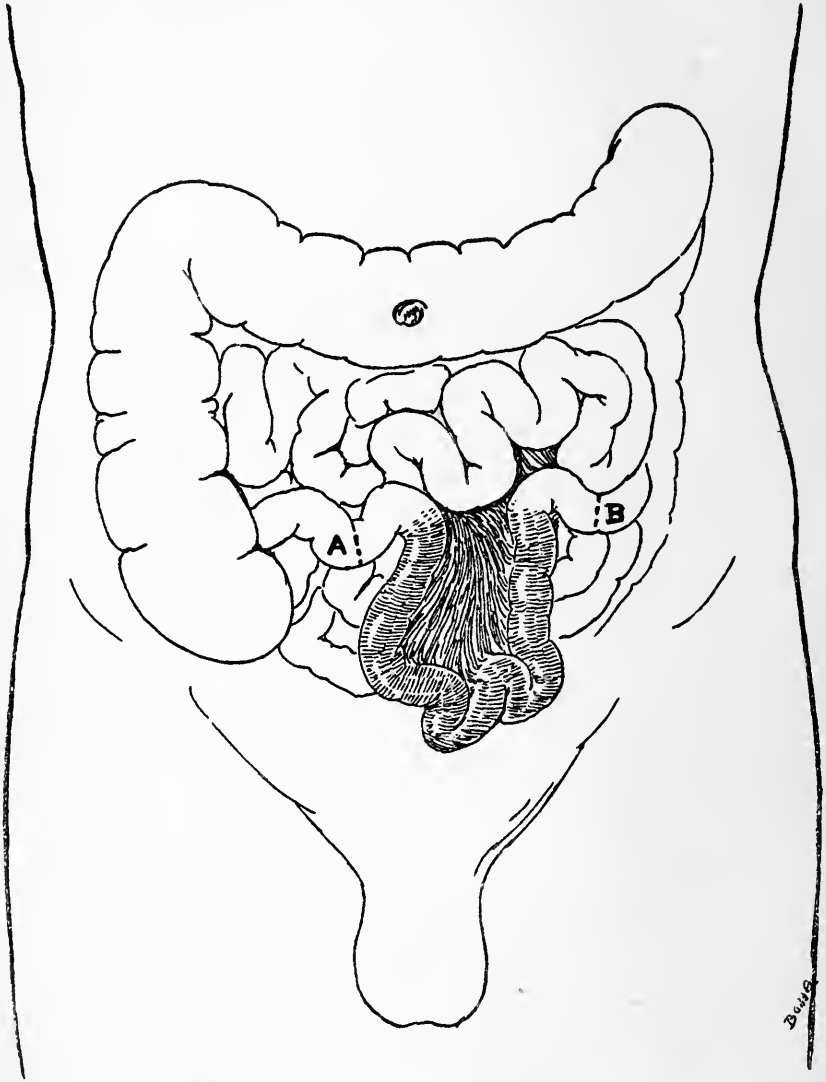

Fig. 6 (Case 6).—Mesenteric thrombosis, venous closure. Resection of nearly two feet of gut.

and peritonitis at another hospital. Since that time she has been troubled greatly with constipation, getting worse. She has had severe abdominal pains accompanied with marked constipation at fairly frequent intervals. For a week before entrance her bowels had not moved. Three days before admission she was taken with unusually severe pain accompanied with persistent vomiting. Her distention was great, the vomitus foul and general condition bad.

Operation: A dense mass of adhesions occupied the pelvis and the right lower quadrant of the abdomen. The exact site of occlusion could not be determined in the time allowed for a safe conclusion of the operation. As in the former case, an enterostomy was considered preferable to an immediate anastomosis. A loop of ileum two feet from the caput was brought into the median wound, and as in the last case, a rubber drain tube inserted proximally. After several weeks of clearing out and drainage, an attempt was made to separate adhesions and find the point of occlusion. The density and extent of the matting together of the bowel made separation impossible, and the only recourse left, to empty the small gut into the large, was accomplished by a direct implantation of the proximal end into the transverse colon, the nearest available large gut free from adhesions. The distal end was likewise implanted that there might be no excluded or occluded intestine. It was impossible to resect this distal part on account of the iron-clad nature of the adhesions, and at the time no other disposition seemed possible. This patient was seen four months after her operation and declared herself well and comfortable. She had gained greatly in weight and appeared in perfect health.

Case 9.—Intestinal Obstruction from Enterolith: J. K., a man 72 years of age, was admitted March 2, 1908. Four days before entering the hospital, he was attacked with a dull grinding pain across the upper part of the abdomen. He took cathartics without result, nor would enemas relieve him. Vomiting occurred two days later, and on admission was of distinctly feculent character. It is of interest to note that the patient, a physician and an intelligent man, asserts that he had no trouble of any sort with his bowels prior to this attack. Two years before he had passed a number of gall-stones, the size of distal joint of index finger, and which had facets. Before their passage he had attacks of gall-stone colic.

Operation: Incision through the right rectus disclosed a collapsed large gut with a distended small gut. On working back from the ileo-cæcal junction a large, smooth, dark mass was found distending the lower part of the jejunum. It could not be moved up or down. An incision opposite the mesentery released a stone the size of a small hen's egg. There was no ulceration of the mucosa of the intestine; there were no diverticula in which the stone could have pocketed. The convalescence of this elderly patient was uninterrupted.

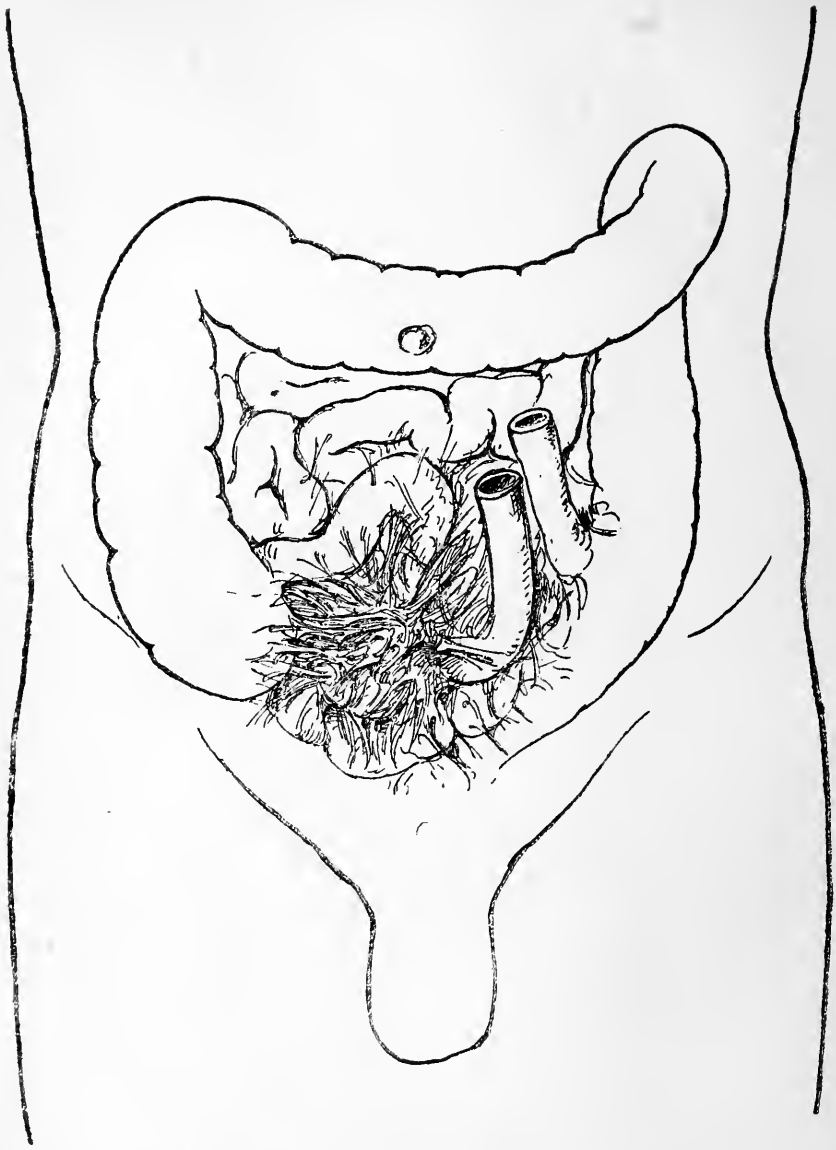

Fig. 7 (Case 7).—Intestinal obstruction following suppurative appendicitis. Numerous and dense adhesions. Enterostomy followed later by freeing adhesions, use of sterile oil and anastomosis by invagination (see Fig. 8).

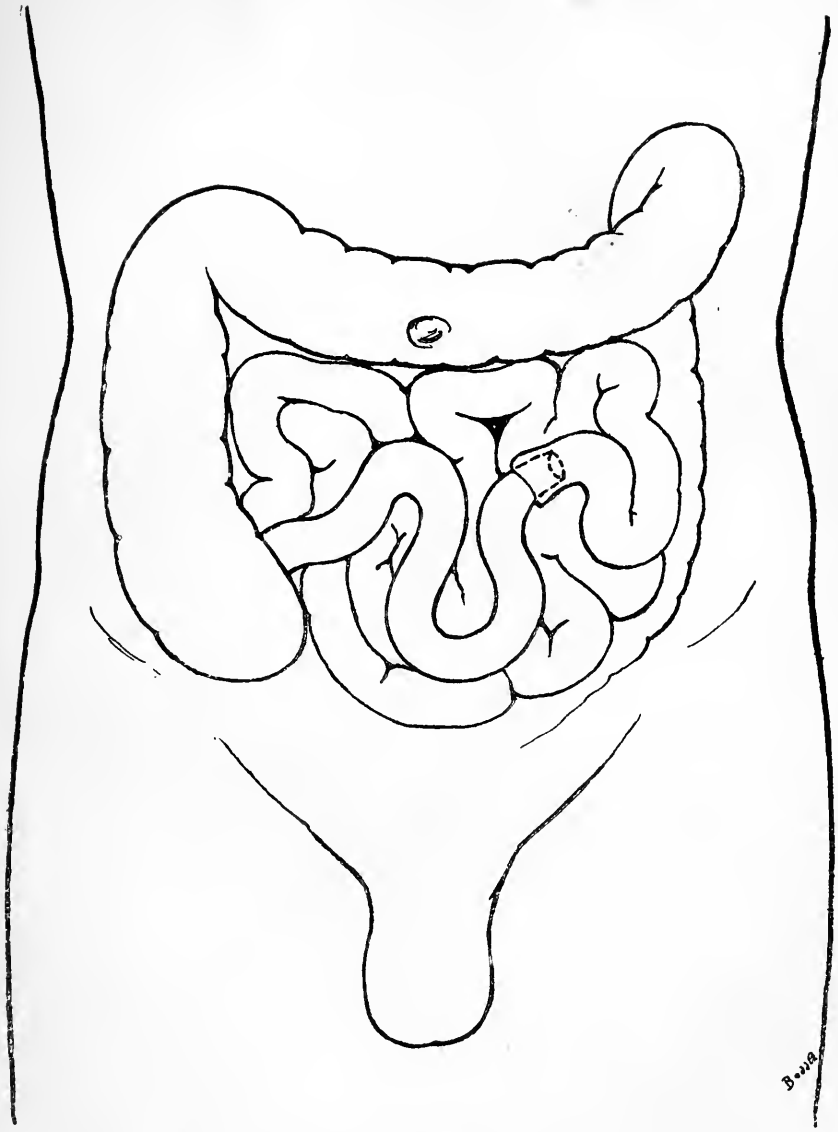

Fig. 8.—Anastomosis in case 7, by invagination.

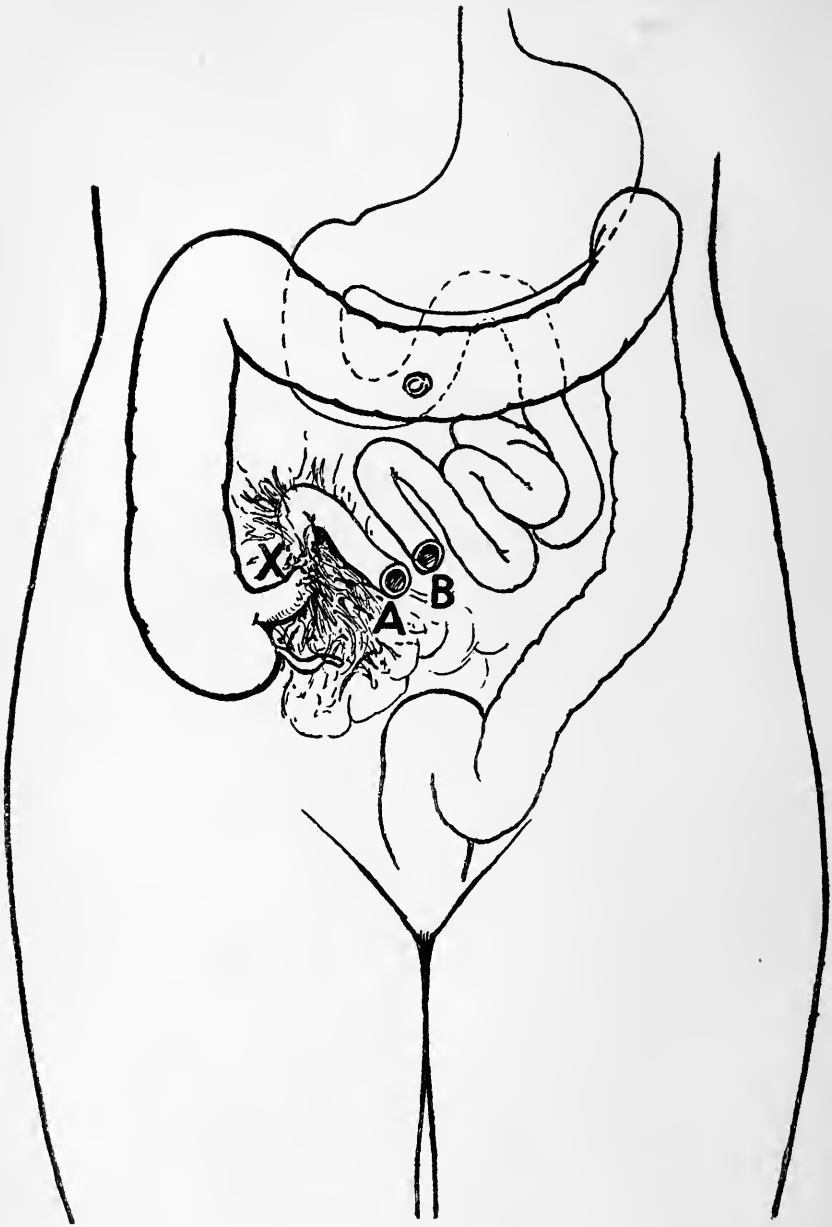

Fig. 9 (Case 8).—Intestinal obstruction following suppurative appendicitis. Dense adhesions, enterostomy, later implantation into colon (see Fig. 10).

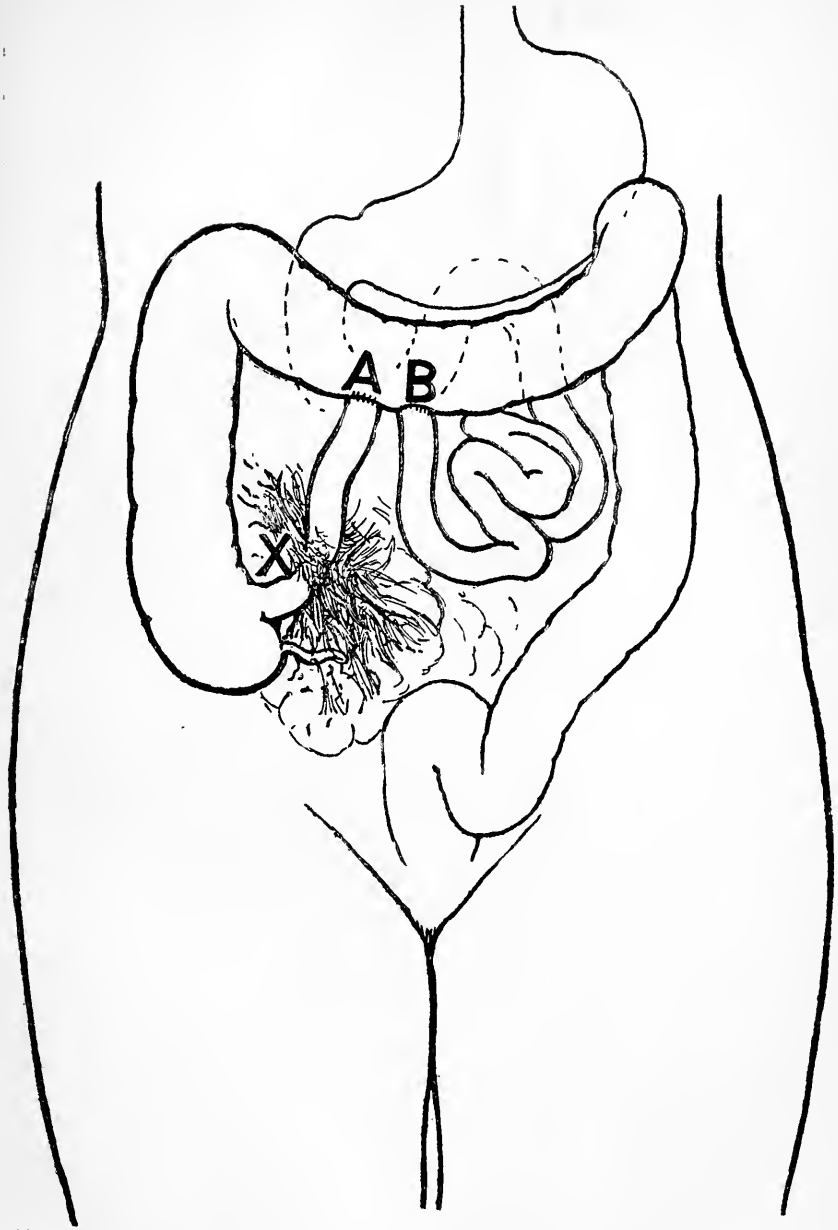

Fig. 10 (Case 8).—Impossible to separate adhesions. Both distal end and proximal implanted into nearest free colon (transverse).

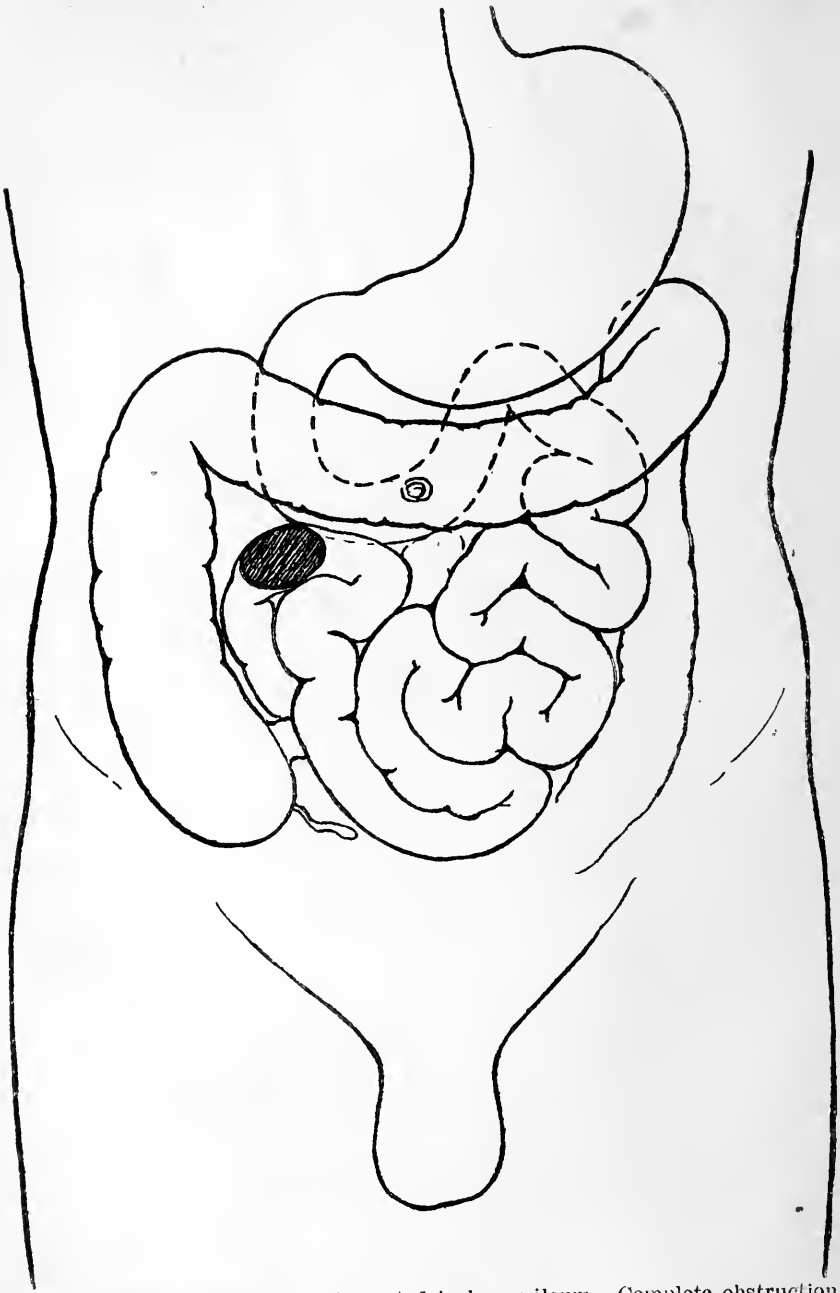

Fig. 11 (Case 9).—Enterolith impacted in lower ileum. Complete obstruction.

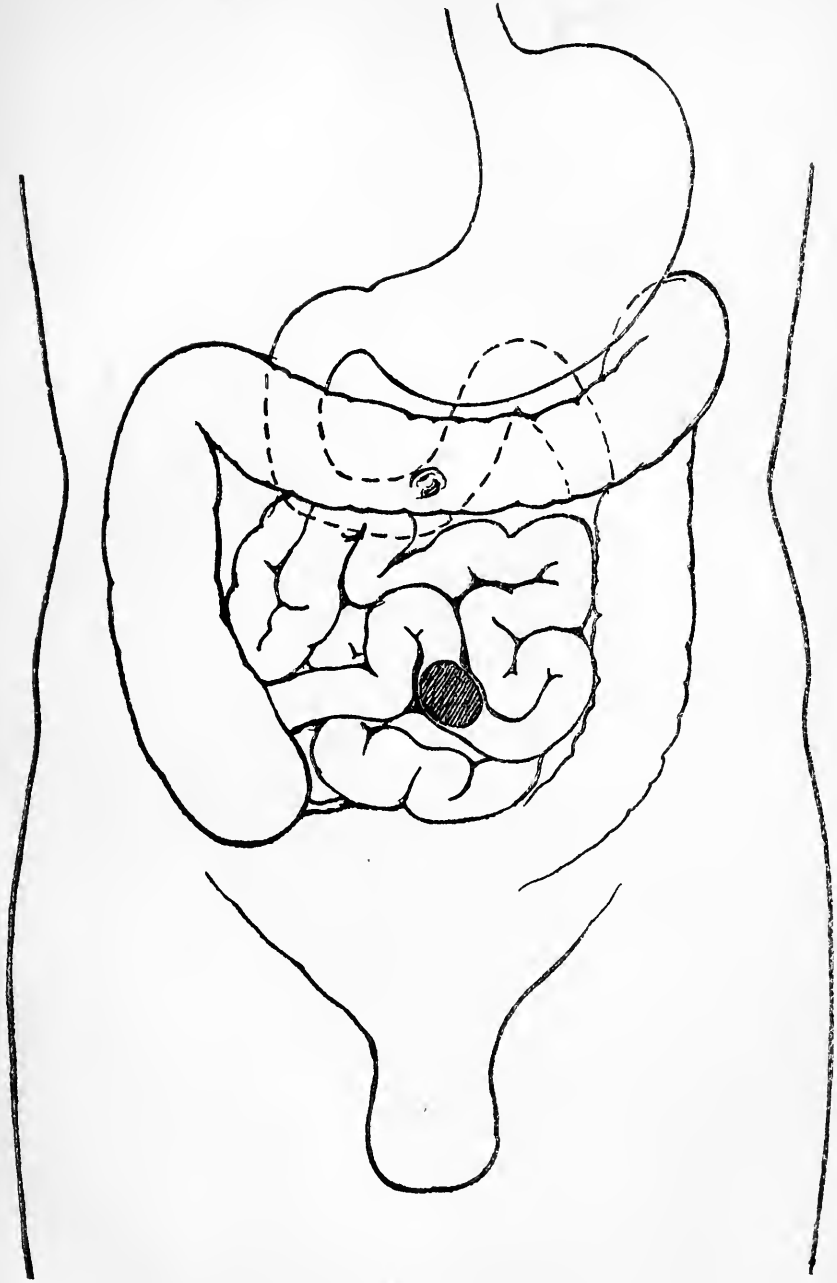

Fig. 12 (Case 10).—Enterolith impacted in lower ileum. Complete obstruction.

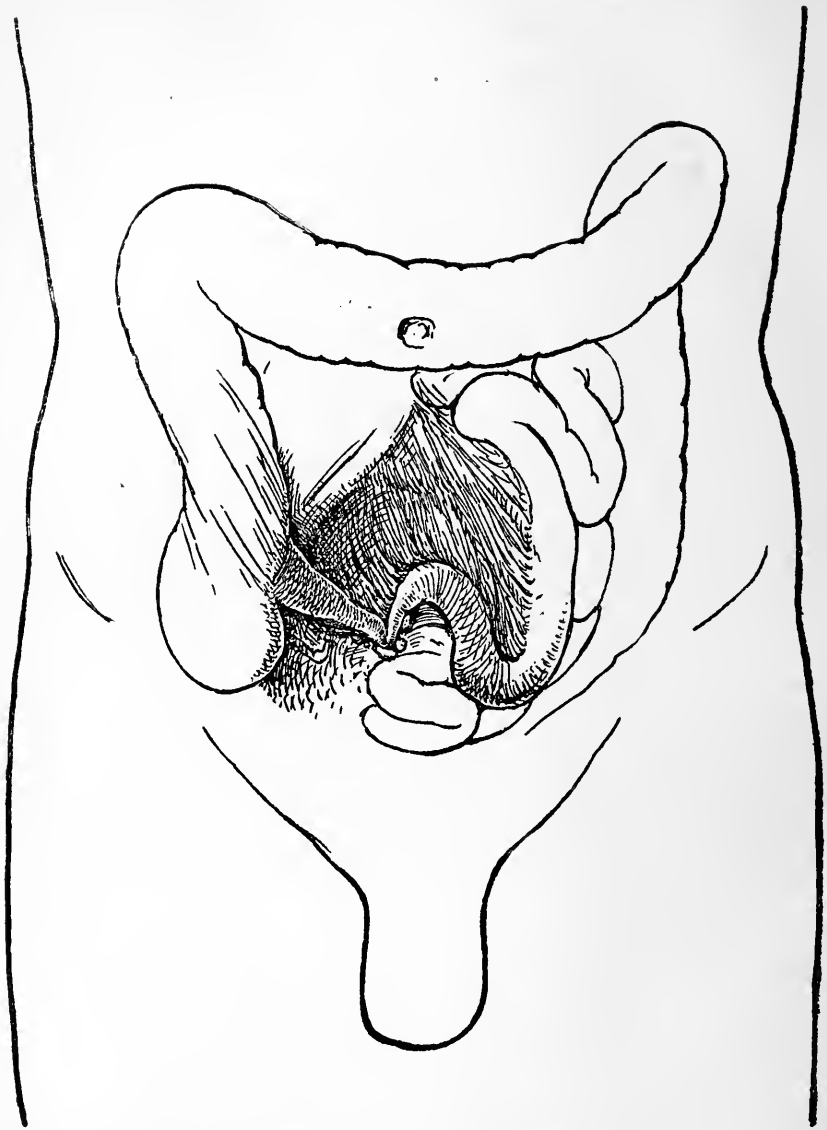

Fig. 13 (Case 11).—Acute obstruction caused by angulated ileum adherent to caseous mesenteric gland.

Case 10.—Intestinal Obstruction from Enterolith: A. H., a woman of 60 years, was admitted to the hospital April 5, 1908. She had been taken with nausea and vomiting five days prior to her entrance. The vomiting was continuous, and she could not retain food or medicine on her stomach. First food, then bile, then feculent material came up. Vomiting gave relief. This patient says that she has never been jaundiced and that her bowels have always been regular. Her general health has always been good. She has had no serious illnesses in the past. She was very ill on entrance with greatly distended abdomen and dry tongue.

Operation: Median incision. Large intestine collapsed and small distended. On working back from the caput coli, a dark mass the size of a pullet's egg was found distending the gut about 18 inches from the ileo-cæcal valve. The intestinal wall was very thin and distended, but it was possible to push the stone up to a higher level where the thinning was not so marked, and remove it through an enterotomy at that point where repair by suture would be easier. As in the former case, there was no ulceration of the intestinal mucosa, nor were there diverticula to be seen. Convalescence here also was most happy. Both of these cases have been followed, and they are apparently in excellent health to-day.

Case 11.—I. T., a small boy in his 9th year, was admitted April 24, 1907. He had been well the earlier years of his life, but for some time before present illness had had occasional abdominal cramps. He was taken three days before admission with severe cramps in the abdomen and vomiting. Bowels moved slightly during the interval before admission. The boy appeared to be a well-nourished child. The abdomen was greatly distended and very tender over the whole surface. Peristalsis could be easily seen.

Operation: Median incision, umbilicus to pubes. Large gut collapsed. Small gut distended and deeply injected. The cause of the obstruction was found to be an adhesion about one-quarter of an inch in diameter, extending from an enlarged old lymphatic gland, near the foot of the mesentery, to the ileum two inches from the caput coli. The obstruction had been caused by the angulation and constriction. The gut was viable and its release was accomplished by cutting the band. This patient made a rapid recovery and has been seen several times since leaving the hospital.

SIMPLIFIED EQUIPMENT AND MANAGEMENT FOR THE OPERATING ROOM.*

W. Scott Schley, M.D.

The natural tendency in matters relating to surgical operative equipment is rather towards elaboration and addition, as new facts are discovered and newer requirements develop in the steady march of progress. The endeavor to have every added essential and accessory immediately to hand is the most fruitful source of complication and elaboration, requiring a very constant attention and study to eliminate and simplify, as the burden of paraphernalia becomes unnecessarily irksome, time-consuming and possibly wasteful.

In operative work a proper economy of time and labor should not be lost sight of, especially when it results in greater good to the patient, greater surety and ease, and a greater saving of materials. These things can best be accomplished by a safe and quick method of handling gauzes, instruments and solutions, the ready accessibility of everything needed, and excellent lighting, making it possible for sterile individuals to conduct the entire work rapidly and without danger of rendering themselves or the material unsterile at any stage.

There is nearly always something in other plants and equipments of actual or suggestive value that well repays time spent in study and observation, for comparison with and often the elucidation of problems at home. From time to time additions and changes in the operative equipment at St. Luke's Hospital, New York City, have been made whenever greater ease and surety of work and a greater saving of time, materials and general labor to all could be gained. These objects have never been lost sight of, and to-day the equipment stands, I believe, second to none in simplicity and working efficiency. The constant endeavor of those interested in these things has been towards elimination and simplification of existing material and its concentration, rather than the addition of more: the perfection of essentials and rejection of unessentials.

*Previously published.

Fig. 1.—Showing instrument sterilizer open. High-pressure steam pipes under the flooring, and entering the sterilizer from below.

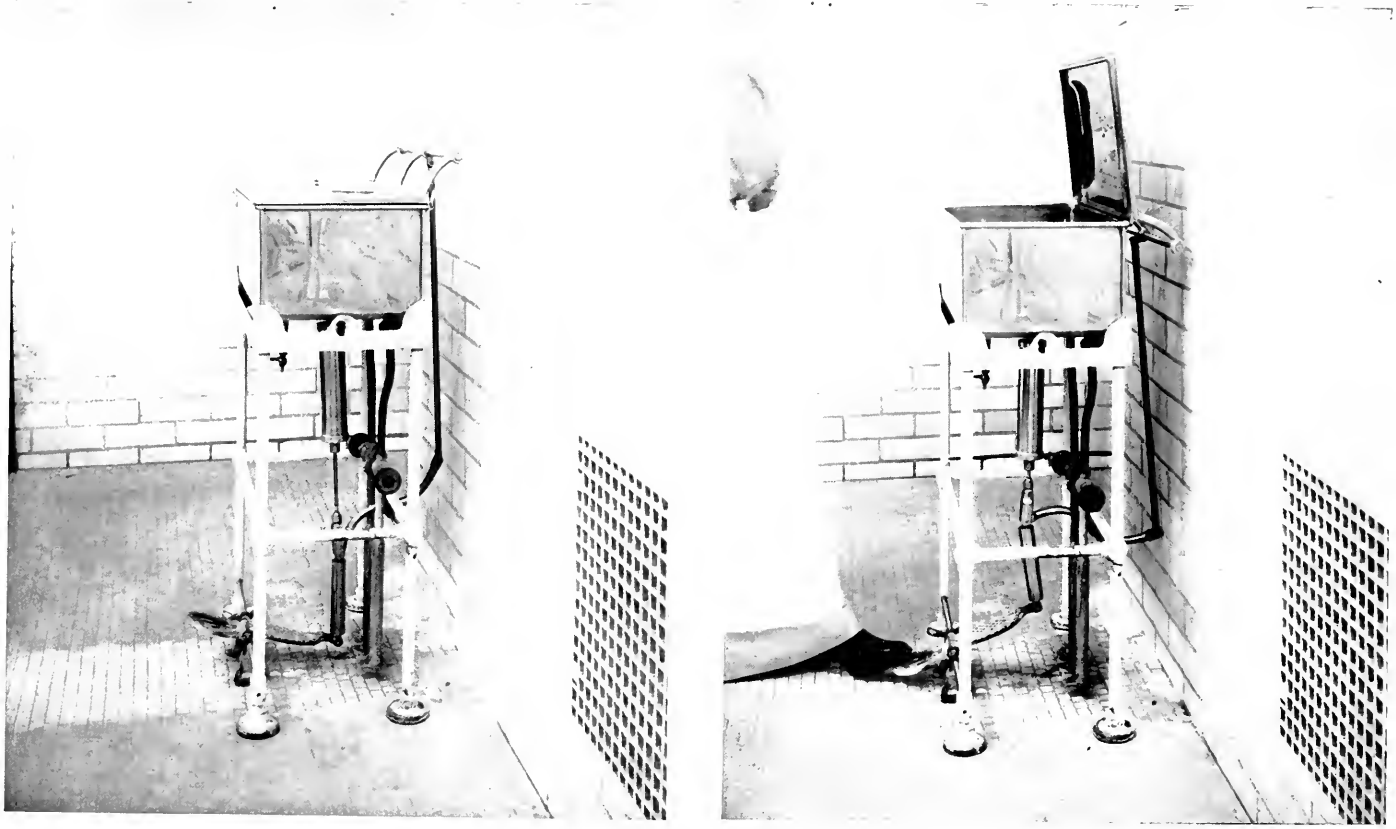

Fig. 2.—Instrument sterilizer. Side view, showing air-check cylinder and cover-elevating mechanism.

The general requirements, which have become recognized through study, observation and experience, will first be mentioned, and then spoken of more in detail.

The operating room should be of fair size and self-contained, the necessary equipment being within the room itself or in its walls, to the saving of many steps and the unnecessary passing of assistants and nurses in and out during the progress of operative work. The furniture can be so reduced and simplified that nothing but the operating table, instrument table, canisters for holding gauzes and draperies and an electric towel heater occupy the free floor space; hot and cold water sterilizer or its taps, instrument and utensil sterilizers and the few solution bottles being arranged along or even within recesses in the walls. Instrument cases are best built in the walls with glass doors flush with the wall, eliminating movable and obstructing furniture and dust accumulations. If two operating rooms adjoin, they can be accessible from either side. An excellent example of this was seen at Sonnenburg's Clinic at the Moabit Hospital, Berlin. Special solutions, anesthetics, hypodermatic solutions, etc., are best arranged similarly. Instrument and utensil sterilizers should be actuated by high pressure steam coil and should be opened by foot pedal, enabling sterile assistants or nurses to operate them without hand contamination or the necessity of calling others to aid. Stock solution bottles (saline, alcohol and sublimate), and the hot and cold sterile water taps should be also arranged for foot release. It is possible by such means to cut down the personnel of the operating staff, and especially to avoid the intermediary handling and exposure of gauzes, drapery and instruments in transit from the sterilizer to the operating table.

Natural lighting, as long recognized, should be from one side of the room as well as from above. Artificial lighting is best accomplished by means of the newer indoor enclosed electric arc, both for general illumination and for direct lighting above the table.

A means that will provide perfect protection for gauzes and drapery and yet allow of instant accessibility was found in Europe and brought to a high state of perfection by von Eiselsburg, of Vienna, in the canister container system. Gauzes are sterilized in closed metal canisters and their air ports closed. They are then ready at any time to be brought in and placed on the stands where the cover in a few seconds is connected with the cover-elevating device worked by foot lever. They are always handy, their contents are instantly accessible

and entirely protected and they can be renewed on their stands, when empty, in a few seconds as cartridges in a gun. Experience has shown to date no quicker and safer way of handling gauzes, towels and drapery. Gauzes may be taken out in small amounts at a time in anticipation of their need, by a nurse or assistant, and with the gloved sterile hand or forceps. These containers were introduced in St. Luke's Hospital in 1905 by Dr. H. H. M. Lyle, have amply proven their worth and are used upon both surgical divisions.

The instrument sterilizer, in addition to being placed in the operating room, should not be too far from the instrument passer's table. It should be a semi-automatic affair, opening by foot lever and closing noiselessly when the pressure of the foot is released. A high pressure steam coil in the bottom will boil water more quickly than will gas. The instruments should be placed in and sterilized in trays, and passed from them to the operating table. The present instrument was worked out by the author while abroad, and later made in this country by one of the large manufacturing firms. This apparatus has been placed in the amphitheater of the hospital and has worked with efficiency for over three years. It is heated by steam coil from the high pressure service of the operating and sterilizing rooms, and will boil warm water in 3 minutes. It is placed upon the instrument passer's side and but 8 feet from his table, so that he is enabled, unassisted, to boil instruments between and during operations, without crossing the path of any one, quickly and without contamination of hands or instruments. This sterilizer is placed against the wall and does not occupy the free floor space. The steam is under complete control and the inlet valve can be turned by foot.

The instruments are placed in shallow copper trays that fit the sterilizer and are put in one over the other. A slightly inturned edge prevents telescoping and perforated bottoms allow the water to drain off on lifting them out.

Through forethought in construction the amphitheater was provided with hot and cold sterile running water, the tanks being behind the scenes, out of the way, and the taps leading through the partition to within a few feet of the operating table. A utensil sterilizer also actuated by high pressure steam and operated by foot pedal, has been added and placed to the rear, where it is accessible and allows the few basins and irrigators used to be boiled between or during operations as necessary.

The operating table is simple and provides the different positions

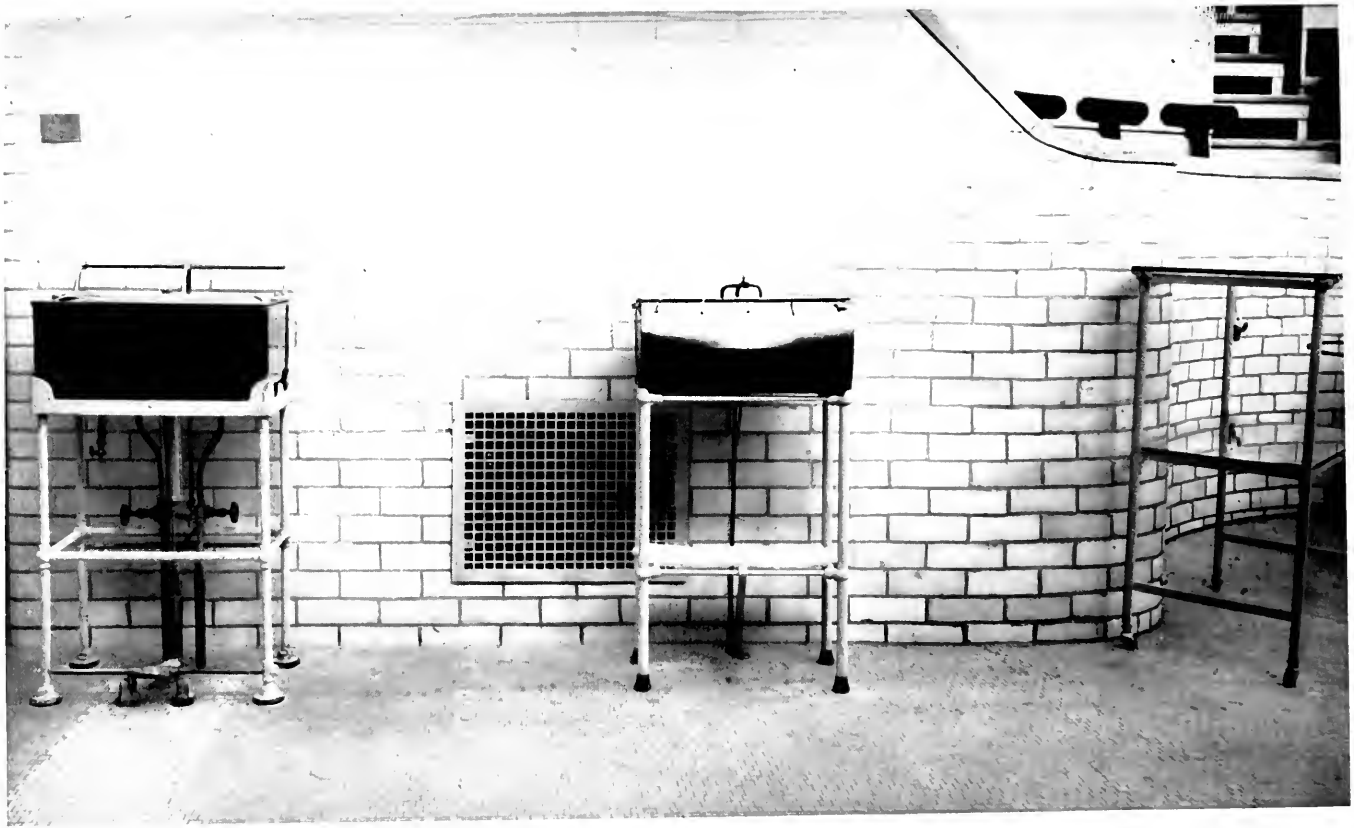

Fig. 3.—Showing the relative positions of instrument sterilizer and instrument table. Box for dry sterile rubber gloves in center (canister container now used).

Fig. 4.—Copper canisters for gauzes and draperies. Canister stand with movable balance weight for cover.

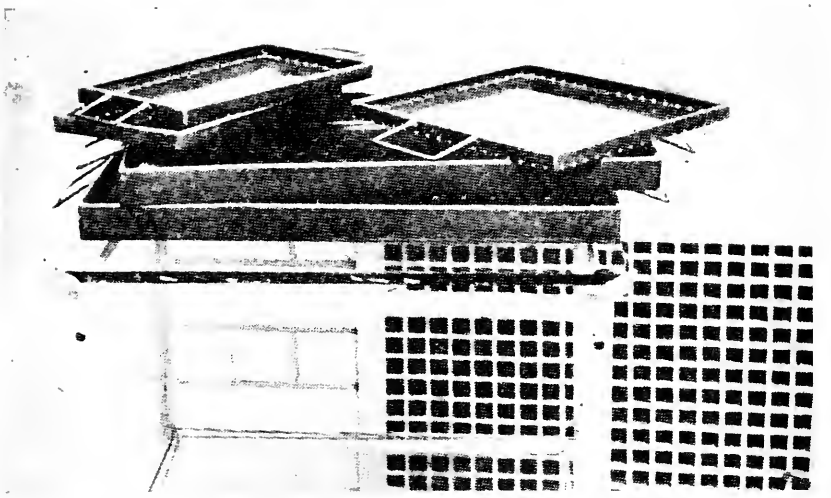

Fig. 5.—Instrument trays. The larger ones have perforated bottoms, and the smaller, for finer instruments, are perforated at the edges.

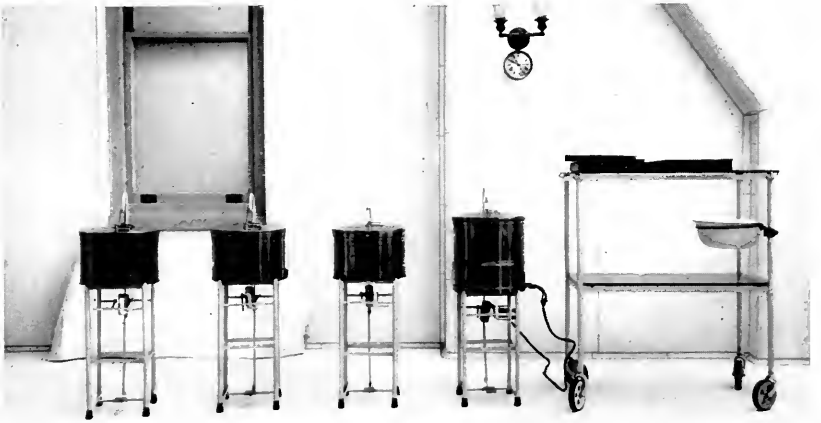

Fig. 6.—A battery of canister containers, with towel heater and instrument table.

quickly. The instrument table is a simple glass or metal top affair with one shelf below and with a basin bracket on one leg. A table 40 x 20 inches and 4 feet in height is sufficiently large for general work. These two articles of furniture need no elaboration beyond the possible addition of a removable stout wire bracket attached to one leg to hold a basin of saline solution.

All solutions except the 10 per cent saline, 70 per cent alcohol and 1-8 bichlorid of mercury, have been banished from the operating room, and these are contained in large stock bottles whose outpour is regulated by foot pedal release, and which are placed against the wall upon the nurse's side, about 8 feet from the operating table and instantly accessible. The proper amount of saline solution is run into the small basin or irrigator and sterile water of the desired temperature is added from the wall taps. Saline or other solutions can be made up in this way and reach the operating table in 10 seconds without hand soiling. Irrigating stands and large irrigators are not used; a 4-quart enameled metal container with 4 feet of tubing answers all purposes, can be easily sterilized and is held by hand when needed.

For artificial illumination the superiority of the electric arc over the incandescent bulb, both for general and direct light, I believe is fully apparent when once tried. It gives infinitely more and better quality of light. Some of the better equipped European clinics have adopted the system with excellent results. Abroad the lights are sometimes arranged with upward reflection, which is the best plan for general illumination where the walls do not exceed 12 feet in height. In the amphitheater at the hospital, where greater height had to be dealt with, it was necessary to find a lamp of suitable downward reflection and diffusion. Such a lamp it was my fortune to find in the newer indoor enclosed arc with small opalescent globe and thin white porcelain reflector and diffuser above. This lamp is seen in Figure 7, where two of these for general illumination take the places of batteries of incandescent globes. After some experimentation with the lights and shadows of the arc lamp, I devised the apparatus shown herewith for 2 lights, with common reflector arranged 4½ feet between carbons, that will illuminate the whole table at once in addition to the field of operation.

The small opalescent globe softens and diffuses the 1,200 or more candle-power and with the superior regulating device of this lamp upon a good circuit and with soft core carbons it is almost absolutely

without flicker. These arc lights are arranged as in incandescent lighting, and use the same current (taking the 110 or 220 volt direct or alternating, and from 3.5 to 5.5 amperes, according to requirement). They are best arranged upon different circuits to avoid the possible chance of simultaneous extinguishment should anything happen to one circuit.

For miniature lamps, head lights, cystoscopes, motors, etc., several ordinary wall taps have been placed within a few feet of the operating table and electrical connection can be instantly made by the ordinary push plug.

More and more it is found that elaborate equipment for general operative work, whether of elaborate tables or special instrument stands, is not required. In addition to the solution bottles upon the nurse's side, there is only a small glass wall-shelf for the few sterile basins and the irrigator.

Upon the First Division the patients are fully prepared in the ward and are given only a light alcohol (70 per cent) rub on the table as a final preparation after removal of the light sterile protective dressing. Elaborate and excessive drapery has been abolished and a maximum of 2 sheets, over rubbers, and 4 towels answer for the majority of cases.

In preparing such a room for work but one unsterile person is necessary to bring in the 3 canisters and towel heater, place them on the stands and connect the covers with the elevating device. The instrument passer, before washing up, places the instruments in the trays and puts them in the sterilizer. One nurse, before washing up, places the few basins and the irrigator in the utensil sterilizer. The operator, assistants and other nurses are in the meantime washing up and dressing, and the patient is being anesthetized. Hot and cold sterile water and the few solutions are always ready. The instrument passer, after washing up, covers the top and shelf of the instrument table with a few sterile towels from a canister, and removes his trays and instruments from boiler to the table. A nurse, upon the nurse's side, after washing up, covers the nurse's shelf with towels and the small solution basins and irrigator are placed thereon. The unsterile orderly or assistant places the suture and ligature containers on the lower shelf of the instrument table and the instrument passer takes his suture and ligature material, catgut, silk on spools and any other material needed for one or two cases and places it in the folds of a sterile towel or two. Any suture or ligature material left over, even

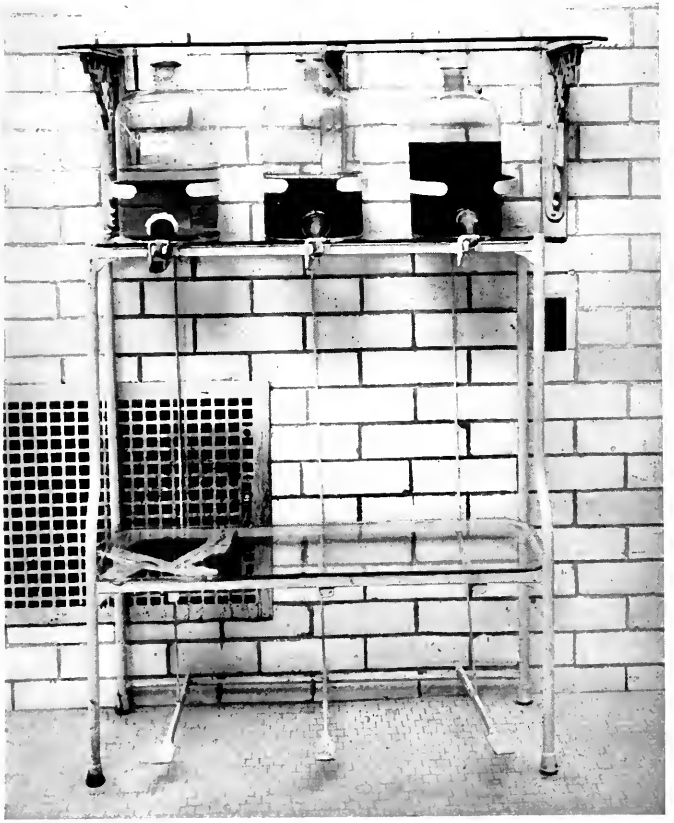

Fig. 7.—Type of stand for stock solution bottles, having foot release.

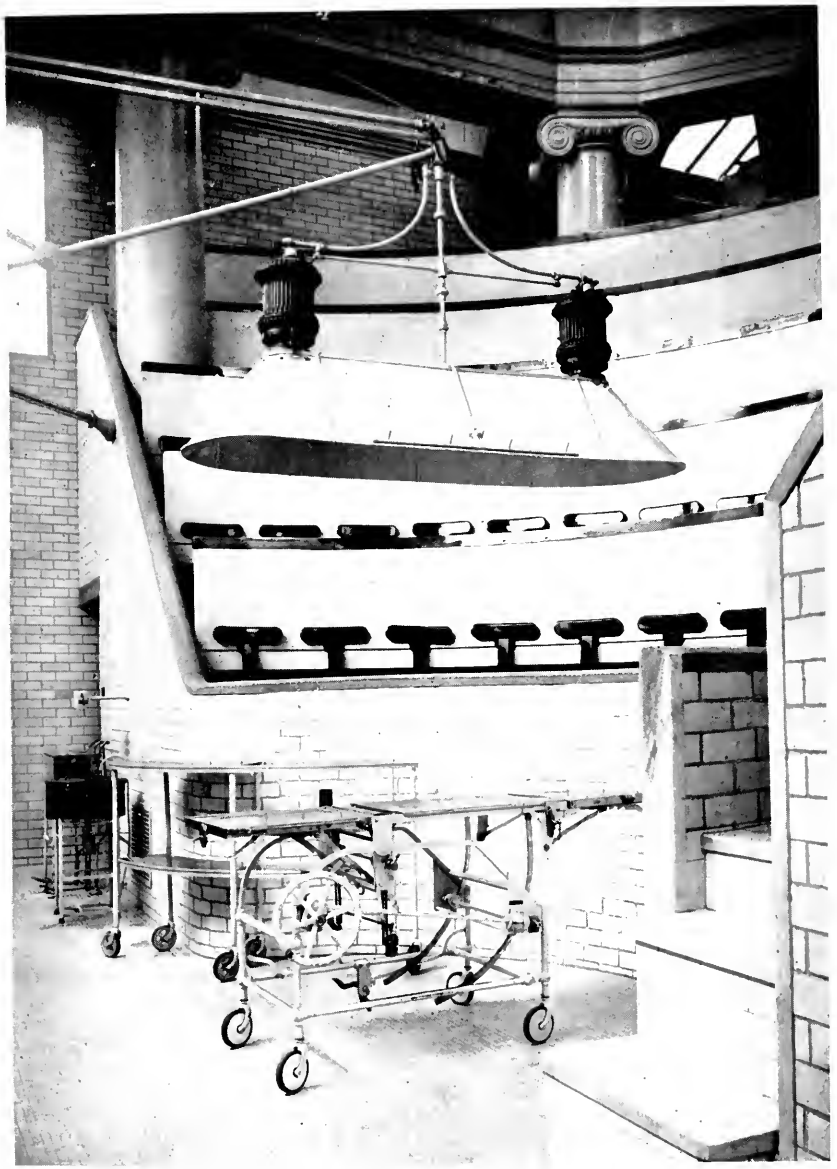

Fig. 8.—Two lamps with common reflector, 4 feet 6 inches between carbons. Apparatus over operating table. Almost complete elimination of shadows is produced.

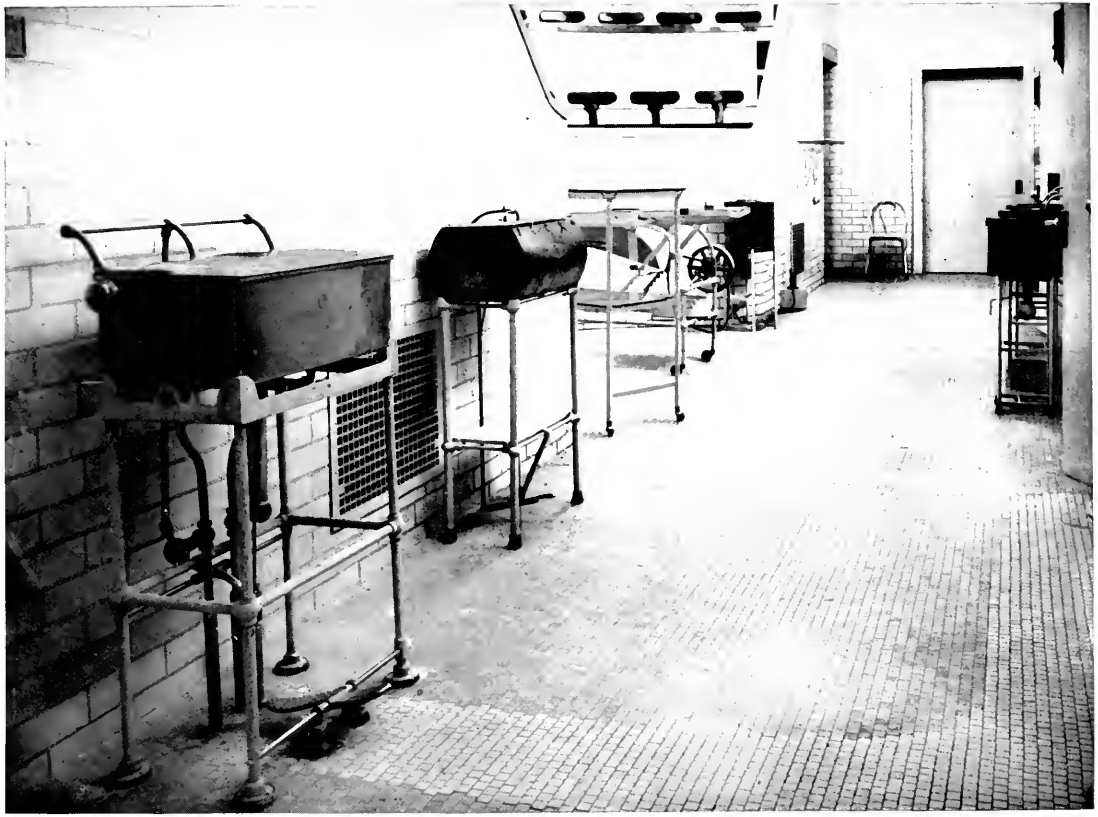

Fig. 9.—Showing general arrangement of the operating room. Solution bottles (not in place) stand under the small wall shelf seen in background. Door leads to etherizing room. Utensil sterilizer and sterile water taps are to the extreme right and behind the foremost canister.

if contaminated, including the iodine catgut, can be easily and quickly resterilized for subsequent days. Anything needed, not anticipated in advance, is immediately accessible.

A perfect division of labor with simplification of apparatus allows quicker handling of cases as well as celerity and ease in the preparation of the room, and a diminished number of helpers, if preferred. All sterilized and necessary materials are accessible to sterile hands and but one unsterile assistant is required to handle unsterile material, to clean up between cases and do the heavier work. Dry sterile rubber gloves are worn by all and are always accessible near the instrument sterilizer in a container opening by foot pedal.

Such are the main features and outline of the scheme it has been the endeavor to instal and perfect at St. Luke's.

The system works out to include in its personnel the undergraduate nurses of the hospital, so necessary in the American plan of educating and preparing them for future surgical usefulness in their private and institutional work and in rounding out their education. Slight modification of duties of the different members of the operating staff are therefore undertaken from time to time.

The suggestions for some of the equipment were gathered at a number of the clinics of the world, some of the most valuable from the rooms of Eiselsburg, of Vienna; Kocher, of Bern; Sonnenburg, of Berlin. Thought and experience have added from time to time new features to a constant betterment, and through the liberal and progressive spirit of the Board of Managers and Superintendent these changes have been quickly consummated.

Because many have been interested in this equipment, and have adopted these ideas, and because of the inquiries received, it has seemed of sufficient general interest and importance to merit this brief article which, leaving the description of mechanical details to photographs, is meant to be suggestive rather than complete and exhaustive.

The canisters were made by one of the larger instrument firms and have already been adopted in several hospitals, and recently for naval use. The instrument sterilizer was made by another of the larger firms and is now manufactured in various forms as regular equipment.

EXTRUSION OF MEDULLARY BONE SPLINT.

W. Scott Schley, M.D.

J. S., a man of 45 years of age, was admitted to the 1st Surgical Division May 27, 1911.

Three years before he had suffered a compound fracture of the left femur in the middle third. He was taken to a hospital, where an operation was finally done, and after 7 months, he left with the wound completely healed. Four months before he entered St. Luke's, he noticed a swelling on the anterior aspect of the left thigh. This broke down, opened, and left a small, discharging wound. He suffered from the suppurating process and had temperature and malaise. Two weeks before coming to the hospital, another swelling appeared on the outer side of the leg in the old scar. This was incised by his physician. Both sinuses led to bare bone at the same point on the shaft. The left leg showed 3 inches shortening.

Operation.—Incision through old scar on the outer side of leg down to the bone showed a hard white object projecting from the shaft (see Fig. 1). It was thought to be a sequestrum, at first sight. A better view showed that it was a bone tube, and by cutting about the shaft and rotating the tube, it could be withdrawn.

The splint had not been absorbed, and had even preserved its surface polish. It had finally acted as a foreign body and had been partly expelled. It is of interest in connection with the operative treatment of fractures.

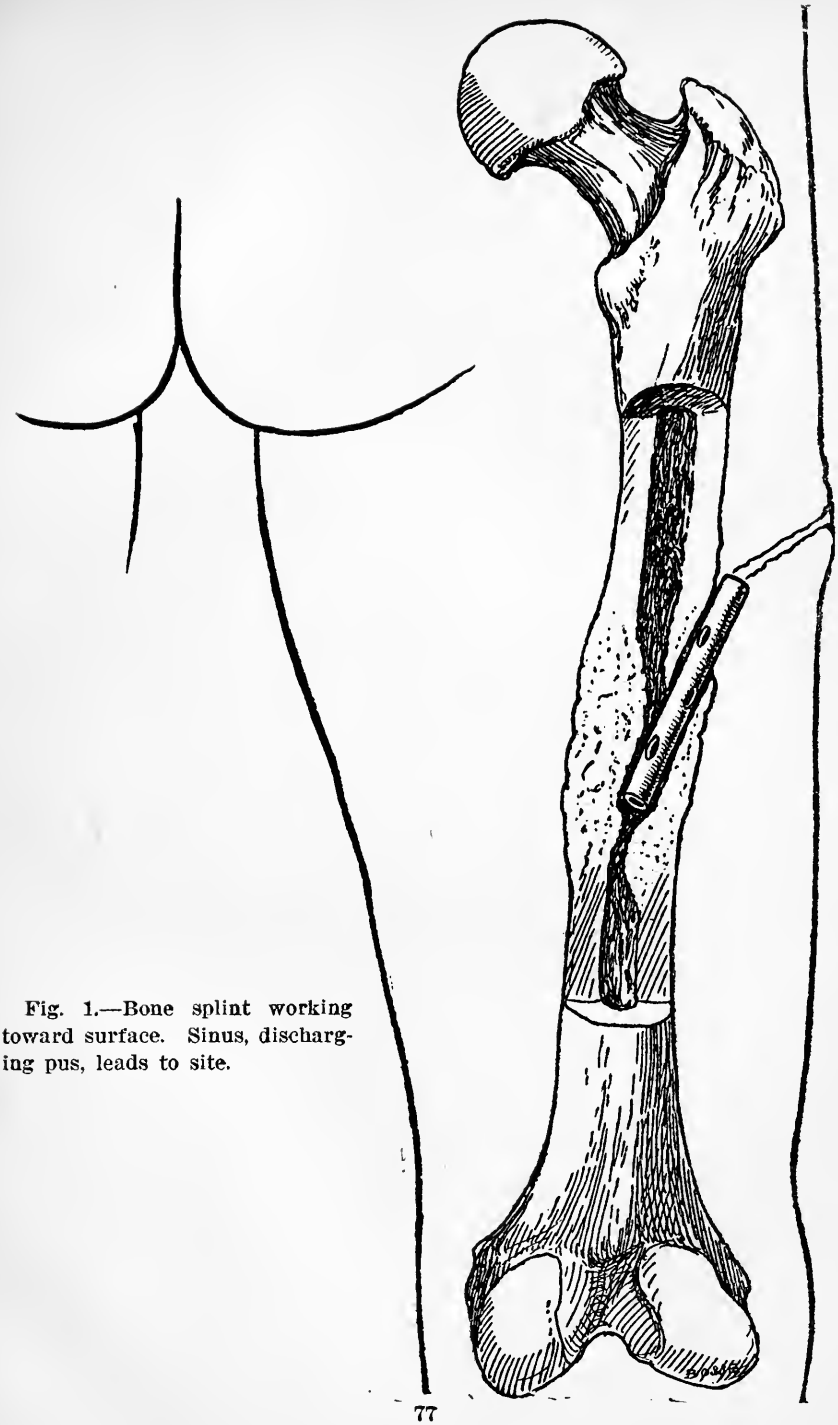

Fig. 1.—Bone splint working toward surface. Sinus, discharging pus, leads to site.

TWO CASES OF STONE IN THE URETER.

W. Scott Schley, M.D.

Among the cases of kidney and ureter troubles the past year, upon the 1st Surgical Division, there have been two of special interest; one, because of the great severity of the subjective symptoms with the minimum amount of damage, and the other because of the almost total absence of subjective symptoms with great and extensive damage to the kidney and ureter.

Case I.—J. O. B., male. Admitted June 21. Discharged July 23 last. A year prior to entrance he was taken with an exceedingly severe pain in the left flank. This pain extended down the left side into the testicle and down the left thigh, and was characteristic in its sharp and cutting character. This condition was intermittent, but he was never free enough from it to return to work for over 2 months. There have been recurring attacks up to the present time. On entrance, he had been having pain more or less continuously for 2 weeks. He is said to have passed blood in the urine from time to time. He was a well-built and nourished young man, with slight rigidity on the left side of the abdomen and left flank without great tenderness.

Cystoscopy and catherization of the ureters, as well as a radiograph, demonstrated a calculus in the lower part of the left ureter 3½ to 4 inches from the bladder. Little or no urine from affected side. Examination for blood on entrance, neg.

Operation (Dr. Schley).—Transperitoneal ureterotomy. Abdomen opened with 6-inch incision through the left rectus. Stone could be felt in the ureter. Field of operation padded off and the peritoneum incised, when ureter could be drawn up and opened (See Figs. 1 and 2). Stone removed and small bougie passed to bladder and pelvis of kidney. Ureter closed with fine interrupted silk stitches. Peritoneum over ureter closed, leaving a very fine cigarette drain just through. Abdominal wound closed in usual way by layer suture.

The convalescence in this case was absolutely uneventful and rapid.

Case II.—M. Z., female. Admitted April 4, 1911. Discharged May 10, 1911. For the last 2 years the patient has had a dragging sensation in the right side of the abdomen, but hardly amounting to pain. It has never been sufficiently bad to prevent her following her occupation as cook. There is no history whatever suggestive of attacks of renal colic and none positive of renal derangement.

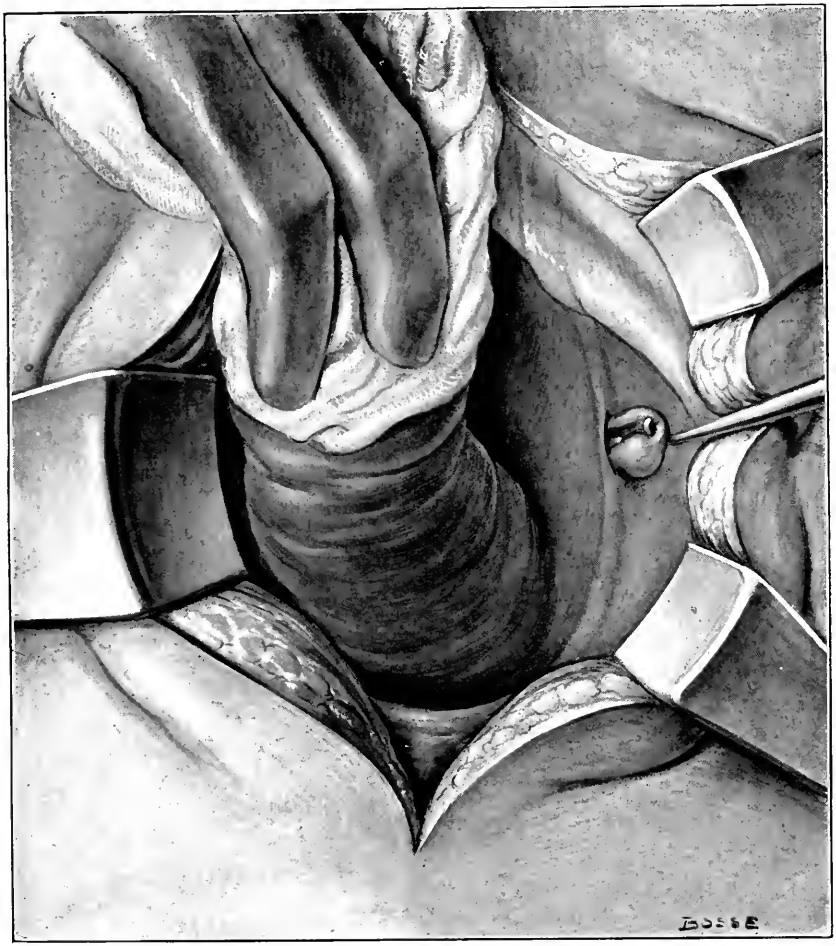

Fig. 1.—Peritoneum opened. Ureter, containing calculus, drawn out.

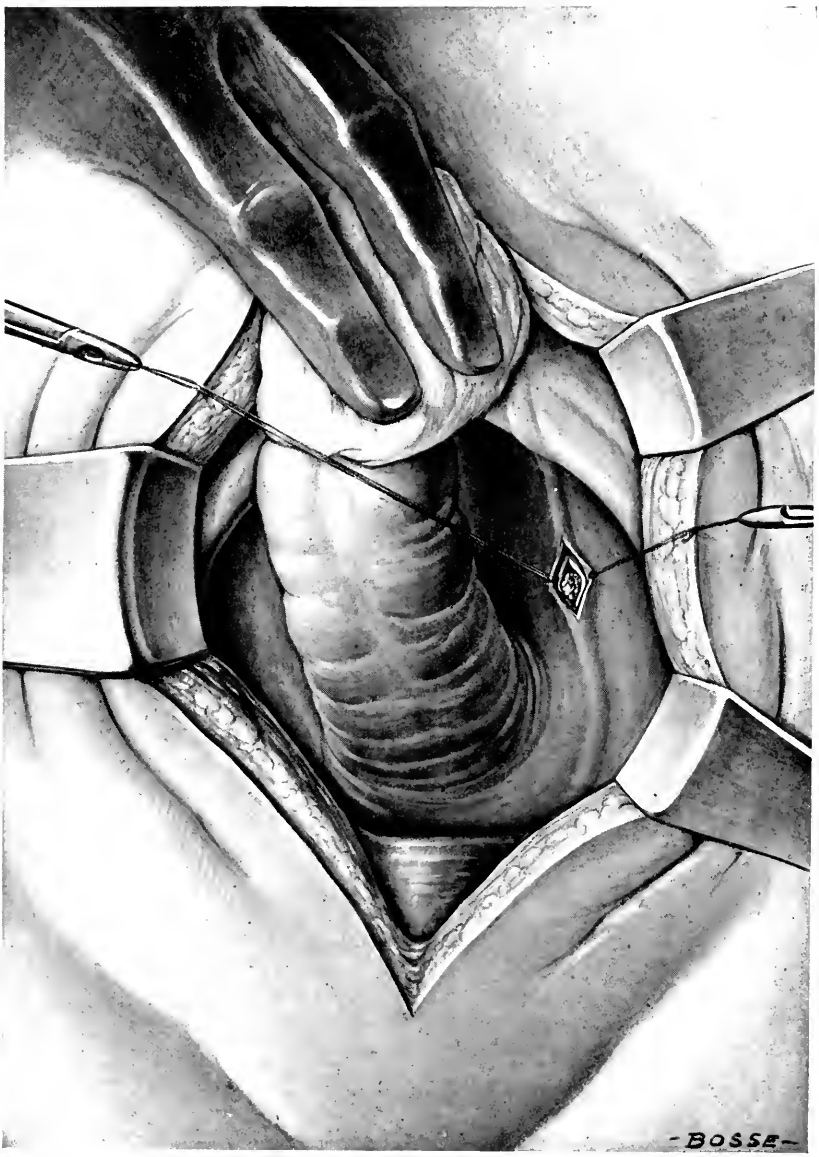

Fig. 2.—Peritoneum and ureter opened. Calculus tightly wedged in place.

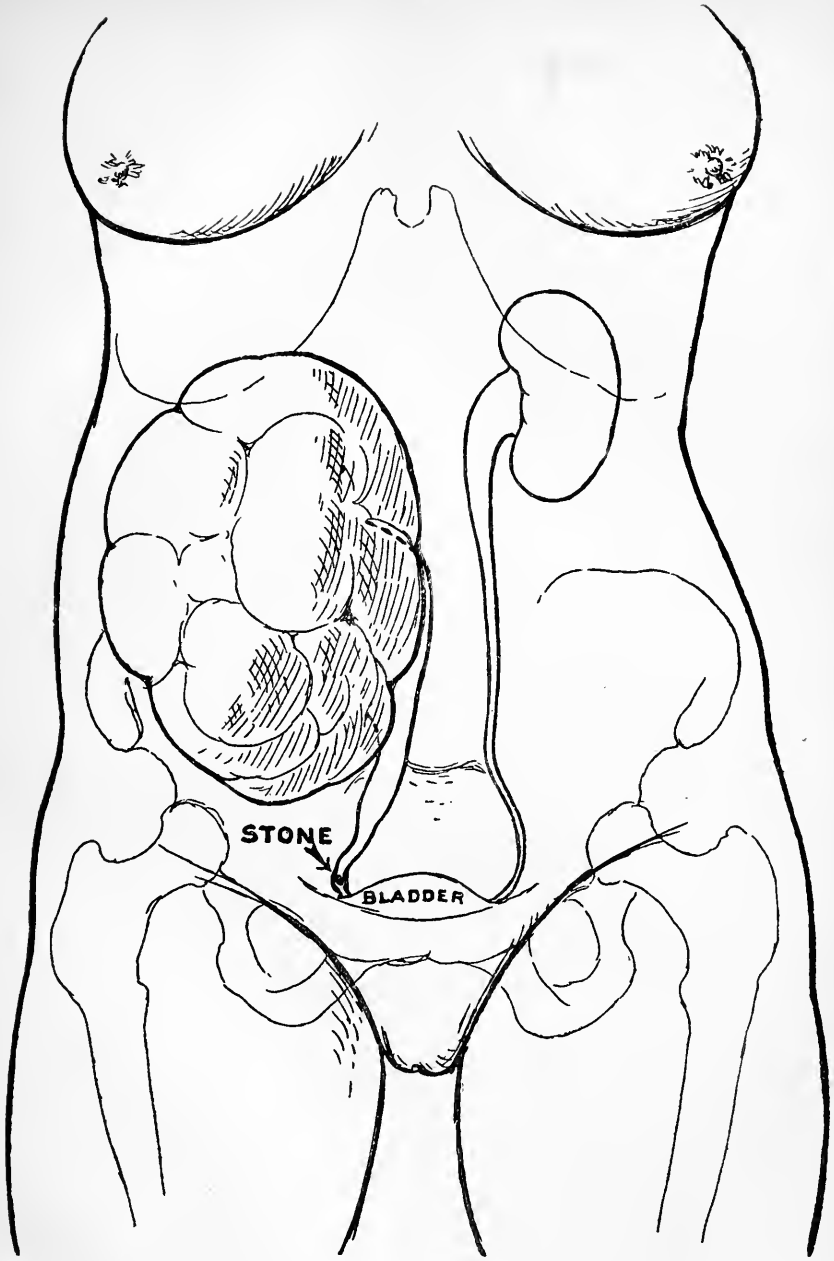

Fig. 3.—Large hydronephrotic kidney mass. Somewhat lower, and more across median line than shown.

She was a fairly well-nourished female of good color to skin and mucous membranes. There was no rigidity of the abdominal muscles, and but slight tenderness over the right side of abdomen. There was a large, tense, elastic mass extending from the free border of the ribs to the pelvic brim and across the median line (See Fig. 3). This mass was dull on percussion, slightly movable, and not of especial tenderness on deep palpation. It produced a very appreciable distention of the abdomen. Vaginal examination gave the sense of an indefinite mass high up on the right side. Colonic inflation and the position of the mass left no doubt of the condition as a kidney tumor. This patient's condition before entrance was diagnosed as ovarian cyst and her reason for consulting a physician was as much because of her increase in girth as because of the discomfort in the side.

Operation (Dr. Schley).—Incision through outer border of the right rectus. Large, dark, elastic mass with colon running over upper part. Extended from behind lower ribs to pelvis. The ureter was greatly dilated, to quite an inch in diameter through greater part of its course. Condition plainly one of hydronephrosis. Passing a bougie through opening in the ureter demonstrated a stone about 2½ inches from the bladder, but so rough and embedded in the greatly thickened ureter that it could not be dislodged, and a 2-stage operation was considered wiser and safer. The kidney, of which but a mere shell of tissue remained, was removed and the ureter excised to within a few inches of the stone. This patient made so perfect a convalescence and felt so well that she preferred to retain the calculus, and operation for its removal was not insisted upon. She has been followed, and remains in good health. Microscopic examination of a small remnant of cortical tissue near the fundus showed swollen glomeruli and marked parenchymatous degeneration of the convoluted tubules. Radiograph showed the calculus near the bladder (Fig. 4). Two photographs show the condition of the kidney (Figs. 5 and 6).

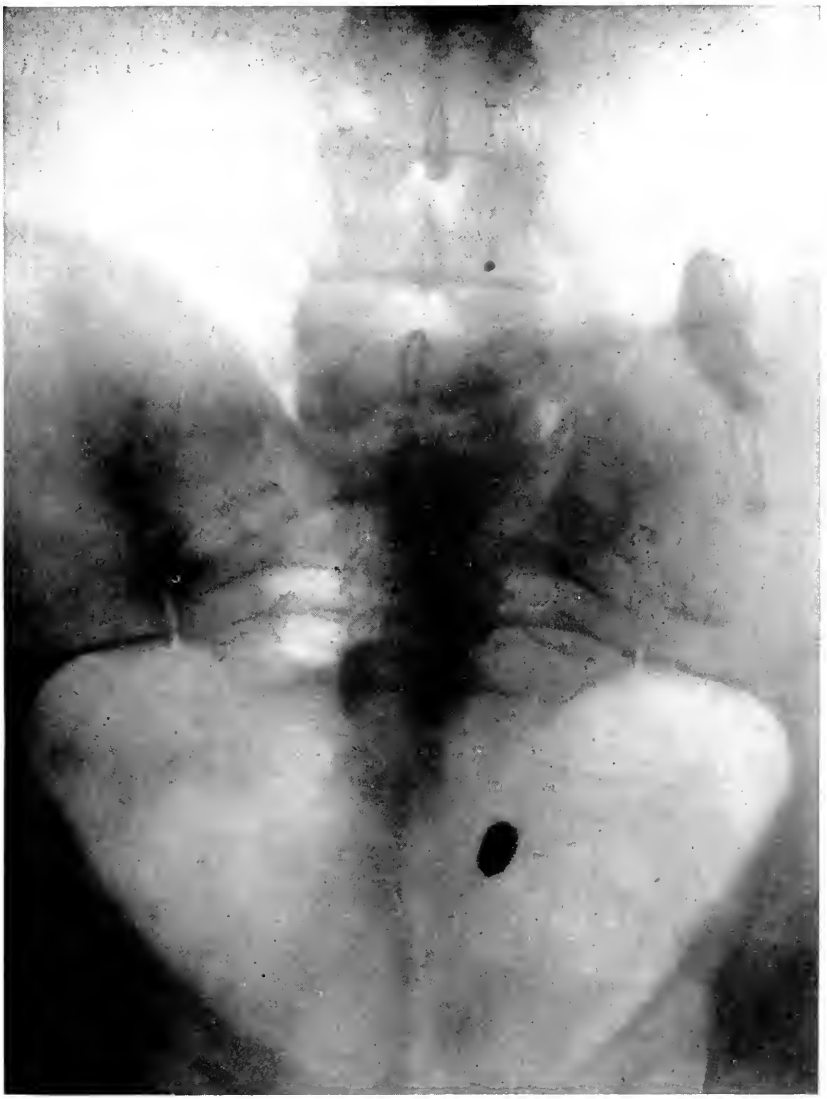

Fig. 4.—Stone in lower part of right ureter.

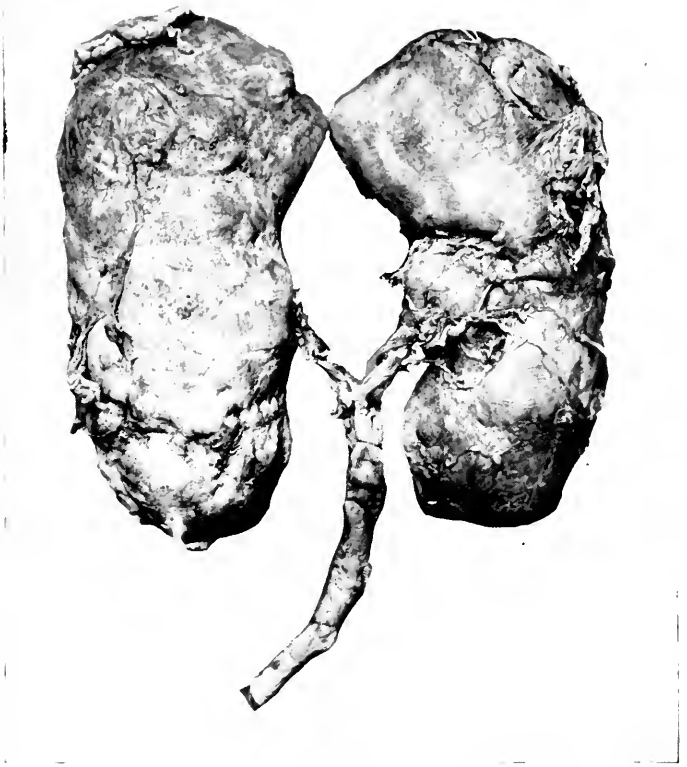

Fig. 5.—Reduced picture of kidney exterior. Specimen was put in strong formalin, without previous distention, and great shrinkage resulted.

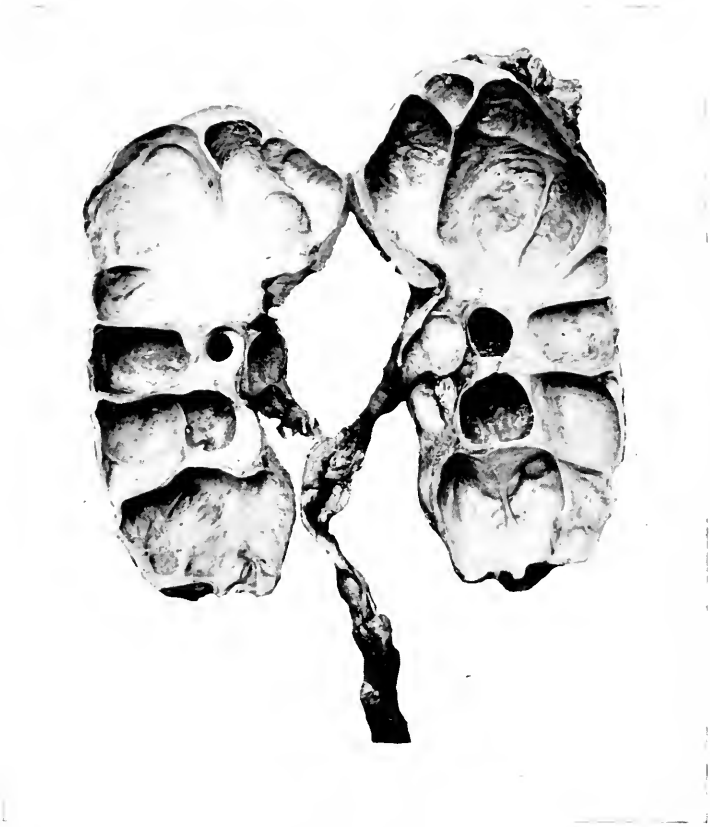

Fig. 6.—Thin shell of cortex at upper pole can be seen. Typical hydronephrosis of extreme degree.

TUBERCULOUS PERITONITIS SIMULATING RECURRING ATTACKS OF APPENDICITIS.

W. Scott Schley, M.D.

H. Le V., a young man of 19 years, was admitted to the hospital December 27, 1911. His chief complaint was pain in the right lower quadrant of the abdomen.

Family History.—Both parents alive and well.

Two years ago he had some cough and expectoration and was told that he had trouble at the left apex. He left work, lived in the country, and apparently recovered. About 8 months ago he began to be troubled with stomach disturbances, gas and gurgling, but did not lose weight. Three months before coming to the hospital, and while working, he was taken with a severe epigastric pain, but managed to finish his work, and the pain had gone by night. There was no nausea with the attack. Six weeks later had a similar seizure, the pain lasting some 8 hours and extending from epigastrium to right lower quadrant. He felt uneasy and tired before the pain began. Two weeks before entrance he had his last attack, with pain chiefly of the right lower quadrant. There was nausea and vomiting and the duration 12 hours. He had been constipated prior to the attack. He was seen by a physician at this time, and the diagnosis of appendicitis made. He has had some soreness in the right lower quadrant since his first attack, and said that he had occasional twinges of pain in the right side.

His general appearance was that of a well-nourished, well-muscled and healthy-looking young man. In the chest a few subcrepitant râles could be heard at the right apex behind. Thorax expanded well and equally on both sides, resonance good. Heart somewhat irregular, 56 per minute on examination. Sounds clear and strong. Slight blowing systolic murmur heard all over chest, and loudest at apex. There was no rigidity to the abdomen or mass felt. Slight tenderness existed in a small area just to right and below umbilicus. Superficial glands were not appreciably enlarged, with the possible exception of the right epitrochlear. His temperature on admission was 97⅗, and on discharge 98. Beyond a slight reaction to 100⅘ following operation, he had no temperature at all. His pulse ran a fairly regular rate, averaging in the seventies.

Operation.—Intermuscular appendix incision. Scattered over the peritoneal surfaces were numerous discrete pearly nodules varying in size from a pin point to a pin head. They were more numerous in the mesentery of the appendix than elsewhere, and became much more scattered as one left that region. All the loops of ileum drawn down into the wound showed

tubercles. They were more numerous upon the visceral than upon the parietal peritoneum. No adhesions could be demonstrated. The serous surfaces were moist, but there was no fluid. The appendix lay below caput, towards pelvic brim. It was but moderately congested, and had but a moderate number of tubercles on the surface. (See Fig. 1.) The presence of so many in the mesentery was thought to possibly indicate ulceration of the mucosa, and it was removed. It lay free and there were no adhesions. Grossly, the appendix showed only moderate thickening of its coats. The internal caliber was even throughout, and there were no constrictions. It was empty. Several hemorrhagic spots in the mucosa, without apparent ulceration, appeared. They were on the side of the mesenteric attachment. The pathological report shows: Chronic appendicitis. Tuberculous peri-appendicitis invading the meso-appendix.

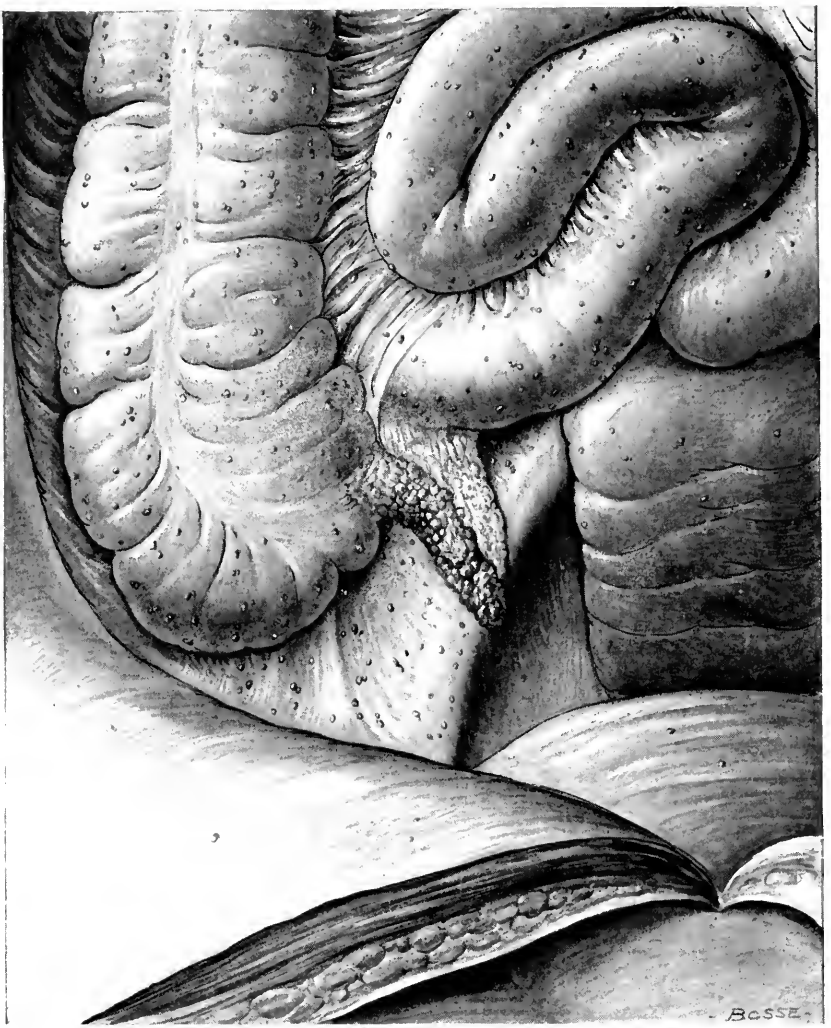

Fig. 1.—Tuberculous periappendicitis. Numerous tubercles in peritoneal investment of appendix and in its mesentery.

THE GATCH BED IN SURGICAL WORK.

W. Scott Schley, M.D.

For nearly a year, in the male surgical ward of the 1st Division, we have been trying a bed that has proved a great success. In addition to the usual spring bed, it is intended to provide for an easy, comfortable and secure sitting or semi-recumbent position. It has served especially well in those abdominal cases for which elevated posture is necessary to facilitate drainage, prevent dissemination of exudates, or for other reasons. Its great advantage over the back-

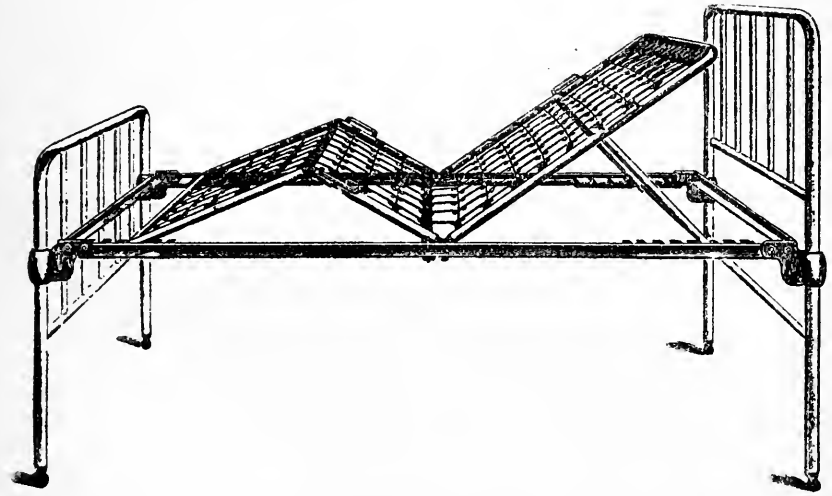

Fig. 1.—Gatch bed in profile. Ratchet on frame provides for a variety of angles of elevation, both for back and legs.

rests of various forms lies not only in the fact of the inclined planes being integral with the bed itself, but in its taking care of the lower extremity as well. This bed was devised by Dr. Gatch, of the Johns Hopkins Hospital, and the first one was made up in that institution about 2 years ago. The adjustable spring is now made up to fit any single bed, and its usefulness is thereby greatly extended, as it can be

adapted to beds in a ward without changing their uniformity. The uprights of the regular bed have also recently been fitted with sockets for securing two levels for the mattress; a high surgical bed or a lower convalescent bed. The ordinary ward mattress takes the angles made by the elevation of the spring planes, and a comfortable curve is the result that can be borne for long periods. (Figs. 1 and 2.)

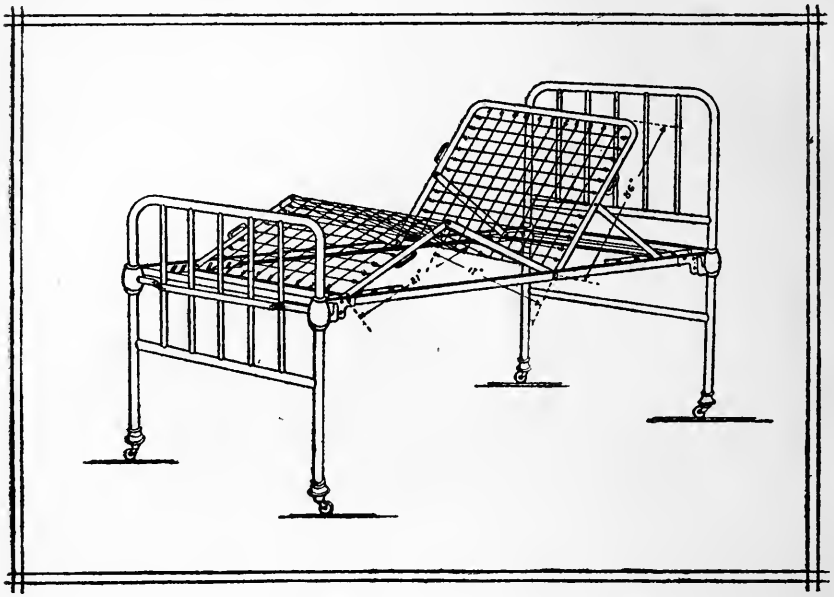

Fig. 2.—Gatch bed, with dimensions in inches.

SUBPHRENIC ABSCESS COMPLICATING APPENDICITIS.*

JOHN DOUGLAS, M.D.

Subphrenic abscess occurs as a complication of acute appendicitis in between .5 and 1 per cent of the cases, according to Ross (Journal A. M. A., August 12, 1911), who has analyzed the reports of a large number of statistics by Treves, Kelly and Hurdon, by Lance, and also 3,891 cases of acute appendicitis occurring in the German Hospital and Mary J. Drexel Home, Philadelphia, Pa. In the 31 cases reported by him, there were 22 deaths. Of the 31 cases, from brief history reports, it would appear that in 15 the abscess was situated below the liver. In the remaining 16, where the pus was between the liver and the diaphragm, or above the lower surface of the liver, there were only 4 recoveries.

The following two quotations from Ross' article prompt the report of this case with the X-ray photograph, illustrating its value as a means of diagnosis: "It is to my mind a refinement of diagnosis at present impracticable of accomplishment for any one to determine definitely the variety of subphrenic abscess before operation. As will be seen later, we are fortunate, indeed, always to diagnose even the existence of a subphrenic abscess." Later on, he states: "Only the most careful study of individual cases will give even an approach to correct diagnosis."

Grace S., aged 13. Seen in consultation, July 31, 1911. Her illness began 11 days before, with an attack of severe vomiting, which lasted 1½ days, when she began to have severe pain in the right side of the abdomen. The pain was severe, and aching in character, continuous, not localized, and radiated to the back and right shoulder. It was increased by respiration. The pain had been gradually decreasing, and was now most marked over the lower right chest, in the axillary line. She had fever (103° to 104° F.) every evening, but no chills. Has had some cough, but no expectoration. Was slightly jaundiced for three or four days, but jaundice had disappeared when she was seen by the writer. The bowels had moved every day.

*Reprinted from "Surgery, Gynecology and Obstetrics."

Physical examination of the chest showed slightly diminished expansion and breath sounds, slight dulness, and a few large, moist râles over the right base.

There was no general abdominal rigidity or tenderness, but over the upper right quadrant of the abdomen rigidity and tenderness were marked. A mass, apparently the lower border of the liver, could be felt 2 inches below the costal arch. This area, and just below it, were very tender. Percussion in the right mammary line was flat from the fifth intercostal space down to the edge of the mass. There was some tenderness in the right costo-vertebral angle. The temperature was 103° F., pulse 140, respiration 30. The leucocyte count was 25,000, with 88 per cent of polynuclear cells.

A diagnosis of high appendicular abscess was made, and she was referred to St. Luke's Hospital for operation.

Operation.—An incision 3½ inches long was made along the border of the right rectus muscle, with its lower end about on a level with the umbilicus. The appendix was found with its outer extremity almost sloughed away, behind a high, undescended cæcum, just underneath the liver. There was a small amount of pus, with the characteristic odor of colon infection, well walled off from the rest of the peritoneal cavity by adhesions and the omentum, and limited above by the under surface of the liver. The appendix was removed. The liver had been apparently displaced downward, and examination demonstrated adhesions between the diaphragm and the upper surface of the right lobe. On separating these adhesions a considerable quantity of bloody pus, smelling of colon infection, was discharged. A thick rubber dam drain was passed up a distance of 12 cm. between the liver and diaphragm, and a counter drainage opening made in the loin. The final incision was partly closed, and drained with a rubber dam drain.

For a week following the operation there was a profuse discharge of bloody, purulent material, which gradually became less, but the patient continued to have an irregular temperature—up to 101° to 102° in the afternoon—the cough became more troublesome, while the physical signs persisted. It was believed that the subphrenic abscess was not draining, so to determine this fact the X-ray shown in the illustration (Fig. 1) was taken. This demonstrated very plainly that the diaphragm, above the liver, was pushed upward almost to a point to the level of the eighth rib behind. So an anæsthetic was administered on August 11th, the eleventh day after operation, and a long, soft rubber tube, 1 cm. in diameter, was inserted, to replace the rubber dam, with a further evacuation of pus. The cavity was irrigated through this rubber tube, and drainage was again profuse. On August 21st a second X-ray was taken, showing the diaphragm considerably lower; the tube was removed, and again replaced by rubber dam. The temperature stayed down for four days, but on August 27th rose to 103° again.

Under light chloroform anæsthesia, the adhesion between the right lobe of the liver and the diaphragm were broken up by the finger, inserted through the incision in the loin, with the resulting escape of considerable pus. A 11 cm. rubber tube was introduced a distance of 12 cm., and the cavity, which was well walled off, irrigated.

As the adhesions were quite dense, the question arose whether the dia-

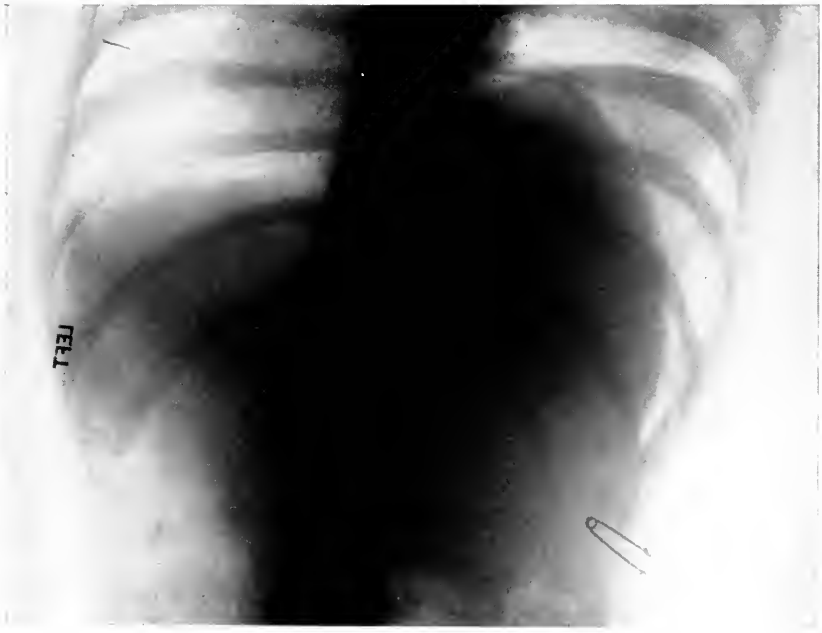

Fig. 1.—Showing diaphragm displaced upward by abscess.

phragm had been perforated, with involvement of the pleural cavity, and a third plate (Fig. 2) was taken, which demonstrated that the end of the tube was below the diaphragm. The following day the temperature was normal, and further recovery was uneventful.

It is also of interest in this case to note the presence of jaundice as Besredka (quoted by Ross) states, as a means of differential diagnosis "there is never jaundice in uncomplicated subphrenic abscesses."

The mortality of subphrenic abscess is about 33 per cent. It is lower in children than in adults, probably because the large majority of cases are due to appendicitis, while in adults a considerable number of cases are caused by perforation of the stomach or duodenum. Intrathoracic complications have been estimated to be present in 66 per cent of the cases. This is probably too high, as Ross states that of 21 cases coming to autopsy, only 5 showed purulent pleurisy and 1 a pleurisy with exudate not purulent. Intraperitoneal abscesses, which are more frequent, are more apt to perforate the diaphragm than extraperitoneal.

The diagnosis is difficult. In addition to the quotation from Ross given above, Reeve (American Practice of Surgery, Vol. 7, 1910, p. 487) says: "There are no certain physical signs by which in all cases collections of fluids above the diaphragm can be distinguished from those situated below this structure." Also, "It is not possible to make a differential diagnosis between a subphrenic abscess and an encapsulated basal empyema." He advises, however, the use of the Roentgen rays, and mentions a case in which Monro demonstrated the presence of subphrenic abscess by the X-ray after rib resection and several punctures had failed. Although writers on the subject allude to the unreliability of the physical signs, this case of Monro's was the only one found by the writer in which the X-ray as a means of diagnosis was made use of.

The physical signs usually found are dulness or flatness, diminished breath and voice sounds and vocal fremitus, with the presence of râles over the base of the lung, the area of dulness being characterized by being convex upward and not changing its area with a change in the position of the patient. In those cases where gas is present in the abscess cavity there are 3 zones of different resonance on percussion. The normal pulmonary resonance above, a zone of tympanic resonance caused by the gas, below this, and the area of flatness caused by the pus, which is continuous on the right side with

the liver flatness. When there is also fluid in the pleural cavity there will be 4 percussion zones, an area of flatness intervening between the normal pulmonary tympany and the tympanitic zone caused by the gas. In right-sided subphrenic abscess the liver is apt to be more or less displaced downward.

It is recommended by all writers on the subject that careful exploration with the aspirating needle be made in all questionable cases —and the diagnosis from the physical signs being so uncertain, makes the majority of cases questionable. The certainty with which the situation and size of the abscess is shown in the accompanying radiographs demonstrates their great value in diagnosis, although probably they would not be so plain in a heavy adult as in a child.

It is believed that had a radiograph been taken in the following case, the diagnosis might have been made, as was impossible from the physical signs, and the life of the patient perhaps saved.

M. P., boy, aged 8. Seen in consultation with Dr. J. F. Bell, of Englewood, N. J., Nov. 7, 1910. Patient had a gangrenous appendix and general peritonitis, a large amount of thin pus being present in the general peritoneal cavity, as was demonstrated by operation at the Englewood Hospital, three hours after being seen by the writer. Patient reacted well from the anæsthetic, and for ten days temperature was between 99° F. and 100° F., and bowels moved daily. Patient, however, did not look well, and on the fourth day complained of pain in the *left* chest. Examination showed pleuritic friction râles over *left* base, which disappeared two days later, although he still complained of pain in left chest and abdomen, which pains continued irregularly for several days. On the tenth day after the operation he still complained of these pains, as well as pain in the throat and ears, and the temperature rose to 104.3° F., pulse 180. Careful, complete examination, as well as exploration of the wound, was negative. This examination was verified on the following (the twelfth) day by two additional physicians, who examined him in consultation. His leucocyte count was 21,400, with 79 per cent of polynuclear cells. On Nov. 19th, the morning of the twelfth day, after a period of apparent relief and temperature subsidence, he suddenly developed at 8 A.M., a harassing cough, with scant mucous expectoration. At 9 A.M. there occurred an enormous purulent expectoration, which became bloody as edema developed, with cyanosis, evidently the rupture of an abscess into the lung. This expectoration showed the presence of streptococci and some diplococci and a bacillus, apparently the bacillus coli communis. The respiration became more shallow, cyanosis was marked, and the pulse ran up to 204. In about an hour and a quarter the patient died, apparently drowned by the pus in the lung.

An autopsy in this case was refused, and while it is possible that the abscess which ruptured might have been in the lung or mediastinum, there were certainly no physical signs indicating its presence

Fig. 2.—Showing tube in abscess cavity, between liver and diaphragm.

Fig. 3.—Showing condition on discharge.

except the friction râles over the left base for a few days, and no subjective symptoms except pain in the left chest and shoulder. It is, therefore, believed that this was a subphrenic abscess which ruptured through the diaphragm into the lung, which, in the absence of physical signs, might have been demonstrated by the X-ray in time to save the patient.

Treatment of subphrenic abscess consists of evacuation of the pus, either by means of an incision in the loin or abdomen, after dealing with the primary cause, and drainage with a rubber tube. This method was followed in the first case reported, and was satisfactory as long as the drainage tube was kept in place.

In abscess high up under the diaphragm, more satisfactory drainage is obtained by the subpleural route, the tenth rib being resected in the mid axillary line and the abscess opened through the diaphragm below the pleural reflection.

If the incision opens the pleural cavity (trans-pleural route), the pleura above should be closed, and the diaphragm sutured to the intercostal muscles in the incision, to close off the cavity above before opening the abscess, which is then drained with a large rubber tube.

FIVE CASES OF ESOPHAGEAL OBSTRUCTION FROM THREE DIFFERENT CAUSES.

NATHAN W. GREEN, M.D.

During the past eight months there have occurred on the service of Dr. Robert Abbe (Surgical Division A) five cases of obstruction of the esophagus from three different causes. The first was in a child of 2 years, due to a foreign body which had lodged in the lower part, just above the diaphragmatic opening. The next two were also in children, one of 3, and the other of 3½ years, due to the drinking of caustic fluids which had been carelessly left about. These were practically impermeable strictures except to small amounts of fluid. The last 2 cases were due to cancer, one situated 10½ inches from the upper alveolar border, and the other at the cardiac extremity of the esophagus.

Brief histories of these cases follow:

OBSTRUCTION OF THE ESOPHAGUS FROM A STEEL BALL CAUSING A BALL VALVE.

Surgical No. 85,611.—J. G., 2 years old, was admitted to the service of Dr. Robert Abbe (Surgical Division A) May 20, 1911. He was previously a normal, healthy baby. For twenty-four hours before admission, he had been unable to retain food. Although he appeared hungry, he expelled everything a few minutes after eating. There was no blood and no fever nor chill. For the three or four hours previous to admission, he had refused both solids and liquids. He had not cried, nor did he have severe pain or discomfort.

On examination he appeared restless and anxious. His mouth and tongue were clean. Skin and mucous membranes clear. There were no rashes. His heart and lungs were normal. There was no distention, no tenderness nor rigidity of the abdomen. No mass was felt, nor localized tenderness in any part of the body. The child vomited everything given him as soon as the smallest quantity was taken. There was obstruction to the passage of food. The child could not swallow water. In the evening of the day of admission (May 20th), he vomited a small quantity of brownish fluid tinged with blood. He was taken to the operating room shortly after, and an instrument was passed about ten inches, but no metal nor stone object could be felt.

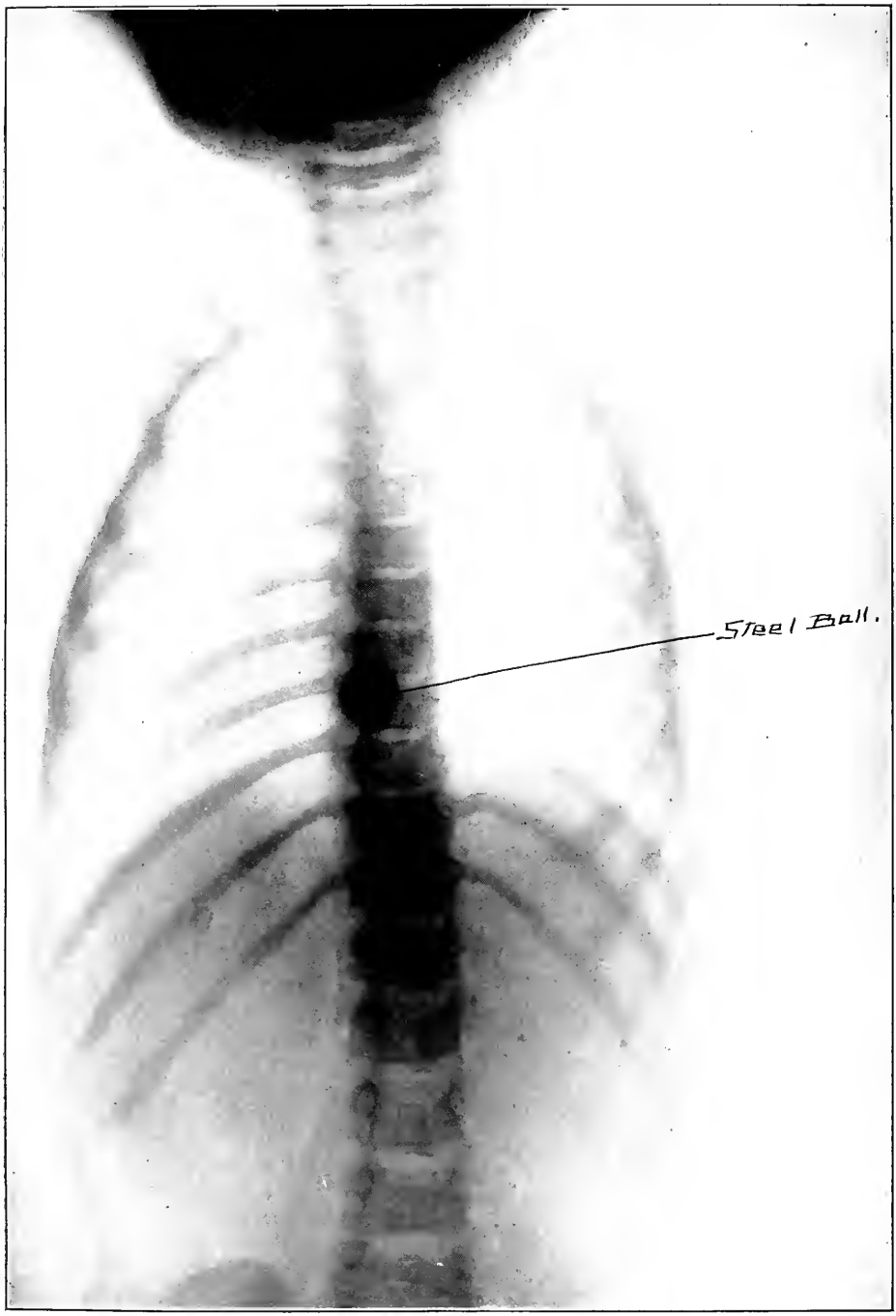

Fig. 1.—This picture shows steel ball at cardiac end of the esophagus before its displacement.

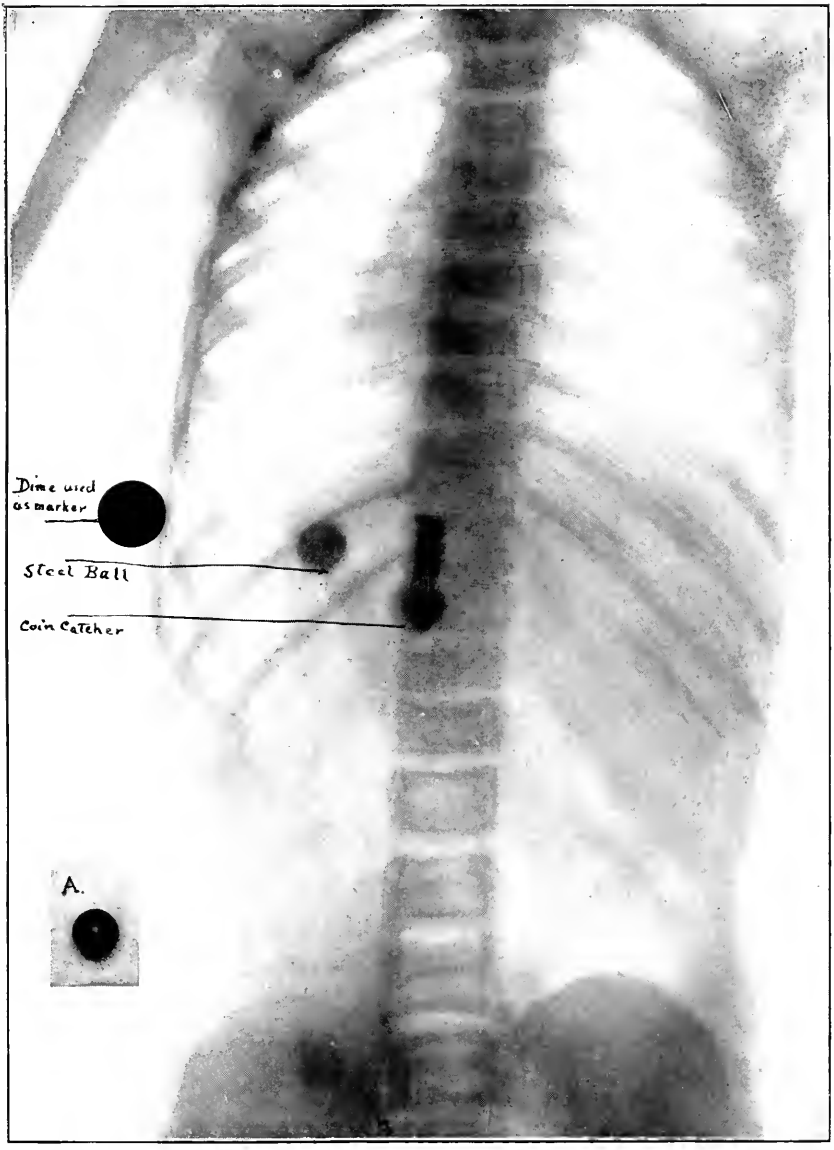

Fig. 2.—X-ray, showing steel ball displaced into stomach, with coin catcher at its side. This picture has been reversed in printing. A.—Picture of steel ball, ½ inch in diameter, which caused esophageal obstruction.

An X-ray was taken on the 21st, showing an object opposite the 8th rib, round and looking like a button. Fig. 1.

On the 22d, he was again taken to the operating room, and the esophagoscope was passed under ether with the aid of vision. The entire mucosa of the esophagus looked healthy, but no definite foreign body could be seen. At the end of the tube there was, however, a dark spot, but this was not clearly defined. The esophagoscope was withdrawn and a coin-catcher thrust into the stomach. An X-ray was taken with this instrument in place, and showed that the foreign body had been displaced, and now lay in the stomach, below the diaphragm. Fig. 2.

The night following this treatment the child did not vomit, nor did he vomit since that time while in the hospital.

On May 26th he passed a large semi-formed stool "containing a marble." This marble proved to be a steel ball such as is used in ball bearings of motor cars. Fig. 2A. This had acted at the cardiac end of the esophagus as a ball for a ball valve, and had absolutely prevented his swallowing.

On June 1st he was discharged in good condition.

TWO CASES OF STRICTURE OF THE ESOPHAGUS FOLLOWING THE INGESTION OF A CAUSTIC FLUID.

Surgical No. 86,880.—C. D., 3 years old, was admitted to the service of Division A, September 12, 1911, with the history of having swallowed some concentrated lye in April, 1910. For a short time after this he was able to swallow solid food, but gradually obstruction increased, and everything solid would be regurgitated immediately after taking. By feeding with thin broths and milk, he had been kept alive.

On admission he was thin, pale and delicate, with some involvement of the right lung. His heart and other viscera were normal, with the exception of an obstruction of the esophagus, impermeable except to small amounts of fluids.

Two days after admission, a gastrostomy was performed by Dr. Schley, and through this his nourishment was given until his condition sufficiently improved to warrant further interference. With the co-operation of Dr. L. T. Le Wald, it was possible to obtain good X-ray pictures of his esophageal condition. Fig. 3.

On the 3d of November he was taken to the operating room, and by the aid of the esophagoscope a filiform bougie was passed through the stricture into the stomach. This was followed by a silk string, and the esophagus was then dilated by means of the Abbe string cutting method[1 2 3] to about a 20 French bougie. The next day the patient retained the water given him by mouth.

On the 6th of November he was again X-rayed, and on the 10th still another picture was obtained, after which he went to the operating room for

[1] G. Gottstein, Keen's Surgery, 1910, vol. iii, p. 808.

[2] R. Abbe, N. Y. Med. Record, 1893, Nr. 25.

[3] R. Abbe, Ann. of Surg., 1893, vol. xii.

a second string cutting operation. The esophagoscope was introduced about 6 inches to the top of the first stricture, a small bougie then introduced through the constriction into the stomach, and to it was tied a silk string. The operation of the previous week was repeated until a number 34 French bougie was passed into the stomach. An attempt was made before passing this bougie to pass a bougie à Boule. This engaged its tip at the lower stricture, but it could not be forced through even with the aid of the string-sawing.[4]

On November 17th and 18th, the note is made that an 8-oz. feeding was taken by mouth, and he retained his nourishment. A number 28 bougie has since been passed twice a week for a number of weeks, and the patient now takes the greater part of his nourishment by mouth.[5]

Highest temperature after operation was 102 3-5°, and highest pulse rate 160°.

Surgical No. 87,688.—D. B., 3½ years old, was referred by Dr. Withington, of Pittsfield, Mass., to the service of Dr. Robert Abbe, Division A, and was admitted November 26, 1911.

About 9 months previous to admission, the child swallowed some potash used for cleaning. From that time there was a constantly increasing difficulty in swallowing. Finally, everything taken into the mouth was regurgitated. Upon examination, she was found weak, greatly emaciated and impassive. Heart and lungs normal. By X-ray examination (Dr. Le Wald), with the aid of bismuth, a distention of the upper part of the esophagus with an almost complete obliteration of the remainder was shown. Fig. 4.

On November 27th a gastrostomy was performed under ether and a tube left in place. Feedings of fluids were begun at once through this tube.

On December 22d, by aid of the esophagoscope, and impossible without it, a filiform bougie was passed through the stricture as in the previous case. This was followed by a silk string, and the string cutting operation was performed by Dr. Abbe. This was continued until the esophagus would permit of the passage of a number 30 French bougie. After a few days, a number 28 bougie was passed every week, and latterly twice a week.

The patient then took and retained almost all of her food by mouth. On January 7th she had not had any gastrostomy feeding for 10 days. On the 31st she weighed 31 lbs. 4 ozs., a gain of 12 pounds. The gastrostomy wound was closed, and all food was taken and retained by mouth.[6]

Highest post-operative temperature, 101°; highest pulse rate, 120°.

TWO CASES OF MALIGNANT STRICTURE OF THE ESOPHAGUS.

Surgical No. 87,414.—Mrs. I. A., 54 years old, Russian, and a widow, was admitted to the service of Dr. Robert Abbe (Surgical Division A), on

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[4]It was interesting to note that the distance from the upper teeth to the cardiac opening, by palpation with the index finger in the stomach, was precisely 10½ inches; in this child 37 inches long.

[5]On May 1, 1912, he had gained several pounds, and takes all his nourishment by mouth, and has a No. 34 F. bougie passed once in 7 to 10 days.

[6]She now weighs 37 pounds and 4 ounces, April 15th.

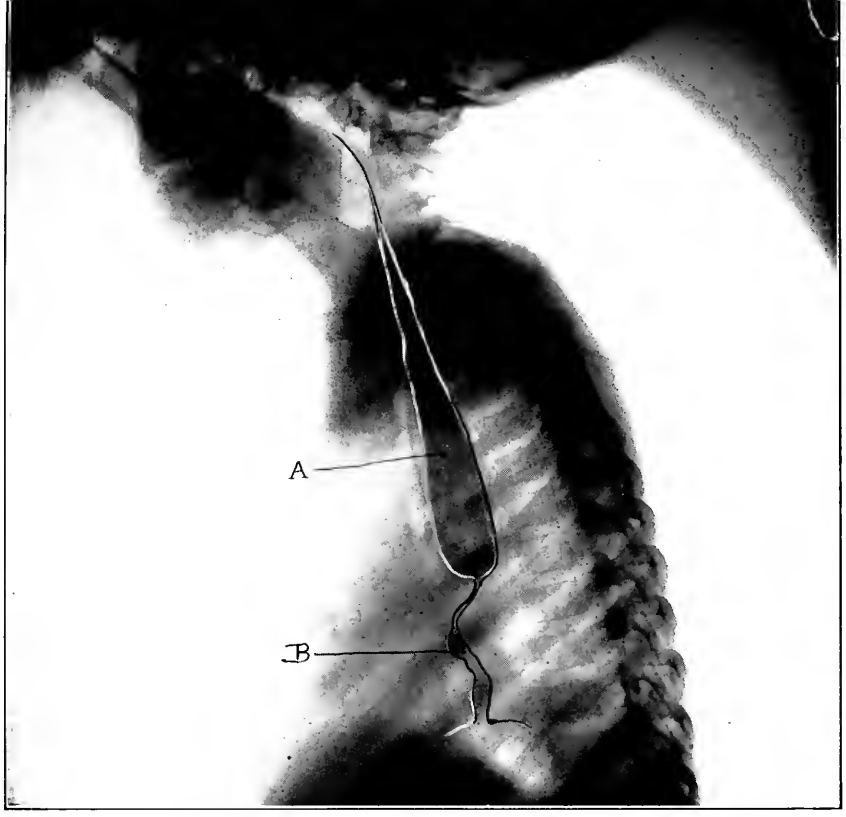


Fig. 3.—X-ray and bismuth picture, showing dilatation (A) of esophagus above stricture (B). It also shows the track of the minute lumen in the strictured portion. This picture was taken after partial dilatation, whereas Fig. 4 (the next case) was taken before any interference of an operative nature. Both of these pictures have been outlined with black and white to facilitate the tracing of the strictured portion.

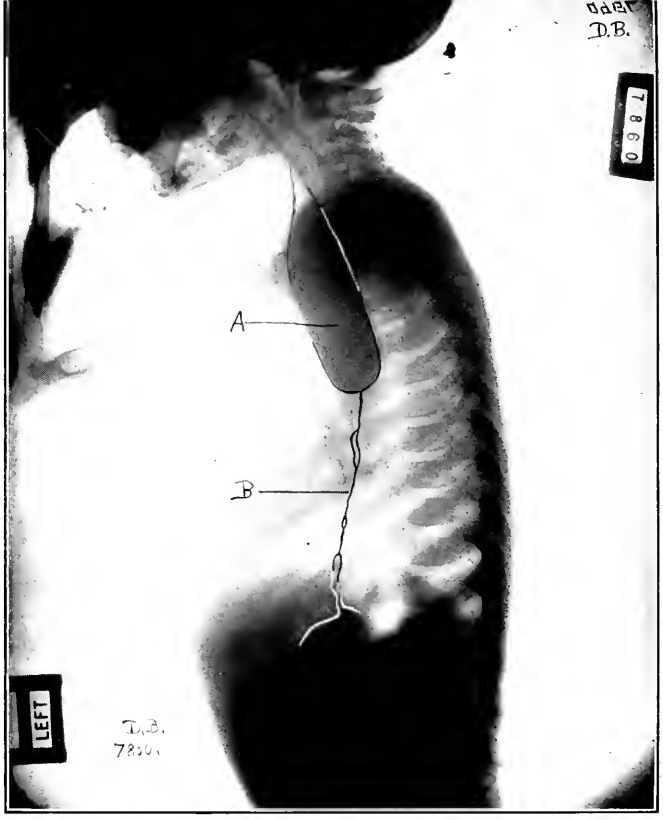


Fig. 4.—X-ray and bismuth picture showing dilatation (A) of esophagus above stricture (B).

October 31, 1911. Her chief complaint was inability to swallow. About six months ago she first noticed this difficulty, which was limited chiefly to solid food. She had grown thin, was hungry, but could not eat. She had no pain, with the exception that she had feeling of discomfort when a piece of food lodged in her esophagus. Neither her past nor her present history had salient points. Her family history was negative. Upon her admission she appeared to be a poorly nourished woman, chronically ill. Upon attempts to swallow, food regurgitated frequently. Her lungs were clear, heart slightly enlarged, her abdomen soft and relaxed, with no tenderness nor masses.

Her liver was two inches below costal margin in middle line. There was no tenderness. Examination of her esophagus showed some obstruction. A gastrostomy was done on November 3d, by Dr. Abbe, under local anæsthesia, and a tube introduced, and by means of this the necessary feedings were carried on.

Examination of the esophagus by bougies showed the presence of a stricture 8 mm. in diameter and 10½ inches from the upper incisor border.

An X-ray and bismuth picture of the stricture was taken with the assistance of Dr. Le Wald. The lower end of the esophagus was plugged by pulling up through the gastrostomy opening the ball of a bougie à Boule upon a stout silk thread. The patient was then directed to swallow 2 ozs. of bismuth sub-carbonate suspended in a fermented milk product, and at once the X-ray was taken. The result is shown in the accompanying picture (Fig. 5). The whole clinical picture was one of malignant disease, but it was impossible to confirm this by a pathological section.

After some dilatation of the stricture with the appropriate bougies and the string sawing method, a lead capsule containing 100 mg. of radium was introduced by Dr. Abbe and left there for six hours. After this procedure the stricture was dilated with a bougie once a week. She was shortly able to swallow with comfort. Upon her discharge from the hospital, December 18th, although it was not possible to hope for a cure, she was much improved. She said she could swallow "everything," and certainly there was a great amelioration of her symptoms. She has returned once a week to the hospital for observation.

Surgical No. 88,040.—Mrs. L. L., aged 59, a widow, was admitted to the service of Dr. Abbe (Surgical Division A), on December 29, 1911. Her chief complaint was inability to swallow. About a year previous to admission, she began to notice that food other than soft food was vomited at once. The beginning was gradual, but the condition grew steadily worse, so that on admission all foods were regurgitated. There was discomfort, but no accompanying pain. Her past history was good and her family history negative. Upon examination her viscera appeared to be normal, with the exception of her esophageal and esophago-gastric region. In her esophagus a bougie passed but 13½ inches and then met with obstruction. (She was rather a short woman and this distance proved to be nearly the length of her esophagus, as was shown later at operation.)

An X-ray and bismuth picture of the esophagus and stomach by Dr. Le

Wald showed a stricture at the cardiac end of the esophagus (Fig. 6). The diagnosis of carcinoma of this region was made. It was confirmed at the operation by Dr. Abbe a few days later. A gastrostomy was performed, and a more or less annular carcinoma was demonstrated.

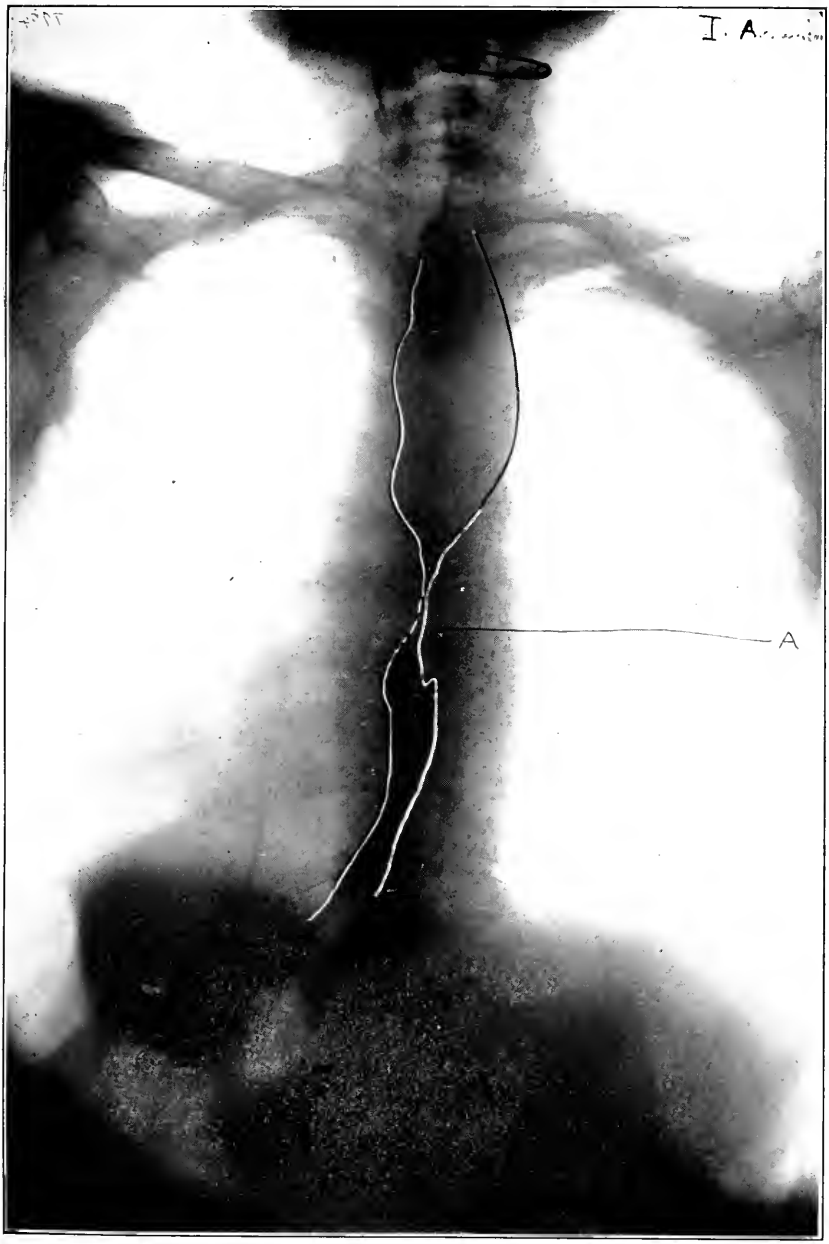


Fig. 5.—This picture shows the dilated portion of the esophagus above the stricture (A), which in this case presents the appearance of malignancy. Before taking this picture the esophagus was plugged by pulling up through the gastrostomy opening the ball of a bougie à Boule. The patient was then directed to swallow the bismuth mixture, and the X-ray was taken. The position has been reversed in printing. The stricture and the lumen of the esophagus have been outlined to facilitate interpretation.

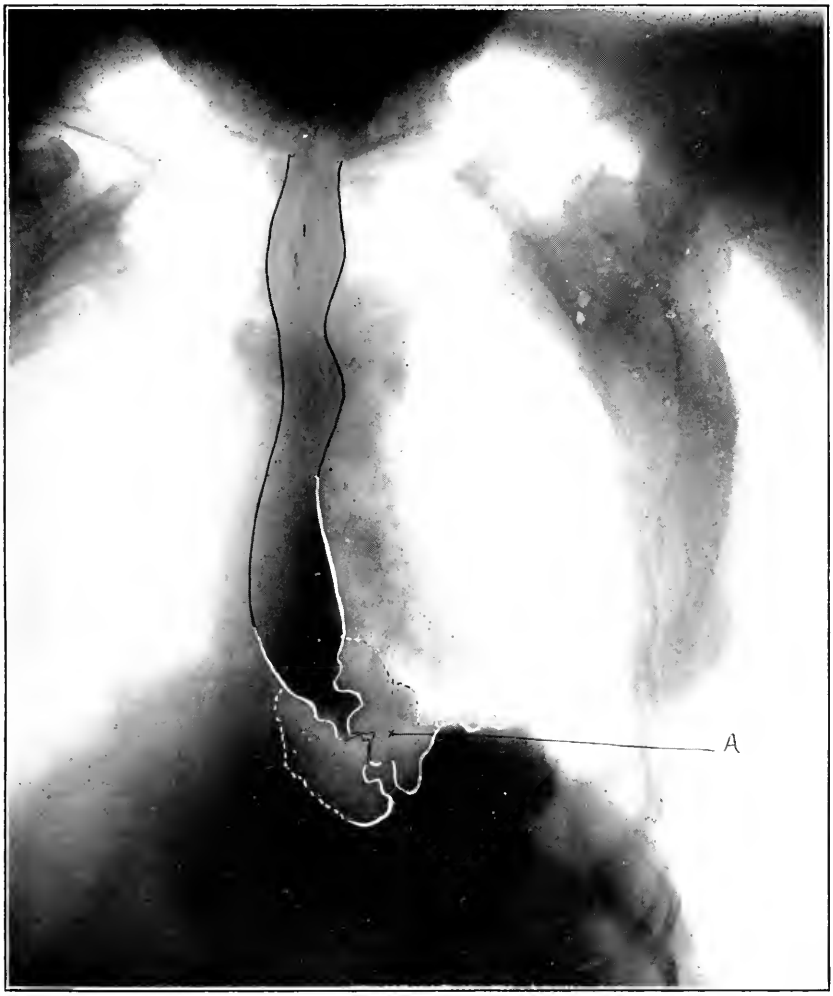


Fig. 6.—This picture shows a malignant stricture (A) at the cardiac end of the esophagus. The esophagus is seen dilated above it, and the stomach is also seen containing bismuth below it. Contour of the esophagus has been outlined to facilitate interpretation. The tumor mass is indicated by the dotted line.

THREE CASES OF ILEO-COLIC INTUSSUSCEPTION WITH REDUCTION AND ANCHORAGE BY MEANS OF THE APPENDIX—TWO RECOVERIES.

NATHAN W. GREEN, M.D.

Since April, 1911, there have occurred on the surgical service of Dr. Abbe, Division A, three cases of acute intestinal intussusception. All three came to operation. Two of them recovered. Two of these cases were sent in early, one had lasted a longer time. The histories and method of dealing with the intussusception may serve to promote discussion, and may be of interest.

Surgical No. 85,972.—J. H., 6 months old, was admitted to the service of Dr. Robert Abbe, Division A, June 19, 1911. For 21 hours previous to admission, his mother noticed he was very cross, and would gag and scream with pain. He refused to nurse, and would not take water. At 5 o'clock in the morning of the day of admission, his mother noticed a bloody stool. Frequently thereafter this was repeated. At 8 o'clock he began to vomit light yellow material. The family physician was called, and sent the infant directly to the hospital. He arrived at 1 o'clock.

Upon examination, a typical sausage-shaped tumor was found extending across the abdomen. By rectal examination, the tip of the intussusceptum could be felt presenting the feel of a "cervix." The child was operated upon at 3 o'clock in the afternoon.

Operation (Dr. Green).—A median incision was made from the umbilicus to the pubis. The transverse colon was found distended and bluish, and the ileum was seen passing into it. This sausage-shaped "tumor" extended down to the rectum. By gentle squeezing and traction, the intussusception was entirely reduced. There was no lack of lustre, nor was there any attempt at adhesion formation. Some means for preventing its recurrence was looked for, and the appendix, presenting itself, was drawn through a small slit in the iliac region, where it was anchored. Irrigation with salt solution was performed through it. The median wound was closed in layers. The appendix sloughed off in two days and both wounds healed uneventfully, leaving no sinus.

The patient was discharged cured July 5, 1911, eighteen days after operation.

Surgical No. 86,746.—G. A. W., 8 months old, was admitted to the service of Dr. Robert Abbe (Division A), on the 30th of August, 1911. The chief

complaint was "a prolapse of the rectum." His illness began 3 months previous to admission, when what appeared to be a small piece of rectal mucosa protruded from the anal ring. There was no vomiting nor passage of blood. The "prolapse," which was at first small, gradually became larger. It could be apparently reduced. Later the stools contained mucus and much blood, still there was no vomiting nor visible distress. On admission, the child vomited once, but did not look ill.

Physical examination showed no rigidity nor tenderness of the abdomen. On the left side, extending from the brim of the pelvis to the left costal border, there was a hard sausage-shaped mass which was not tender. This was best felt when the "prolapse" was reduced. Protruding from the anus there was a large sausage-shaped mass consisting of bowel. The tissue was quite red and bled easily on handling. At the end of the mass there were two openings, one of which was blind, but admitted a probe for 2 inches, the other admitted a catheter indefinitely. This entire mass was easily reducible just within the sphincter ani, but came out immediately upon relaxing pressure.

On rectal examination, a firm ring could be felt as far as the finger reached, and the ring was much enlarged.

The child was operated upon the afternoon of September the 1st.

Operation (Dr. Green).—Through a median incision, the lower part of the ileum, the cæcum with appendix and ascending colon were found intussuscepted into the transverse and descending colon to an extent sufficient to allow the ileo-cæcal valve and the mouth of the appendix to present at the tip of the prolapsed tumor 5 inches outside of the anus (see picture). With gentle traction and pressure below the intussuscepted gut, it was quite easily reduced and found in a good and healthy condition. The appendix was identified and carried through a small stab wound made in the abdominal wall just above and in front of the anterior superior spine of the ilium. It was anchored here and the main wound closed. The child returned to the ward in fairly good condition, but at 11 o'clock that evening he suddenly became worse, and died ten minutes later.

Surgical No. 87,792.—M. H., 4 months old, was admitted to the service of Dr. Abbe, Division A, on December 6, 1911. In the early morning of the day of admission, the mother noticed that the child was restless, and refused to nurse. Five hours later it passed a bloody stool. The mother then sent for her family physician, who came that afternoon and sent the child immediately to the hospital. Upon examination, the patient presented the appearance of a fat, healthy baby. A typical sausage-shaped swelling existed, extending transversely across the abdomen just below the umbilicus. The diagnosis of an acute intussusception was made and at 5 o'clock in the afternoon she was operated upon.

Operation (Dr. Green).—Through a median incision extending from the umbilicus to the pubis, the bowel was reduced by careful squeezing with one hand and traction with the other. The intussusceptum proved to be the appendix, caput coli ileum and ascending colon in the order named. The intussuscipiens was the transverse colon. There was no evidence of any

Fig. 1.—Ileo-colic intussusception, showing protrusion of tumor, with ileo-cæcal valve at the tip.

Fig. 2.—Another view of protruding ileo-colic intussusception in Case. 2.

tendency to adhesion formation, nor was any lymph thrown out. Through a stab wound in the right iliac fossa the appendix was drawn out, and its mesentery and serosa anchored to the peritoneum, the appendix being placed between two wipes. The median wound was closed, using one suture for the peritoneum and closing the remaining layers with through and through silkworm gut.

The appendix sloughed away on the 5th day. The stump was closed by touching it from time to time with silver nitrite, and 4 weeks after operation the patient was discharged cured.

The child was chiefly breast-fed, after the operation, with the addition of a little extra feeding at the suggestion of Dr. Charles F. Collins, who kindly regulated the post-operative diet for a week.

MESENTERIC THROMBOSIS WITH RESECTION OF SIX FEET OF SMALL INTESTINE—RECOVERY.

NATHAN W. GREEN, M.D.

Surgical No. 85978.—W. A. H., American, school teacher, 43 years old, was admitted to the service of Surgical Division A, on the 19th of June, 1911. Her family history recorded the death of three brothers and one sister from tuberculosis. Two sisters were living and well. Her previous history was good, and she has had two children, the last eleven years ago. Both were well. One week previous to her admission, she ate something at a restaurant to which she attributed a diarrhœa, with cramps, which lasted two days in spite of castor oil (her elder boy, 14 years old, was also ill after eating the same). Three days previous to admission she passed, without accompanying pain, a black stool. After that she felt well until the morning of the day of admission, when at three o'clock she was awakened by a severe pain one inch to the left of the umbilicus. The pain did not change its position but became steadily worse. She then took an enema with but slight result, and collapsed. She had nausea, and after medication she vomited. The pain was so severe that a hypodermatic injection of morphine was required. Her physician, Dr. E. J. Richardson, came in the afternoon, and at once sent her to the hospital. The appearance was that of intestinal obstruction. On admission, her temperature was 99° F., pulse 106°, respiration 28. She appeared severely and acutely ill. Her abdomen was somewhat distended, with acute tenderness all over, but most severe near the midline. The distension was symmetrical, percussion tympanitic, but no mass was felt. She was operated upon 16 hours after her initial pain.

Operation (Dr. Green).—Resection of 5 feet 9 inches of ileum with end to end anastomosis with a Murphy button. A median incision was made, and on opening the peritoneal cavity, a litre of blood-stained fluid drained off. Lying more or less transversely from the left iliac-fossa to the ileo-cæcal region was a coil of gut 1½ feet which was of a reddish-black color, with 4 feet of a dark red color. There was no lymph exudate observable. The intestinal border of the mesenteric fan attached to the impaired gut was thickened, red and dusky. There was no angulation nor volvulus. The affected portion of gut, together with the thickened part of the mesentery, was cut away about 4 inches each side of the line of demarkation. But as there was not satisfactory bleeding from the remaining ends, more was removed until sharp arterial bleeding was encountered. The mesentery was ligated with an interrupted chain ligature, and the ends of the gut were

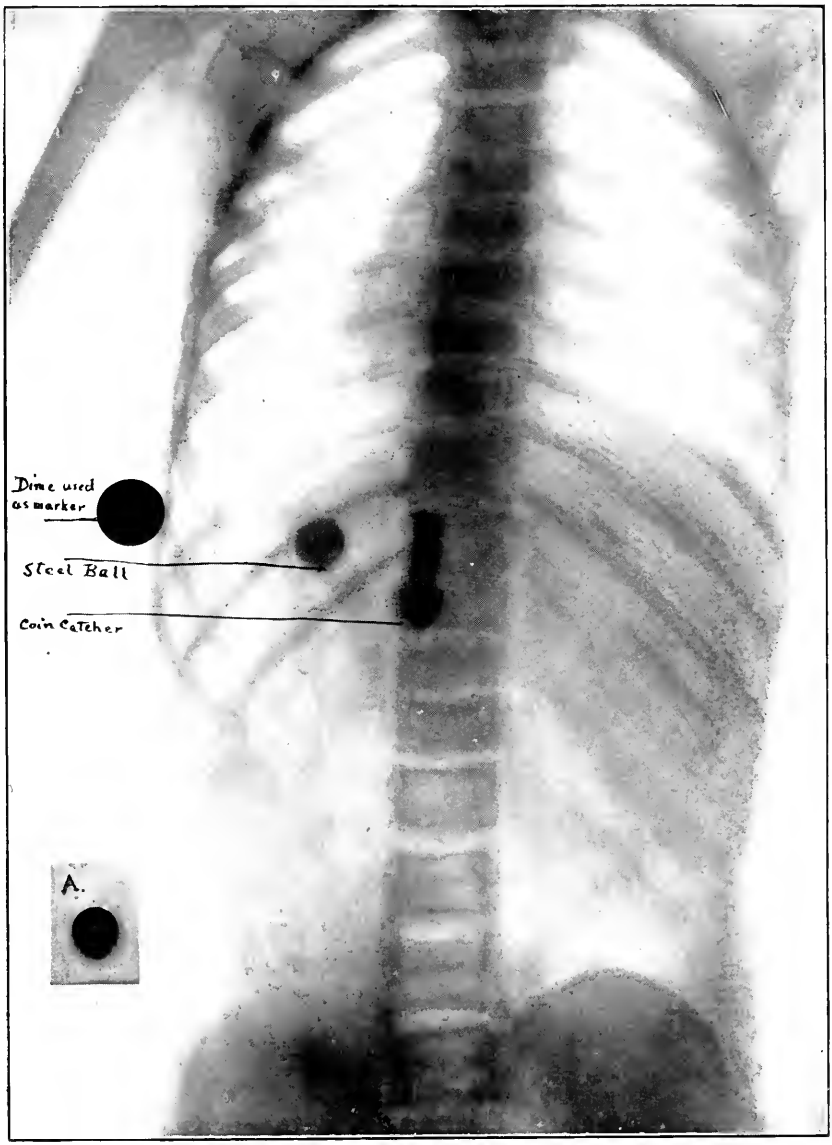


Fig. 2.—X-ray, showing steel ball displaced into stomach, with coin catcher at its side. This picture has been reversed in printing. A.—Picture of steel ball, ½ inch in diameter, which caused esophageal obstruction.

united with a Murphy Button reinforced by a Cushing Stitch. The peritoneum was washed with a Blake Tube. Drainage to the site of anastomosis was instituted and the wound closed with, through and through, silk worm gut sutures.

As deliberation in testing the ends of the remaining gut was used before anastomosis, the time of the operation lasted nearly an hour. The first day after operation her temperature rose to 100 4-5° F., pulse, 156; respiration, 28. Her highest post-operative temperature was 102 2-5 on the 14th day after operation, and was due to an accumulation of pus which then escaped through the drainage sinus. She had an intercurrent apical bronchitis, of rather severe type, and her sinus discharged for some weeks. An X-ray taken July 18, 1911, to locate the Murphy button, showed it in the pelvis (Fig. 1). It passed a few days later.

The patient has gained 35 pounds since her discharge from the hospital on August 29, 1911, and has been for several months discharging her duties as a teacher. She reported, on February 1st, that she was as well as she ever was.[1]

The section of intestine (Fig. 2), when freshly removed, measured 5 feet 9 inches, without traction upon it. Later, when it was received in the Pathological Department, the length was reported as 2½ meters. The pathological report is as follows: "Specimen consists of 2½ meters of small intestine, with mesentery attached. The diameter of the bowel is about normal. The greater portion is of a purplish-red color, and the surface has lost its normal gloss. Ten centimeters from the anterior end is a definite line of demarkation, on the proximal side of which the intestine appears normal. Immediately beyond this point, it assumes a dark-colored appearance, which gradually becomes less marked. At a point about 50 centimeters from the distal end is a less distinct line of demarkation, but even beyond this the intestine is much congested. The mesentery contains much fat, and the veins are distinctly dilated.

"Microscopical examination of a section taken from near the root of the mesentery shows a large vein occluded by a thrombus, in which are a number of fibro blasts indicating beginning organization. There is some hemorrhage into the areolar tissue, but no marked inflammatory reaction. A section of the mesentery, made nearer the intestine, shows a smaller vein with a thrombus attached to its wall on one side, which does not, however, completely block the vessel, and shows no signs of organization. The tissues about the vessel contain many red blood cells and a few leucocytes. In other areas, both arteries and veins are free from thrombi.

"A section of the intestinal wall shows no remains of epithelial lining. The villi and muscularis are densely packed with blood cells, which almost completely hide the connective tissue framework and the muscle. The nuclei of the muscle and connective tissue cells stain very faintly. The small blood-vessels beneath the peritoneum are free from thrombi."

[1]For the first two or three months after her resection she suffered from a diarrhœa, or, rather, a frequency of defecation. This has corrected itself, and now she is normal in this respect.

The striking appearance at operation, the lack of both arterial and venous bleeding on sectioning the gut and its mesentery and the findings in the Pathological Report all warrant the diagnosis of Mesenteric Thrombosis. The clinical picture was almost a facsimile of Dr. W. S. Schley's case, reported before the New York Surgical Society in 1911, and which is on record in the Medical and Surgical Reports of St. Luke's Hospital for 1910.

PAPILLOMA OF THE BLADDER TREATED BY EXCISION—RECURRENCE TREATED WITH RADIUM AND THE HIGH FREQUENCY CURRENT.

HENRY G. BUGBEE, M.D.

Prior to 1910, tumors of the bladder were treated by one method—excision. In the *Journal of the American Medical Association* for May 28, 1910, Dr. Edwin Beer, of New York, described a new method of treating papillomata of the bladder by the high frequency current. At that time he reported 2 cases so treated with excellent result.

Since then, Keyes, in the *American Journal of Surgery,* July, 1910; Buerger and Wolborst, *New York Medical Journal,* October 27, 1910, and McCarthy, have reported cases which have confirmed Beer's observations.

Beer's second report, *Annals of Surgery,* August, 1911, gives a more detailed account of his early cases, and he adds 3 more. He comments as follows: "From all of these observations (references above), based on the application of the high frequency treatment as used in some 38 papillary growths, it must be evident to the most sceptical that in this new method we have raised a mighty rival to the older suprapubic and to the transperitoneal and operative cystoscopic methods. I believe it will supplant previous methods, because of its greater simplicity and its great effectiveness."

The case which I wish to report is that of a patient, 56 years of age, who has been under the care of Dr. Robert Abbe since 1903, and which I have had the pleasure of studying in conjunction with him, for the past 2 months.

The record of the case is as follows:

There is nothing of note in the patient's history until 1903. He had always enjoyed good health, was of large frame, well nourished. Eight years ago he began to notice a slight irritation in the bladder and a faint, bloody tinge to the urine. Urination became more frequent, was accompanied by slight burning, but no pain or actual distress, and the stream had good volume and force. An X-ray examination was made with negative result.

This condition prevailed until July, 1905, when a cystoscopic examination

by Dr. Abbe revealed a papilloma of the bladder. The growth was benign, villous, pedunculated, the size of a hen's egg, located above and slightly posterior to the right ureter in the Bas-fond. This Dr. Abbe removed through a suprapubic opening. Not only was the growth removed, but a wide excision of the bladder mucous membrane made about the pedicle. The convalescence was rapid, the wound closing at once, and the patient was well until 1907, when blood again appeared in the urine. Cystoscopy revealed a recurrence of the growth at its former site, i.e., above and posterior to the right ureteral orifice. This recurrence was a tumor of the same characteristics as the original growth, but smaller (about the size of a walnut). An application of a radium tube, bound to a probe, was made to the growth through a direct cystoscope, by Dr. Abbe and Dr. F. Tilden Brown. The tumor disappeared rapidly and the patient was free from symtoms for 1 year. In 1908 he again had hematuria, but a cystoscopic examination by Dr. Abbe showed no growth. The blood disappeared after administering gallic acid.

The following year, a return of the hematuria led him to consult Dr. Charles A. Powers, of Denver, near which city he was then residing, and Dr. Powers reported to Dr. Abbe on the case at that time, December 8, 1909, as follows:

Mr. J. S. B. consulted me December 6th, regarding a recent recurrence of bladder hemorrhage. I learn of your operation for the removal of a growth in June of 1905, of your application of radium in December, 1907, of the slight bleeding through the winter of 1907-08, this controlled by capsules of gallic acid. Also of your further examination and good report in December, 1908. Mr. B. seems to have gone on without definite symptoms until a return of hemorrhage during this past month. Of this he will doubtless give you a detailed history. He consulted me in order to ascertain, if possible, whether it is now best for you to see him in New York. I told him that I could give no opinion without a complete cystoscopic examination, and this was made yesterday morning.

Mr. B. presents no symptoms whatever, excepting hematuria. He has no evidence of cystitis, he does not arise at night to urinate, the bladder capacity is good. Urination is not painful, there is no residual urine, there is only occasional slight staining of the clothing from the meatus. The prostate is but very slightly enlarged, its consistency is good, it is not tender.

A cystoscopic examination was made by Dr. Lyons and myself yesterday morning. The bladder wall presents a typically healthy appearance. We did not learn the site of your operation, but there seemed to be evidence of a scar a little above and to the right of the base of the trigone. Just back of the trigone and about in the midline, there is a reddish area less than one-half inch in diameter. This area is not ulcerated. It was not bleeding at the time of the examination, even when rubbed with the end of the cystoscope.

There was a slight hemorrhage coming from the right side of the prostatic urethra, little flakes of blood fell from this area, and the membrane

here was a bit raised. I judge that all portions of the bladder were thoroughly examined; at the end of the 35 minutes the bladder solution was not at all discolored.

Dr. Lyons and I think it probable that the bleeding comes from the prostatic urethra. Urine was seen coming from the orifice of the left ureter; we could not be certain of this on the right side. The ureters were not catheterized. The patient has no kidney symptoms.

Mr. B. is in excellent general condition; his weight and strength are good; he presents no other symptoms than the hematuria. He will send this letter to you, and you will advise him. The condition seems to be splendid, in view of the removal of a growth in 1905.

The hematuria was again controlled by gallic acid, and did not again appear until June, 1911, when a slight tinging of the urine was noticed by the patient. This color became deeper, and he again consulted Dr. Powers, who cystoscoped the patient, with Dr. Lyons. Their report, June 25, 1911, is as follows:

The bladder held with little or no pain about 12 ounces of fluid; the right ureteral opening was found presenting a normal appearance, also the superior posterior wall of the bladder and the trigone; the left ureter was found a little puffy and edematous, blood was noticed coming from around the opening, but on passing a catheter, the urine from the left kidney was found to be clear; there was found situated a little above and to the outside of the left ureteral opening a smooth, white, heavily stocked growth, the size of a hickory nut, a slight congestion and edema of the bladder wall surrounded the tumor, but no indurations could be detected.

From June 25, 1911, to October 17, 1911, the patient passed blood very frequently. There was no pain accompanying urination, or at other times, but an irritation and sensation of an incomplete emptying of the bladder.

I saw the patient with Dr. Abbe on October 17, 1911. He was then in excellent general health. There was slight frequency of urination, a good stream, no pain, and but slight irritation about the vesical neck. There was a tinge of blood in the last of the urine passed. Rectal examination showed enlarged prostate, but the remainder of the physical examination was negative.

A cystoscopic examination was made, with the following results:

The cystoscope entered the bladder without difficulty or discomfort to the patient. The urine evacuated from the bladder was pale and clear. There was no blood in it, in contradistinction to that passed by voluntary urination, where the last contraction of the bladder, in emptying itself, caused a very slight hemorrhage. The bladder was filled and held 10 ounces of fluid without discomfort. An examination of the mucous membrane of the bladder showed a smooth, grayish, glistening surface, throughout the fundus, with the exception of 2 areas. The blood vessels were slightly congested. The

first of the 2 areas above mentioned was located posteriorly and to the outer side of the right ureteral orifice. This area, about 1 cm. in diameter, was paler than the surrounding mucous membrane, devoid of blood vessels, and resembled scar tissue. This was apparently the site of the original growth, and the first recurrence. The second abnormal area was in a similar position on the opposite side of the bladder. Here was found a growth about 2½ x 1½ cm. in size. The growth was grayish in color, villous, and fairly solid in appearance. On first sight, it appeared to surround the left ureteral orifice, but a pedicle was later observed. The mucous membrane about the growth was edematous. The urine coming from the ureteral orifice was clear. The growth could be made to bleed by touching it with the cystoscope.

The prostate gland was moderately enlarged, and the vessels on its surface congested. The right ureteral orifice was normal in appearance and functionated regularly, clear urine being emitted.

The area of the trigone was negative.

October 18, 1911.—Through an indirect catheterizing cystoscope the insulated wire from the high frequency machine was passed into the bladder and made to impinge the growth. Four applications of 30 seconds each were made to the growth, which became charred and gray and rapidly disintegrated. The operation gave the patient no pain. Following the application the urine was tinged with blood for 3 days, but there was no discomfort.

November 3, 1911.—Cystoscopic examination showed the central area of the growth destroyed, and a gray spot, 1 cm. by 1 cm., to the outer and posterior aspect of the left ureter. Above and below this spot was a small nodule of growth, soft, villous, and not bleeding. The mucous membrane about the ureteral orifice was edematous. Clear urine came from the opening. Remainder of the bladder as when first examined.

November 16, 1911.—There is much less edema of the mucous membrane about the site of the growth. A soft, villous, reddish growth the size of a pea has appeared high up on the left lateral wall of the bladder since the last cystoscopy. The high frequency current was applied to this and to the small nodules at the margin of the left ureter, for 1½ minutes each.

November 20, 1911.—Cystoscopy showed that the new growth on the lateral wall, high up, had disappeared. One of the nodules about the left ureteral orifice had disappeared, and the second was so small as to be scarcely visible. There was less surrounding edema than after the first application of the current.

November 27, 1911.—Cystoscope passed for the purpose of making one more application of the high frequency current, but examination showed that the bladder wall was everywhere normal, no vestige of a growth being visible.

December 3, 1911.—Patient states that he has been perfectly comfortable, and left for his home in the West.

This case is reported primarily to show the results of the treatment of the tumor with the high frequency current.

Two applications, an aggregate of 3½ minutes of contact with the current, destroyed the growth, first seen about the left ureteral orifice.

One application of 1½ minutes destroyed the small growth on the lateral wall.

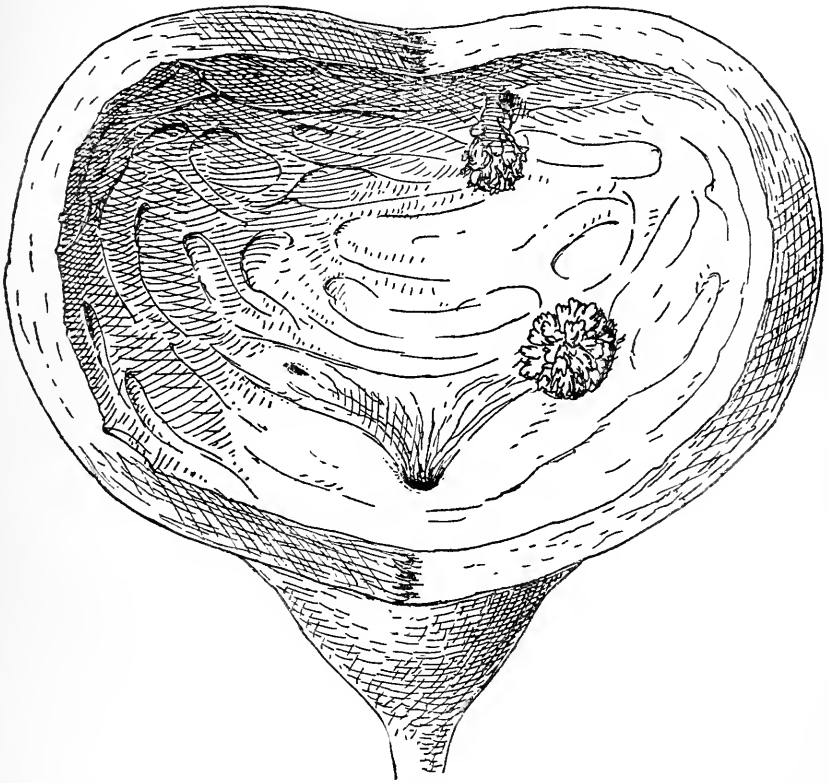


Fig. 1.—Cross-section of bladder, showing position of recurrence; also small growth in fundus, which appeared between treatments, and was destroyed by one application of the high-frequency current.

The treatment was painless, and followed by no uncomfortable or serious consequences. There was no resulting ulceration, and but slight bleeding after the application of the current.

This method of treatment of benign papillomata is simpler than any other, and in this case, as in others reported, is quite as effectual. There is no reason to believe that the growth will not recur, but recurrence is the rule after removal by any method.

BILATERAL STRICTURE OF THE URETERS.

HENRY G. BUGBEE, M.D.

Mrs. H., 33 years, married. Family and past history to 1897 negative. At this time she was operated upon for double pyosalpinx and both tubes and ovaries were removed. Following this operation she developed dull pains in the lumbar region of the back on either side and severe pain in the pelvic region. In 1907 she was operated upon for pelvic adhesions and a second operation for the same cause was performed later in the year. There was little relief from the lumbar pains.

In 1908 a third operation for a pelvic tumor. The pain in the lumbar region has continued. Urination has been more frequent by day; once or twice at night. Slight burning at the end of urination. No blood or cloud in urine.

November 1, 1911. Chief Complaints.—Frequency of urination, pain in each lumbar region of the back.

Physical Examination.—Medium frame. Well nourished. Good color. Chest, negative. Abdomen: There is a scar 4 inches long in the lower, median line of the abdomen. Also one 2 inches to either side of it. There is a slight bulging of the median scar when the patient coughs. Each kidney can be palpated, is tender, but not perceptibly enlarged. Liver and Spleen: not felt; no masses or other points of tenderness.

Vaginal Examination.—Uterus normal size and position. Analysis of 24-hour specimen of urine was negative. Cystoscopic examination shows a normal bladder.

The right ureteral orifice was slightly edematous, the left normal. Each shows very slight contraction when functionating. No swirl of urine could be observed coming from either.

A catheter entered the orifice on either side, but was arrested 3 cm. from the bladder on the left side and 4½ cm. from the bladder on the right side. Other catheters were substituted, but none would advance beyond these points of constriction. A filiform was passed through the constriction and on to either kidney. The feeling conveyed by the filiform was that of gripping rather than encountering an obstruction. The condition was that of a stricture of each ureter.

The catheters placed in either ureter, low down, allowed a separation of the urine from the kidneys. The dropping of the urine was not in four or five drops, then a pause, but was very slow and regular. The urine was clear.

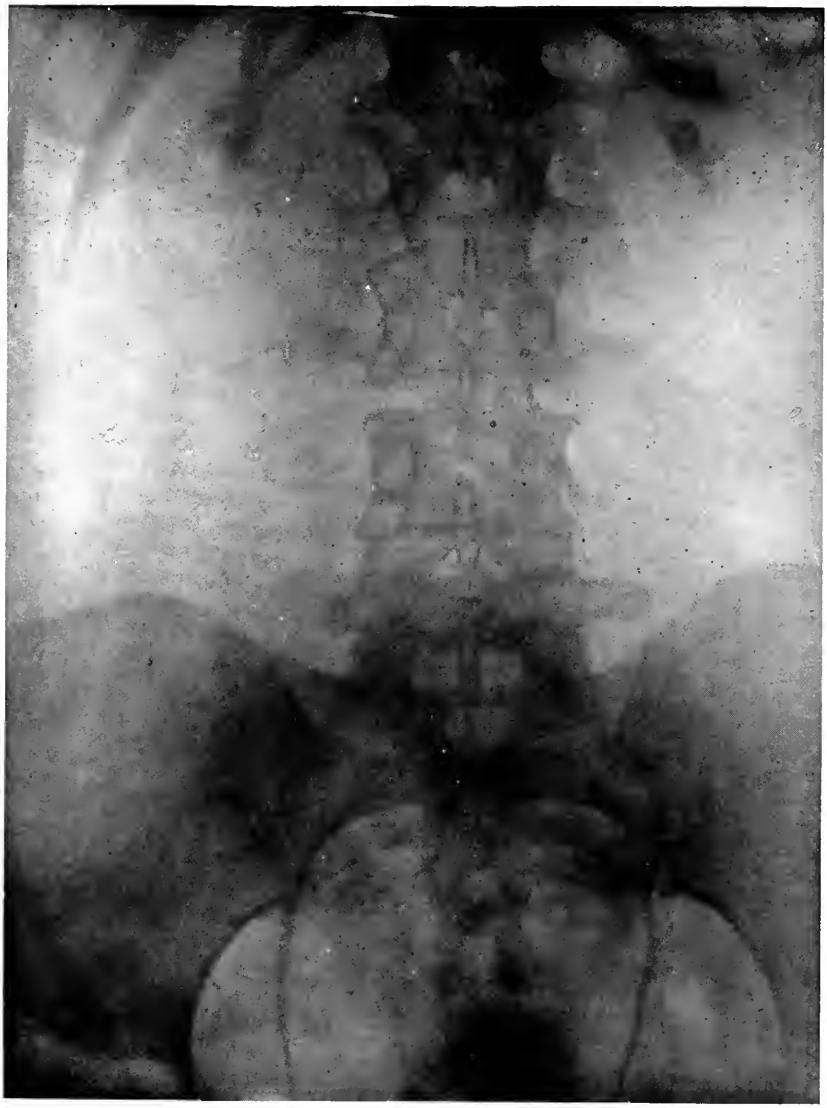


Fig. 1.—The ureteral catheters show the point of constriction in either ureter. The inability to distend fully the pelves and calices of the kidneys is also shown.

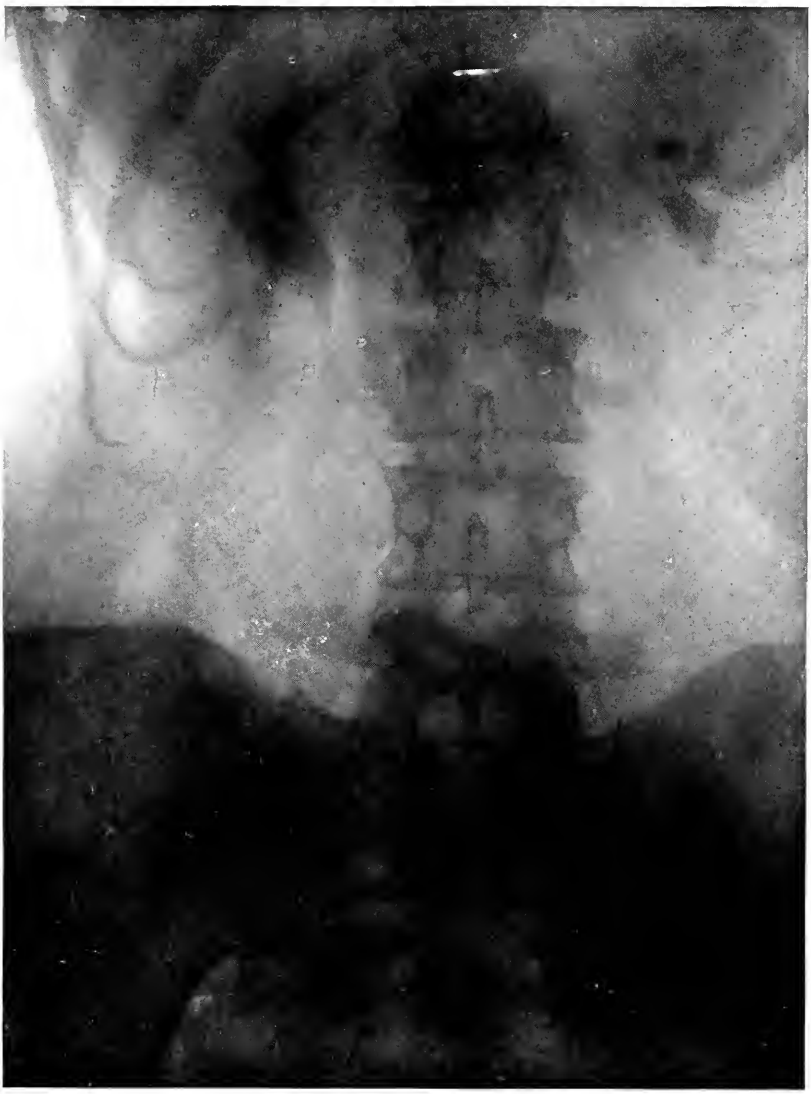


Fig. 2.—The ureteral catheter on the right side is seen, having passed through the stricture. The left ureteral catheter is not clearly defined, but both kidneys are distended. The left kidney, pelvis and calices are dilated.

Following the examination, the patient had a sharp attack of pain in either kidney region, resembling renal colic. This lasted several hours, and subsided.

November 14, 1911.—The cystoscope was again introduced and an attempt made to catheterize the ureters, but the same obstruction was encountered. Filiforms were passed as before, followed by olivary bougies, sizes 2 and 3 F. There was no pain following the stretching, but some relief of the old pain in the back with less frequency of urination.

November 23, 1911.—It was possible to pass a No. 5 F. catheter through the constriction of the right ureter and the flow of urine from the catheter was rapid. The catheter was obstructed 3 cm. from the bladder on the left side. Dilatation was carried out as on the previous occasions.

December 1, 1911.—Catheters were passed up the right ureter to the pelvic brim, and in the left but 3 cm. Argyrol injections of the ureters and kidneys were made (40% Argyrol in 2% boric acid). Six c.c. in the right and 4 c.c. in the left. There was slight pain in either kidney following the injection. The patient stated that this pain was exactly like the pain which she had previously suffered. X-ray pictures were taken which show the point of stricture in either ureter, that in the left being lower down. There is a slight dilatation of the pelvis of the left kidney.

December 12, 1911.—It is possible to pass a No. 5 F. catheter to either kidney. Patient has very little of the old pain and but slight frequency of urination.

December 19, 1911.—Ureters again dilated and catheters passed. No. 5 F. catheters to both kidneys.

December 27, 1911.—Catheters passed beyond stricture of either ureter and Argyrol injections made. The Argyrol passed to the kidneys easily, allowed a dilatation of the kidney pelvis, and the patient had a slight renal colic on either side.

The X-ray photograph shows the ureters to be open throughout their extent and a dilatation of the pelvis of the left kidney.

The condition is stricture of either ureter, caused by an inflammation in the ureteral wall, probably from the same cause as the tubal infection. It is analogous to a urethral stricture and may be treated in the same manner. A relief of the urinary symptoms and diminution of the renal pain have followed the dilatation of the strictures, and show the possibilities of conservative treatment. This dilatation can, in all probability, be carried up to No. 8 or 10 F., and the stretching done less frequently.

The process of slow dilatation has given the patient no pain or inconvenience, and seems to be justified in every case where a stricture can be diagnosed.

Kelly, in the Journal of American Medical Association, August 16,

1902, reports several cases treated in this manner. The symptoms in his cases were similar to the above. He states that the diagnosis is seldom made, and that the majority of cases are treated for hydronephrosis. Strictures are rarely bilateral, usually found in the pelvic ureter, and are caused most frequently by a gonorrhœal or tubercular inflammation.

The symptoms are those of vesical and renal inflammation. The ureters can often be palpated through the vaginal wall.

The ureteral orifice, through the cystoscope, appears swollen, may be ulcerated, the opening is often obscured, may be a dimple, or indicated only by radiating lines.

Urine usually flows freely after passing a catheter through the stricture.

Kelly has found dilatation the ideal treatment, except in tuberculosis.

Other methods of treatment are ureteral catheterization and irrigation, freeing of adhesions, resection of the ureter, extirpation of the tract, and transplantation.

Medical Service

DISEASES DUE TO MICRO-ORGANISMS INFECTIVE DISEASES	Sex		Results				Totals
	Male	Female	Cured	Improved	Unimproved	Died	
Cerebrospinal fever	....	2	2	....	....	....	2
Diphtheria	1	2	1	1	1	....	3
Dysentery (amœbic)	2	....	1	....	1	....	2
Dysentery (amœbic), bronchitis, polycythemia	1	....	....	1	....	....	1
Enteric fever, intestinal hemorrhage	1	....	....	....	....	1	1
Erysipelas	1	3	2	2	....	....	4
Filiariasis, chyluria	1	....	....	....	1	....	1
Gonococcus arthritis of elbow	....	3	....	2	1	....	3
Gonococcus arthritis of knee	1	5	1	5	....	....	6
Gonococcus arthritis, pregnancy	....	1	....	1	....	....	1
Gonococcus arthritis, urethritis	1	....	....	1	....	....	1
Influenza	7	29	35	1	....	....	36
Influenza, otitis externa	....	1	1	....	....	....	1
Malaria	6	2	8	....	....	....	8
Malaria (tertian)	2	2	4	....	....	....	4
Rheumatism (subac. artic.)	6	3	3	6	....	....	9
Rheumatism (subac. artic.), cardiac arrhythmia	....	2	....	2	....	....	2
Rheumatism (subac. artic.), mitral insufficiency	....	6	1	5	....	....	6
Rheumatism (subac. artic.), retroversion of uterus, nephritis, cardiac hypertrophy	....	1	....	1	....	....	1
Rheumatism (subac. artic.), strongyloides intestinalis	1	....	....	1	....	....	1
Rheumatism (ac. art.)	14	17	24	7	....	....	31
Rheumatism (ac. art.), bronchitis, emphysema, nephritis, uremia	1	....	....	1	....	....	1
Rheumatism (ac. art.), carcinoma of gall bladder	....	1	....	1	....	....	1
Rheumatism (ac. art.), herpes zoster	1	....	....	1	....	....	1
Rheumatism (ac. art.), lymphangitis	....	1	....	1	....	....	1
Rheumatism (ac. art.), mitral and aortic insufficiency	....	4	2	2	....	....	4
Rheumatism (ac. art.), mitral insufficiency	3	4	2	5	....	....	7
Rheumatism (ac. art.), mitral stenosis, fibrinous pericarditis	....	1	....	....	....	1	1
Rheumatism (ac. art.), nephritis	....	1	....	1	....	....	1
Rheumatism (ac. art.), pericarditis	1	1	....	2	....	....	2
Rheumatism (ac. art.), pericarditis (fibrinous), lobar pneumonia	....	1	....	....	....	1	1
Rheumatism (ac. art.), regurgitation (mitral), pericarditis (fibrinous)	....	1	1	....	....	....	1
Syphilis (secondary)	1	....	....	1	....	....	1
Syphilis (secondary), multiple alcoholic neuritis	....	1	....	1	....	....	1
Syphilis (tertiary)	2	3	1	3	1	....	5
Syphilis (tertiary), aortic aneurysm	1	....	....	1	....	....	1
Syphilis (tertiary), aortic insuff., aortitis	1	....	....	1	....	....	1
Syphilis (tertiary), aortitis, mitral and aortic insuff., cardiac decompensation	1	....	....	....	....	1	1
Syphilis (tertiary), aortitis, tabes dorsalis, chr. nephritis, cirrhosis of liver	....	1	....	1	....	....	1
Syphilis (tertiary), cerebral endarteritis, thrombosis	....	1	....	1	....	....	1
Syphilis (tertiary), gumma of post-pharyngeal walls, keratitis	....	1	....	1	....	....	1
Syphilis (tertiary), gumma of spinal cord	2	....	....	2	....	....	2
Syphilis (tertiary), hepatitis	2	....	....	2	....	....	2

DISEASES DUE TO MICRO-ORGANISMS—Continued	Male	Fem.	C.	Imp.	Un.	Died	Tot.
Infective Diseases—Cont.							
Syphilis (tertiary), hepatitis, aneurysm of arch of aorta	1	....	....	1	....	....	1
Syphilis (tertiary), periosteitis of cranium	....	1	1	....	....	....	1
Syphilis (tertiary), periosteitis of femur	1	....	....	1	....	....	1
Syphilis (cerebral)	2	....	....	2	....	....	2
Syphilis (cerebral), hemorrhage into cerebrum	1	....	....	1	....	....	1
Syphilis (cerebral), lobar pneumonia	....	1	....	....	....	1	1
Tuberculosis of axillary glands, tbc. fibrosis of lungs and pleuræ	1	....	....	....	1	....	1
Tbc. of chest wall	....	1	....	1	....	....	1
Tbc. of kidney	....	2	....	2	....	....	2
Tbc. of kidney and bladder	....	1	....	1	....	....	1
Tbc. of knee joint	....	1	....	....	1	....	1
Tbc. of lungs	24	37	....	34	9	18	61
Tbc. of lungs, arterio-sclerosis, chr. nephritis	1	....	....	....	....	1	1
Tbc. of lungs, bronchitis	1	1	....	2	....	....	2
Tbc. of lungs, diabetes mellitus, hydropneumothorax, gangrene of lung	1	....	....	....	....	1	1
Tbc. of lung, gastritis, cirrhosis of liver	....	1	....	1	....	....	1
Tbc. of lungs, lobar pneumonia	....	1	....	....	....	1	1
Tbc. of lungs, ischio-rectal abscess	1	....	....	1	....	....	1
Tbc. of lungs, nephritis	1	1	....	1	1	....	2
Tbc. of lungs, pernicious anemia, arterio-sclerosis	1	1	....	1	....	1	2
Tbc. of lungs, pleurisy with effusion	4	2	....	3	2	1	6
Tbc. of lungs, pneumothorax	1	1	....	2	....	....	2
Tbc. of lungs, tbc. enteritis	2	4	....	....	....	6	6
Tbc. of lungs, tbc. fistula in ano	1	....	....	1	....	....	1
Tbc. of lungs, tbc. of larynx	1	1	....	1	....	1	2
Tbc. of lungs, tbc. of larynx, aortic regurgitation	1	....	....	....	....	1	1
Tbc. of lungs, tbc. meningitis	1	....	....	....	....	1	1
Tbc. of lungs, tbc. of spine, bronchitis, fibrinous pleurisy	1	....	....	1	....	....	1
Tbc. of lungs, tbc. of spine, tachycardia	....	1	....	....	....	1	1
Tbc. of hip	1	....	....	....	1	....	1
Tbc. of peritoneum	....	2	....	....	2	....	2
Tbc. of pleura	3	3	2	4	....	....	6
Tbc. of spine	1	....	....	1	....	....	1
Tbc. of spine, tbc. of lungs, tachycardia	....	1	....	1	....	....	1
Tbc. meningitis	4	....	....	....	....	4	4
Tbc. meningitis, chr. nephritis	1	....	....	....	....	1	1
Tuberculosis (miliary), tbc. peritonitis, nephritis	1	....	....	....	....	1	1
Typhoid fever	36	29	61	....	1	3	65
Typhoid fever with hemorrhages	2	1	1	....	....	2	3
Typhoid fever with relapse	1	4	5	....	....	....	5
Typhoid fever, bronchitis	1	1	1	....	....	1	2
Typhoid fever, broncho-pneumonia	1	1	1	....	....	1	2
Typhoid fever, ischio-rectal abscess	....	1	1	....	....	....	1
Typhoid fever, laryngitis (ac.)	....	1	1	....	....	....	1
Typhoid fever, mitral and aortic insuff	1	....	1	....	....	....	1
Typhoid fever, otitis media	3	1	3	1	....	....	4
Typhoid fever, periosteitis	1	....	1	....	....	....	1
Typhoid fever, phlebitis	....	1	1	....	....	....	1
Typhoid fever, pulmonary embolism	....	1	....	....	....	1	1
Typhoid fever, pneumothorax, cholecystitis	....	1	1	....	....	....	1
Typhoid fever, pyelitis	....	1	1	....	....	....	1
Typhoid fever, peritonitis, ac. catarrhal cholecystitis	1	....	1	....	....	....	1
Typhoid fever (para-typhoid)	....	1	1	....	....	....	1
Typhoid meningitis	....	1	....	....	....	1	1
	166	189	171	128	23	53	375
ALIMENTARY SYSTEM							
INTESTINES							
Colitis	....	1	....	1	....	....	1
Colitis (ulcerative)	....	5	1	1	3	....	5
Colitis (ulcerative), bronchitis	....	1	....	....	....	1	1
Colitis (ulcerative), ethmoiditis	....	1	....	....	....	1	1
Constipation, arterio-sclerosis	....	1	1	....	....	....	1
Constipation, mitral stenosis and insuff., aortic insuff	1	....	1	....	....	....	1

ALIMENTARY SYSTEM—Continued	Male	Fem.	C.	Imp.	Un.	Died	Tot.
Intestines—Cont.							
Diarrhœa	1	....	1	....	....	....	1
Duodenal ulcer	1	2	1	2	....	....	3
Entero-colitis	....	1	1	....	....	....	1
Enteroptosis, hyperchlorhydria, pyloric stenosis	....	1	....	....	1	....	1
Enteroptosis, retroversion of uterus, constipation (chr.)	....	1	....	....	1	....	1
Gastro-enteritis	2	1	2	1	....	....	3
Ileus	1	....	....	1	....	....	1
Ileus, aortic and mitral insuff., aortic stenosis	1	....	....	1	....	....	1
Ileus, nephritis, uremia	1	....	....	....	....	1	1
Jejunal ulcer	....	1	....	....	1	....	1
	8	16	8	7	6	3	24
LIVER							
Abscess of liver	1	1	....	....	2	....	2
Abscess of liver, empyema of gall bladder	....	1	....	1	....	....	1
Cirrhosis of liver	5	5	....	6	1	3	10
Cirrhosis of liver, alcoholic peripheral neuritis	....	4	1	3	....	....	4
Cirrhosis of liver, alcoholic psychosis	1	....	....	1	....	....	1
Cirrhosis of liver, arthritis (ac.), ascites, myocarditis	1	....	....	1	....	....	1
Cirrhosis of liver, ascites, cardiac dilatation, nephritis	....	1	....	1	....	....	1
Cirrhosis of liver, catarrhal gastritis	1	1	....	2	....	....	2
Cirrhosis of liver, hematemesis	1	....	....	1	....	....	1
Cirrhosis of liver, hemorrhoids, mitral insuff	....	1	....	1	....	....	1
Cirrhosis of liver, fistula in abdominal wall	....	1	....	1	....	....	1
Cirrhosis of liver, intestinal hemorrhages, alcoholic delirium	1	....	....	1	....	....	1
Cirrhosis of liver, Korsikoff's psychosis	....	1	....	....	1	....	1
Cirrhosis of liver, mitral regurgitation	....	1	....	1	....	....	1
Cirrhosis of liver (atrophic), emphysema	....	1	....	1	....	....	1
Cirrhosis of liver (hepatic), secondary anemia	1	....	....	1	....	....	1
	12	18	1	22	4	3	30
BILE PASSAGES							
Catarrhal jaundice	....	1	1	....	....	....	1
Cholangitis	1	....	1	....	....	....	1
Cholelithiasis	1	4	....	5	....	....	5
Cholelithiasis, biliary colic	....	1	....	1	....	....	1
Cholelithiasis, cholecystitis	2	1	....	2	1	....	3
Cholelithiasis, goiter, hypochlorhydria	....	1	....	1	....	....	1
Stenosis of bile duct (congenital), icterus	....	1	....	....	....	1	1
	4	9	2	9	1	1	13
MOUTH, TEETH AND GUMS							
Alveolar abscess	....	1	1	....	....	....	1
Pyorrhea alveolaris	1	....	....	1	....	....	1
Stomatitis (mercurial), ac. nephritis	1	....	....	1	....	....	1
Ulceration of tooth	....	1	1	....	....	....	1
	2	2	2	2	....	....	4
ŒSOPHAGUS							
Œsophagodynia	1	....	....	1	....	....	1
	1	....	....	1	....	....	1
PANCREAS							
Pancreatic cyst, cirrhosis of liver	1	....	....	1	....	....	1
	1	....	....	1	....	....	1
PERITONEUM, ETC.							
Inguinal hernia, angina pectoris	....	1	....	....	1	....	1
Peritoneal adhesions, hysterical mania	....	1	....	1	....	....	1
Subphrenic abscess	1	....	....	....	1	....	1
	1	2	....	1	2	....	3

ALIMENTARY SYSTEM—Continued	Male	Fem.	C.	Imp.	Un.	Died	Tot.
PHARYNX, TONSILS AND NASOPHARYNX							
Abscess (peritonsillar.)	3	3	6	....	....	....	6
Pharyngitis	2	....	2	....	....	....	2
Quinzy	....	1	....	1	....	....	1
Tonsillitis	2	7	9	....	....	....	9
Tonsillitis, pharyngitis	1	....	1	....	....	....	1
Tonsillitis (follicular)	11	6	17	....	....	....	17
Tonsillitis (follicular), stomatitis	1	....	1	....	....	....	1
Tonsils (hypertrophied), phimosis, sciatica	1	....	....	....	1	....	1
	21	17	36	1	1	....	38
RECTUM							
Fecal fistula, mitral and aortic insufficiency	1	....	....	1	....	....	1
Hemorrhoids, epididymo-orchitis, empyema of tunica vaginalis	1	....	....	1	....	....	1
Hemorrhoids (internal)	2	....	....	2	....	....	2
	4	....	....	4	....	....	4
STOMACH							
Anacidity, fracture of rib	1	....	1	....	....	....	1
Atony, hyperchlorhydria, senile dementia	....	1	....	1	....	....	1
Dilatation	....	1	....	1	....	....	1
Dyspepsia	6	1	7	....	....	....	7
Dyspepsia (nervous)	1	1	....	2	....	....	2
Dyspepsia (nervous), anacidity	....	1	....	1	....	....	1
Gastritis (alcoholic	2	1	3	....	....	....	3
Gastritis (alcoholic), catarrhal jaundice, mitral and aortic insufficiency	1	....	....	1	....	....	1
Gastritis (alcoholic), multiple neuritis, tbc. of face	....	1	....	1	....	....	1
Gastritis (acute.)	....	6	5	1	....	....	6
Gastritis (acute), mitral stenosis	....	1	1	....	....	....	1
Gastritis (chronic)	2	8	1	8	1	....	10
Gastritis (chronic), hyperacidity	....	1	....	1	....	....	1
Gastritis (chronic), neurasthenia	....	1	....	1	....	....	1
Gastroptosis	1	1	....	2	....	....	2
Hyperchlorhydria	2	....	....	2	....	....	2
Hyperchlorhydria, gastritis	1	1	1	1	....	....	2
Hyperchlorhydria, gastroptosis	....	1	....	....	1	....	1
Hypochlorhydria	....	1	....	1	....	....	1
Hypochlorhydria, insomnia	1	....	....	1	....	....	1
Hypochlorhydria, senile atrophy of stomach	1	....	....	1	....	....	1
Stenosis of pylorus of stomach	1	1	....	1	1	....	2
Stenosis of pylorus of stomach, gastric dilatation, cardio-spasm	....	1	....	1	....	....	1
Ulcer of stomach	2	7	3	5	1	....	9
Ulcer of stomach, cystic kidney	....	1	....	1	....	....	1
Ulcer of stomach, pyloric stenosis	1	....	....	1	....	....	1
	23	38	22	35	4	....	61
VERMIFORM APPENDIX							
Appendicitis (chronic)	....	3	....	2	1	....	3
Appendicitis (chr.), catarrhal gastritis	....	1	....	1	....	....	1
Appendicitis (acute) with abscess	1	1	....	1	1	....	2
Appendicitis (acute) with peritonitis	1	....	....	....	1	....	1
Appendicular colic	....	1	1	....	....	....	1
	2	6	1	4	3	....	8
CARDIO-VASCULAR SYSTEM							
BLOOD							
Anemia (pernicious.)	2	1	....	2	....	1	3
Anemia (secondary)	....	1	....	1	....	....	1
Anemia (secondary), nervous exhaustion	....	1	....	1	....	....	1
Anemia (simple)	....	1	....	1	....	....	1
Anemia (splenic)	....	1	....	1	....	....	1
Chlorosis	....	3	1	2	....	....	3
Chlorosis, influenza, otitis media	....	1	....	1	....	....	1
Chlorosis, mitral stenosis and insufficiency	....	1	....	....	....	1	1
Leukemia (lymphatic)	....	1	....	1	....	....	1

CARDIO-VASCULAR SYSTEM—Continued	Male	Fem.	C.	Imp.	Un.	Died	Tot.
Blood—Cont.							
Leukemia (myelogenous)	1	....	....	1	....	....	1
Leukemia (lymphatic), herpes zoster, broncho-pneumonia, mitral insufficiency	....	1	....	....	....	1	1
Pseudoleukemia, suppuration of axillary glands.	....	1	....	1	....	....	1
	3	13	1	12	....	3	16
ARTERIES							
Aneurysm of aorta	5	1	....	3	1	2	6
Aneurysm of aorta, aortitis, aortic insufficiency, arterio-sclerosis, ac. art. rheumatism	1	....	....	....	....	1	1
Aneurysm of aorta, cholelithiasis, nephritis	....	1	....	....	....	1	1
Aneurysm of aorta (ruptured), arterio-sclerosis nephritis	1	....	....	....	....	1	1
Aneurysm of aorta (ruptured), broncho-pneumonia, emphysema	1	....	....	....	....	1	1
Aneurysm of iliac and femoral arteries	1	....	....	....	1	....	1
Aneurysm of innominate artery	1	1	....	....	1	....	1
Arterio-sclerosis	1	1	....	2	....	....	2
Aneurysm of aorta (ruptured), hemothorax, sero-fibrinous pleurisy, lobar pneumonia	....	1	....	....	....	1	1
Arterio-sclerosis, bronchitis (acute)	1	....	....	1	....	....	1
Arterio-sclerosis, constipation (chr.)	2	....	....	2	....	....	2
Arterio-sclerosis, emphysema, myocarditis	2	....	....	1	....	1	2
Arterio-sclerosis, emphysema, senility	1	1	....	2	....	....	2
Thrombosis of cervical arteries, paralysis of pharynx	....	1	....	....	....	1	1
Ventricular hemorrhage	....	1	....	....	....	1	1
Embolism (cerebral), rheumatic endocarditis	1	....	....	....	....	1	1
Embolism (coronary), mitral and aortic insuff. mitral stenosis	....	1	....	....	....	1	1
	18	8	....	11	3	12	26
VEINS							
Thrombosis of innominate vein, pyelitis	....	1	1	....	....	....	1
Thrombosis of popliteal vein, varicose veins of legs	....	1	1	....	....	....	1
	....	2	2	....	....	....	2
HEART							
Angina pectoris (?), mitral and aortic insuff.	1	....	....	1	....	....	1
Dilatation, cardiac hypertrophy, mitral and aortic insuff., mitral stenosis	4	....	....	4	....	....	4
Dilatation, mitral stenosis, aortic insuff.	2	....	....	2	....	....	2
Endocarditis (chr.)	1	1	....	2	....	....	2
Dilatation (acute), hydrothorax, pneumonia	....	1	....	....	....	1	1
Endocarditis (septic)	1	2	....	....	....	3	3
Endocarditis (septic), cerebral embolism	....	1	....	....	....	1	1
Endocarditis (septic), emphysema, mitral stenosis and insuff.	1	....	....	....	....	1	1
Endocarditis (septic), mitral stenosis, aortic insuff., sero-fib. pleurisy	....	1	....	1	....	....	1
Endocarditis (septic), mitral and aortic insuff.	1	....	....	1	....	....	1
Endocarditis (rheumatic), aortic regurgitation	....	1	....	1	....	....	1
Endocarditis (rheumatic), pregnancy	....	1	....	....	....	1	1
Endocarditis (rheumatic), terminal pneumonia	1	1	....	....	....	2	2
Fatty heart, sclerosis of coronary arteries	1	....	....	1	....	....	1
Myocardial degeneration, arterio-sclerosis, chronic nephritis	1	....	....	1	....	....	1
Myocardial degeneration, fatty degeneration of liver	....	1	....	....	....	1	1
Myocarditis	1	5	1	4	....	1	6
Myocarditis, aortic stenosis	1	....	....	....	....	1	1
Myocarditis, arterio-sclerosis, emphysema	2	1	....	3	....	....	3
Myocarditis, arterio-sclerosis, hydrothorax	1	....	....	1	....	....	1
Myocarditis, decompensation	2	2	....	1	....	3	4
Myocarditis, endocarditis, angina pectoris	1	....	....	1	....	....	1
Myocarditis, nephritis	2	....	....	2	....	....	2
Myocarditis, osteo-arthritis of hip	....	1	....	1	....	....	1
Myocarditis, polycythemia, cardiac insuff.	1	....	....	....	....	1	1

CARDIO-VASCULAR SYSTEM—Continued	Male	Fem.	C.	Imp.	Un.	Died	Tot.
Heart—Cont.							
Valvular Diseases:							
Mitral insufficiency	3	5	....	7	....	1	8
Mitral insuff., artic. rheumatism	....	1	....	1	....	....	1
Mitral insuff., hydrothorax, nephritis	2	....	....	1	....	1	2
Mitral insuff., œdema of lungs	....	1	....	....	....	1	1
Mitral insuff. and stenosis	1	2	....	3	....	....	3
Mitral insuff. and stenosis, aortic insuff	2	7	....	4	2	3	9
Mitral insuff. and sten. arterio-sclerosis, chr. nephritis	1	3	....	3	....	1	4
Mitral insuff. and sten., aortic stenosis	2	2	....	2	....	2	4
Mitral insuff. and sten., decompensation	1	....	....	....	....	1	1
Mitral insuff. and sten., pleurisy	1	....	....	1	....	....	1
Mitral and aortic insufficiency	7	....	....	5	....	2	7
Mitral and aortic insuff., fibrinous pleurisy, inguinal hernia	1	....	....	1	....	....	1
Mitral and aortic insuff., rheumatic endocarditis	1	....	....	....	....	1	1
Mitral and aortic insuff. and stenosis	2	2	....	3	....	1	4
Mitral and tricuspid insuff., mitral stenosis	....	2	....	1	....	1	2
Mitral stenosis	1	2	1	2	....	....	3
Mitral stenosis and aortic insuff	1	2	....	3	....	....	3
Mitral stenosis, decompensation, anasarca	1	....	....	....	....	1	1
Aortic insufficiency, aortitis, hemorrhoids	1	....	....	....	1	....	1
Aortic insuff., aortitis, angina pectoris	1	....	....	1	....	....	1
Aortic insuff. and stenosis, decompensation	1	....	....	....	....	1	1
Aortic stenosis	3	1	....	1	2	1	4
	58	49	2	66	5	34	107
LYMPH GLANDS							
Ac. lymphangitis, lymphadenitis, tenosynovitis	....	1	1	....	....	....	1
Supp. lymphadenitis of axilla	1	....	....	1	....	....	1
	1	1	1	1	....	....	2
DUCTLESS GLANDS							
Goitre (simple), hyperthyroidism, mitral stenosis and regurgitation	....	1	....	1	....	....	1
Goitre (exophthalmic)	....	4	....	4	....	....	4
Goitre (exophthalmic), cardiac hypertrophy and dilatation, pneumothorax	....	1	....	....	....	1	1
Elephantiasis, abscess of leg	....	1	....	1	....	....	1
Hodgkin's disease	3	....	....	1	....	2	3
Toxemic hyperthyroidism	....	1	1	....	....	....	1
	3	8	1	7	....	3	11
MUSCULAR SYSTEM							
Chr. muscular rheumatism	1	6	1	6	....	....	7
Myalgia	1	1	2	....	....	....	2
Progressive muscular atrophy	1	....	....	1	....	....	1
	3	7	3	7	....	....	10
NERVOUS SYSTEM							
BRAIN							
Abscess, supp. meningitis, polycythemia	1	....	....	....	1	....	1
Apoplexy	2	2	1	1	....	2	4
Abscess of brain, otitis media, mitral stenosis	....	1	....	....	1	....	1
Apoplexy, cerebral thrombosis	....	2	....	1	....	1	2
Apoplexy, hemiplegia	....	2	....	2	....	....	2
Meningitis, bulbar paralysis, pulmonary tbc	1	....	....	....	....	1	1
Meningitis (pneumococcus)	1	....	....	....	....	1	1
Paralysis (facial), hyp. tonsils	....	1	....	1	....	....	1
Paralysis (post-diphtheritic)	....	1	1	....	....	....	1
	5	9	2	5	2	5	14
DISEASES OF THE MIND							
Mania (acute), bronchitis, emphysema, mitral insuff	....	1	....	....	1	....	1

NERVOUS SYSTEM—Continued	Male	Fem.	C.	Imp.	Un.	Died	Tot.
Diseases of the Mind—Cont.							
Melancholia	....	1	....	....	1	....	1
Paranoia	....	1	....	....	1	....	1
Paresis (general)	1	....	....	1	....	....	1
	1	3	....	1	3	....	4
NERVES							
Neuralgia of cranial nerve	....	2	....	2	....	....	2
Neuralgia of intercostal nerve	2	....	2	....	....	....	2
Neuralgia of sciatic nerve	4	3	....	7	....	....	7
Neuritis (alcoholic multiple), Korsikoff's syndrome, pulmonary tbc	1	....	1	....	....	....	1
Neuritis (brachial)	1	....	1	....	....	....	1
Neuritis (multiple)	....	2	1	1	....	....	2
Neuritis (multiple), pulmonary tbc	....	2	1	....	1	....	2
Neuritis (peripheral)	1	3	2	1	1	....	4
Psychasthenia	....	1	....	1	....	....	1
Zoster	1	4	5	....	....	....	5
	10	17	13	12	2	....	27
NERVOUS DISEASES OF UNKNOWN ORIGIN							
Chorea	3	2	3	2	....	....	5
Claustrophobia	....	1	....	1	....	....	1
Epilepsy (petit mal)	....	3	....	3	....	....	3
Hysteria	....	9	1	8	....	....	9
Nervous exhaustion	1	2	1	2	....	....	3
Neurasthenia	2	15	....	13	4	....	17
Neurasthenia, retroversion of uterus	....	1	....	1	....	....	1
Neurosis (gastric)	1	....	....	1	....	....	1
	7	33	5	31	4	....	40
SPINAL CORD							
Hematomyelia	....	1	....	1	....	....	1
Myelitis (transverse)	1	....	....	....	1	....	1
Poliomyelitis (ac. anterior)	3	1	....	2	1	1	4
Tabes dorsalis, gen. paresis, gastric crisis	1	....	....	1	....	....	1
Tabes dorsalis, rheumatism	1	....	....	1	....	....	1
	6	2	....	5	2	1	8
OSSEOUS SYSTEM							
BONES							
Curvature of spine	1	....	....	1	....	....	1
Leontiasis ossia, Paget's disease, mitral insuff	....	2	....	2	....	....	2
Sacro-iliac disease	....	1	....	1	....	....	1
Spondylitis deformans	....	1	....	1	....	....	1
	1	4	....	5	....	....	5
JOINTS							
Arthritis deformans	....	5	....	5	....	....	5
Chronic articular rheumatism, nephritis	1	....	....	1	....	....	1
Osteo-arthritis of hip	....	1	....	1	....	....	1
	1	6	....	7	....	....	7
REPRODUCTIVE SYSTEM							
OVARIES AND TUBES							
Salpingitis (acute)	....	2	....	1	1	....	2
	....	2	....	1	1	....	2
PREGNANCY							
Pregnancy, acidosis	....	1	....	1	....	....	1
Vomiting of pregnancy	....	3	3	....	....	....	3
	....	4	3	1	....	....	4

REPRODUCTIVE SYSTEM—Continued	Male	Fem.	C.	Imp.	Un.	Died	Tot.
UTERUS AND FALLOPIAN TUBES							
Dysmenorrhea	....	3	....	3	....	....	3
Endometritis, ac. bronchitis	....	1	....	1	....	....	1
Menorrhagia	....	2	1	1	....	....	2
Pyosalpinx	....	1	....	1	....	....	1
Pyosalpinx, appendicitis	....	1	....	1	....	....	1
Pyosalpinx, fibroma uteri, broncho-pneumonia, fibrinous pleurisy	....	1	....	....	1	....	1
Retention of placenta, septic uterus	....	1	....	....	1	....	1
	....	10	1	7	2	....	10
RESPIRATORY SYSTEM							
BRONCHI							
Asthma, bronchitis	....	4	2	2	....	....	4
Asthma, emphysema	....	1	....	1	....	....	1
Asthma, erysipelas	3	2	1	4	....	....	5
Asthma, nephritis (chr.)	1	....	....	....	1	....	1
Bronchiectasis	1	....	....	....	1	....	1
Bronchitis	3	13	12	4	....	....	16
Bronchitis (ac.), cardiac incompetency	....	1	....	....	....	1	1
Bronchitis (ac.), dextracardia	....	1	1	....	....	....	1
Bronchitis (ac.), emphysema	2	1	....	3	....	....	3
Bronchitis (ac.), mitral insufficiency	1	....	1	....	....	....	1
Bronchitis (ac.), mitral regurgitation, purpura rheumatica	....	1	....	1	....	....	1
Bronchitis (ac.), myelogenous leukemia	1	....	....	1	....	....	1
Bronchitis (ac.), strongyloides intestinalis	1	....	....	1	....	....	1
Coryza	1	1	2	....	....	....	2
	14	25	19	17	2	1	39
LARYNX							
Laryngitis	....	2	2	....	....	....	2
Laryngitis (catarrhal), fibromyomata uteri, neurasthenia	....	1	....	1	....	....	1
	....	3	2	1	....	....	3
LUNGS							
Abscess of lung and gangrene, following lobar pneumonia	....	1	....	....	1	....	1
Emphysema (pulmonary), bronchitis	....	3	....	3	....	....	3
Emphysema (pul.), bronchitis, asthma, paroxysmal tachycardia	....	1	....	1	....	....	1
Emphysema (pul.), bronchitis, mitral insuff.	....	1	....	1	....	....	1
Pneumonia (broncho-)	2	4	4	....	....	2	6
Pneumonia (broncho-), arterio-sclerosis, chr. nephritis	....	1	....	....	....	1	1
Pneumonia (broncho-), varicose ulcer	....	2	1	....	....	1	2
Pneumonia (lobar)	32	12	29	....	....	15	44
Pneumonia lobar), arterio-sclerosis, chr. nephritis	1	....	....	....	....	1	1
Pneumonia (lobar), alcoholic delirium	2	....	1	1	....	....	2
Pneumonia (lobar), articular rheumatism, mitral insuff., fibrinous pericarditis	....	1	1	....	....	....	1
Pneumonia lobar), atresia of lung	1	1	2	....	....	....	2
Pneumonia (lobar), bronchitis, emphysema	....	2	....	2	....	....	2
Pneumonia (lobar), fibrinous pleurisy, childbirth, dilatation of stomach	1	....	....	....	....	1	1
Pneumonia (lobar), fibrinous pericarditis, infarct of lung, mitral insufficiency	....	1	....	1	....	....	1
Pneumonia (lobar), mitral insufficiency	1	....	....	....	....	1	1
Pneumonia (lobar), mitral insufficiency, infarct of lung	....	1	....	....	....	1	1
Pneumonia (lobar), œdema of lungs	....	4	1	....	....	3	4
Pneumonia (lobar), œdema of lungs, myocarditis, chr. nephritis	....	1	....	....	....	1	1
Pneumonia (lobar), morphinism	1	1	2	....	....	....	2
Pneumonia (lobar), nephritis, hydrothorax	....	2	....	....	....	2	2
Pneumonia (lobar), pleurisy (dry)	1	....	1	....	....	....	1
Pneumonia (lobar), pleurisy (suppurative)	2	6	4	4	....	....	8

RESPIRATORY SYSTEM—Continued	Male	Fem.	C.	Imp.	Un.	Died	Tot.
Lungs—Cont.							
Pneumonia (lobar), pleurisy with effusion	2	1	3	....	....	....	3
Pneumonia (lobar), streptococcic bacteremia	....	1	....	....	....	1	1
Pneumonia (lobar, resolution delayed), fibrinous pleurisy	1	1	2	....	....	....	2
Pneumonia (lobar, unresolved)	....	1	1	....	....	....	1
Pneumonia (terminal), chr. nephritis	....	1	....	....	....	1	1
	47	50	52	13	1	31	97
PLEURA							
Pleurisy (dry)	....	1	1	....	....	....	1
Pleurisy (dry), pneumonia	....	1	....	1	....	....	1
Pleurisy (fibrinous)	3	5	4	3	....	1	8
Pleurisy (fibrinous), tachycardia	....	1	....	1	....	....	1
Pleurisy (sero-fibrinous), retroversion, endometritis	....	1	....	1	....	....	1
Pleurisy (suppurative)	3	2	1	1	3	....	5
Pleurisy with effusion	16	5	15	6	....	....	21
Pleurisy with effusion, asthma, bronchitis	....	1	1	....	....	....	1
Pyopneumothorax	1	....	....	....	1	....	1
	23	17	22	13	4	1	40
SENSE ORGANS							
ORGAN OF HEARING							
Mastoiditis	....	2	1	1	....	....	2
Mastoiditis, influenza, otitis media	....	1	....	....	1	....	1
Otitis media (suppurative)	1	....	1	....	....	....	1
	1	4	2	1	2	....	5
ORGAN OF VISION							
Atrophy of optic nerve	....	2	....	....	2	....	2
Cataract, diabetes	1	....	....	1	....	....	1
Choroido-retinitis	....	1	....	....	1	....	1
Keratitis	2	....	....	1	1	....	2
Strabismus	1	....	....	1	....	....	1
	4	3	....	3	4	....	7
TEGUMENTARY SYSTEM							
SKIN, ETC.							
Erythema multiforme	....	1	1	....	....	....	1
	....	1	1	....	....	....	1
URINARY SYSTEM							
KIDNEY							
Albuminaria	1	....	....	1	....	....	1
Nephritis (acute)	1	....	1	....	....	....	1
Nephritis (chronic interstitial)	9	20	19	....	....	10	29
Nephritis (chr. in.), anemia	1	1	....	2	....	....	2
Nephritis (chr. in.), appendicitis	1	....	....	....	1	....	1
Nephritis (chr. in.), arterio-sclerosis	3	1	....	3	1	....	4
Nephritis (chr. in.), arthritis deformans	1	....	....	....	1	....	1
Nephritis (chr. in.), anemia, stomatitis	1	....	....	....	....	1	1
Nephritis (chr. in.), aortic insufficiency	2	....	....	2	....	....	2
Nephritis (chr. in.), cirrhosis of liver	3	....	....	3	....	....	3
Nephritis (chr. in.), cirrhosis of liver, cerebral hemorrhage	1	....	....	....	....	1	1
Nephritis (chr. in.), cirrhosis of liver, parotitis, pericarditis, fib. pleurisy	1	....	....	....	....	1	1
Nephritis (chr. in.), cystitis, dilatation, myocardial insufficiency	....	1	....	1	....	....	1
Nephritis (chr. in.), colitis, aortitis, rheumatoid arthritis, diphtheria	1	....	....	1	....	....	1
Nephritis (chr. in.), dry pleurisy, cirrhosis of liver	1	....	....	1	....	....	1

URINARY SYSTEM—Continued	Male	Fem.	C.	Imp.	Un.	Died	Tot.
Kidney—Cont.							
Nephritis (chr. in.), emphysema, cardiac insufficiency	....	1	....	1	....	....	1
Nephritis (chr. in.), hydrothorax	1	....	....	1	....	....	1
Nephritis (chr. in.), mitral stenosis and insufficiency	4	6	....	4	6	....	10
Nephritis (chr. in.), myocarditis	2	....	....	1	1	....	2
Nephritis (chr. in.), peritonitis	1	....	....	1	....	....	1
Nephritis (chr. in.), œdema of lungs, lobar pneumonia, myocarditis	1	....	....	....	....	1	1
Nephritis (acute), parotitis, status lymphaticus	1	....	....	....	....	1	1
Nephritis (chr. in.), pleurisy with effusion, cardiac insufficiency	1	1	....	1	....	1	2
Nephritis (chr. in.), pulmonary hemorrhages, entero-colitis, uremia	1	....	....	....	....	1	1
Nephritis (chr. in.), pulmonary tbc., cirrhosis of liver	1	....	....	1	....	....	1
Nephritis (chr. in.), mitral regurgitation, hemiplegia, motor hysteria	....	1	....	1	....	....	1
Nephritis (chr. in.), uremia	4	7	....	4	....	7	11
Nephritis (chr. in.), uremia, fib. pericarditis	1	1	....	....	....	2	2
Nephritis (chr. in.), uremia, mitral and aortic insuff.	....	1	....	1	....	....	1
Nephritis (chr. in. with acute exacerbation)	1	....	....	....	....	1	1
Nephritis (chr. parenchymatous)	1	....	....	....	....	1	1
Nephritis (sub-acute), facial paralysis	....	1	....	....	....	1	1
Nephritis (sub-acute), hemianopsia papillitis	....	1	....	....	....	1	1
Nephritis sub-acute) hemorrhage into pons	....	2	....	....	....	2	2
Hydronephrosis	1	....	....	1	....	....	1
Nephrolithiasis	1	....	....	....	1	....	1
Pyelonephritis	....	2	1	1	....	....	2
Pyelitis	3	5	6	2	....	....	8
Pyonephrosis, broncho-pneumonia	....	1	....	1	....	....	1
Uremia	1	2	....	2	....	1	3
Uremia apoplexy	1	....	....	....	....	1	1
	54	55	27	37	11	34	109
SPLEEN							
Enlarged spleen, polycythemia	....	1	....	1	....	....	1
	....	1	....	1	....	....	1
URINARY BLADDER							
Cystitis, nephritis, cardiac decompensation	1	....	....	1	....	....	1
Cystitis, urethral stricture, catheter fever	1	....	....	1	....	....	1
	2	....	....	2	....	....	2
DISEASES DUE TO ANIMAL PARASITES							
Tapeworm	....	2	2	....	....	....	2
Unciniarisis	1	....	1	....	....	....	1
	1	2	3	....	....	....	3
GENERAL INJURIES							
Heat prostration	1	1	2	....	....	....	2
Smoke inhalation	1	....	1	....	....	....	1
Starvation, psychosis, chr. colitis	....	1	....	....	....	1	1
	2	2	3	....	....	1	4
LOCAL INJURIES							
Concussion	1	....	1	....	....	....	1
Incised wound of hand	....	1	1	....	....	....	1
	1	1	2	....	....	....	2
NEOPLASMS							
Carcinomatosis	1	2	....	....	....	3	3
Carcinoma of breast, mediastinal metastases	....	1	....	1	....	....	1
Carcinoma of breast, mitral regurgitation, subacute rheumatism	....	1	....	1	....	....	1

NEOPLASMS—Continued	Male	Fem.	C.	Imp.	Un.	Died	Tot.
Neoplasms—Cont.							
Carcinoma of colon	1	....	....	....	1	....	1
Carcinoma of liver and bile duct	....	3	....	....	1	2	3
Carcinoma of œsophagus	....	1	....	....	1	....	1
Carcinoma of pancreas	....	1	....	....	....	1	1
Carcinoma of stomach	1	2	....	....	1	2	3
Carcinoma of stomach and rectum, chr. nephritis	1	....	....	....	....	1	1
Carcinoma of stomach, metastases in liver	1	....	....	....	....	1	1
Carcinoma of stomach, omental metastases, tabes dorsalis, arterio-sclerosis	2	....	....	....	2	....	2
Carcinoma of uterus, anasarca, secondary anemia, cardiac insufficiency	....	1	....	....	....	1	1
Cyst-adenoma (papillary), metastases in lung	....	1	....	....	1	....	1
Epithelioma of œsophagus	1	....	....	....	1	....	1
Epithelioma of pleura, hydrothorax	....	1	....	....	1	....	1
Epithelioma of scalp, chr. nephritis, arterio-sclerosis	....	1	....	....	....	1	1
Exostoses on humerus	1	....	....	1	....	....	1
Fibromyoma of uterus, anemia	....	2	....	2	....	....	2
Infected arm	....	1	1	....	....	....	1
Infected finger	....	1	1	....	....	....	1
Sarcoma (retroperitoneal)	....	1	....	....	1	....	1
Sarcomata in lumbar region	1	....	....	....	....	1	1
Tumor of brain	1	2	....	....	3	....	3
Tumor of brain, pulmonary tuberculosis	....	1	....	1	....	....	1
Tumor of mediastinum	1	....	....	....	1	....	1
Tumor of abdomen	1	....	....	....	1	....	1
Tumor of spinal cord	1	....	....	....	1	....	1
	14	23	2	6	16	13	37
INTOXICATIONS AND POISONS							
EXOGENOUS INTOXICATIONS							
Alcoholism, acute	5	1	4	2	....	....	6
Alcoholism (chr.) delirium tremens	2	....	....	....	1	1	2
Alcoholism (chr.), Korsikoff's psychosis	....	1	....	1	....	....	1
Chronic poisoning by lead	4	....	2	2	....	....	4
Chronic poisoning by mercury	1	1	1	....	....	1	2
Chronic poisoning by morphine	2	1	1	1	1	....	3
Chronic poisoning by morphine, aneurysm of iliac and femoral arteries	1	....	....	....	1	....	1
Chronic poisoning by veronal	1	....	....	1	....	....	1
Chronic poisoning by strychnine	....	1	1	....	....	....	1
Acute poisoning by Paris green	1	....	....	....	....	1	1
Pneumococcus septicemia, pulmonary tbc	....	1	....	....	....	1	1
Septicemia following abortion, mitral insuff	....	1	....	1	....	....	1
Streptococcic septicemia, imitative meningitis	1	....	....	....	....	1	1
Streptococcic septicemia, septic endocarditis	....	1	....	....	....	1	1
	18	8	9	8	3	6	26
ENDOGENOUS INTOXICATIONS							
Auto-intoxication	1	1	1	1	....	....	2
Auto-intoxication, cardiac arrhythmia	....	1	....	1	....	....	1
Diabetes mellitus	2	3	....	5	....	....	5
Diabetes mellitus, chr. nephritis	....	3	1	2	....	....	3
Diabetes mellitus, pulmonary tbc	2	....	....	2	....	....	2
Diabetes mellitus, eczema	....	1	....	1	....	....	1
Diabetes mellitus, lobar pneumonia	....	1	....	....	....	1	1
Gout	3	....	1	2	....	....	3
	8	10	3	14	....	1	18
MISCELLANEOUS CONDITIONS							
Debility	....	1	1	....	....	....	1
Heat stroke, meningitis	1	....	....	....	....	1	1
Mallngering	....	1	....	....	1	....	1
No diagnosis made	3	7	....	2	8	....	10
	4	9	1	2	9	1	13

SUMMARY	Male	Fem.	C.	Imp.	Un.	Died	Tot.
Micro-organic Diseases	166	189	171	128	23	53	375
Alimentary System	76	102	71	80	18	7	178
Cardio-vascular System	80	73	6	90	8	49	153
Ductless Glands	3	8	1	7	....	3	11
Muscular System	3	7	3	7	....	....	10
Nervous System	29	64	20	54	13	6	93
Osseous System	2	10	....	12	....	....	12
Reproductive System	....	16	1	11	4	....	16
Respiratory System	84	95	95	44	7	33	179
Sense Organs	5	7	2	4	6	....	12
Tegumentary System	....	1	1	....	....	....	1
Urinary System	56	56	27	40	11	34	112
Animal Parasites	1	2	3	....	....	....	3
General Injuries	2	2	3	....	....	1	4
Local Injuries	1	1	2	....	....	....	2
Neoplasms	14	23	2	6	16	13	37
Intoxications	26	18	12	22	3	7	44
Miscellaneous	4	9	1	2	9	1	13

REPORT OF CASES OF HODGKIN'S DISEASE.

AUSTIN W. HOLLIS, M.D., OTTO H. LEBER, M.D., and F. C. WOOD, M.D.

Case 1.—The patient, a civil engineer, aged 31 years, came to the hospital September 5, 1910, with a general history of fever, and progressive loss of flesh and strength extending over a period of a whole year. In September, 1909, he had been taken sick with fever and general prostration, but stayed in bed for a few days only, and then was able to get up and go about his work until January 1. During all this time, however, he felt badly, and having acquired the habit of taking his temperature himself, frequently found that he had fever. He then spent 3 months at home, taking moderate exercise, but doing no work. At this time he was thought to have incipient tuberculosis, and in April went to a boarding-house in Sullivan County, and then, 4 weeks before admission, to the Loomis Sanatorium. He thought he had fever three-fourths of the time during the past year, but during the month of June there was a complete remission.

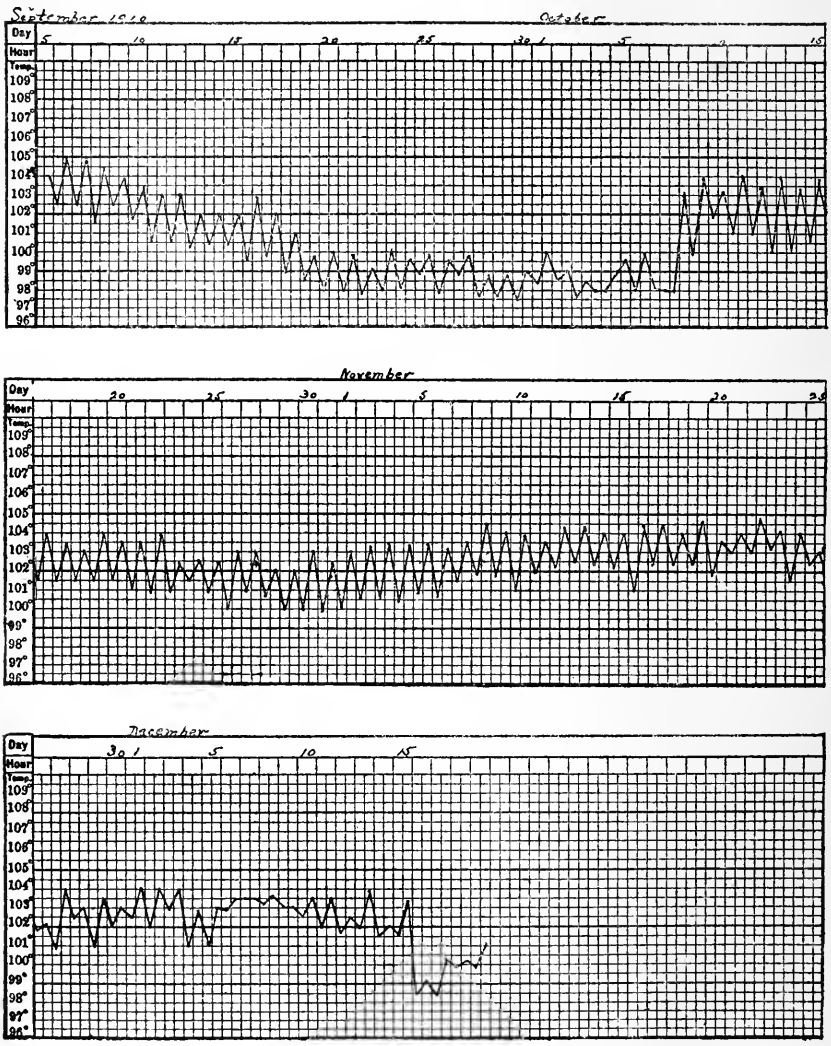
At the Loomis Sanatorium his temperature ranged from 100.8° to 105°. According to the patient, he had a moderate cough at the times he had fever, and occasionally expectorated a small quantity of whitish or yellowish sputum, which once contained a clot of blood. He thought he lost about 20 pounds in weight. He had a number of heavy night sweats in the spring, and had them almost nightly when admitted to the hospital. His appetite was poor when he had fever; he had no epigastric pain or symptoms of indigestion; the bowels had moved regularly with slight catharsis, and he was never troubled with pain anywhere.

His past history was negative, except for the diseases of childhood. His work in engineering has been in this part of the country, mostly in wet, swampy districts. He took a glass of beer occasionally, and smoked to excess before the onset of his illness.

There was no family history of tuberculosis. His mother died of carcinoma of the stomach; his father is alive and well.

On admission to the hospital, the patient was moderately prostrated, and markedly emaciated, the latter being accentuated by the patient's height of 6 feet 6 inches. The eyes, mouth and throat ap-

Case No. 1



peared normal; the chest showed very markedly the great degree of emaciation; expansion was good, and equal on both sides. The heart appeared normal in size, at the apex and over the pulmonic area there

was a soft systolic murmur. The action was regular, rapid and forcible. The lungs showed moderate dulness at both apices. At the right apex, and just below the spine of the scapula were a few subcrepitant râles. The abdomen was scaphoid; the liver appeared normal in size; the spleen descended about 2 fingers below the free border on inspiration. The right kidney could easily be felt. The extremities were negative. There were no signs of any glandular enlargement, superficial or deep. The temperature on admission ranged between 102 and 104 4/5, pulse about 100, and respirations 20 to 32. The blood count showed 3,300,000 red blood cells, with 35 per cent hemoglobin; white blood cells 3,400; polynuclear 46 per cent; leucocytes 54 per cent.

A blood culture, taken a short time after admission, showed what appeared to be a short chained streptococcus, and from this an autogenous vaccine was prepared, and the patient received increasing doses with what appeared to be excellent results, for in 2 weeks his temperature had gradually come down to normal, and remained there for almost 3 weeks, when, without any apparent cause, it jumped to 103, and thereafter remained above normal, ranging between 100° and 104°. The vaccine was discontinued shortly after, as 2 subsequent blood cultures remained sterile.

The course of the disease was uneventful, and marked only by progressive asthenia, anemia and emaciation, finally ending fatally 3½ months after admission, on December 18.

In view of the findings at autopsy, the symptom complex presented was peculiarly confusing, especially from the standpoint of the temperature course and the glandular involvement.

The temperature course in Hodgkin's disease can be very variable, as is well known, but has been classified by Longcope[1] into 3 more or less distinct types:

1. Mild and continuous, slightly irregular fever, varying a few degrees, rarely going above 101 or 102°.

2. Temperature quite irregular, light and intermittent; and

3. The relapsing type, a very unusual one, showing periods of pyrexia lasting several days or weeks, and alternating with similarly variable periods of apyrexia.

While this last type was observed as far back as 1870 by Murchison,[2]

[1]Longcope: Bull. Ayer Clin. Lab., No. 1, 1903; N. Y. Path. Soc., 1908, N. S. viii, 153.

[2]Murchison: Trans. of the Path. Soc. of London, 1870, xxi, 372.

and again by Pel[3] [4], in 1885, Ebstein[5] in 1887, Ruffin[6] in 1906, and by a few others, it will be seen from the scarcity of the cases that it is by far the most unusual type, so much so that Ebstein considered the peculiar temperature course sufficient ground for a separate classification, and called the disease "Chronic Relapsing Fever."

While the present case was under observation for only part of the course of the disease, there were apparently 3 periods of pyrexia, and 2 of apyrexia, the first of about a month, the second lasting almost 3 weeks.

The second and more unusual phase of this case was the distribution of the glandular involvement. At no time in the course of the disease was there any enlargement of lymph nodes palpable, and at no time was there any enlargement of the deeper nodes, either by direct evidence, or by secondary pressure signs. The patient never suffered from any pain which might have been taken as sign of pressure on any nerve. There was no sign of any pressure upon the larynx, trachea, bronchi or esophagus, and so also, no dyspnea or dysphagia. There was never any edema or evidence of ascites. By no physical signs in the chest could one assume the enlargement of any bronchial or mediastinal lymph nodes, and no amount of palpation could elicit any enlargement of the abdominal glands. There have been cases of Hodgkin's disease reported in which the deep glands were apparently the only ones involved,[7] but they seem quite regularly, at least in the later stages, to have given secondary signs of pressure, so notably absent here; and Reed,[8] in the comprehensive discussion of the disease, goes so far as to say, "We know of no case where the pathological anatomy was described in sufficient detail to permit of a positive diagnosis, in which the disease commenced elsewhere (than in the cervical region)."

The blood findings in our case were interesting. While at the Loomis Sanatorium, in August, he first had 4,640,000 red blood cells, and in 2 weeks actually gained 700,000. On September 6, he had 3,332,000, and 50 per cent hemoglobin. On September 23 he had 3,500,000, and 50 per cent hemoglobin. Thus, in spite of fever, he

[3]Pel: Berliner Klin. Wochenschrift, 1885, xxii.
[4]Pel: Berliner Klin. Wochenschrift, 1887, xxiv.
[5]Ebstein: Berliner Klin. Wochenschrift, 1887, xxiv.
[6]Ruffin: Am. Journ. Med. Sciences, 1906, cxxxi.
[7]Stall: Medical Record, N. Y., 1905, lxvii, 773.
[8]Reed: Johns Hopkins Hospital Reports, 1902, x, 133.

gained in red blood cells and hemoglobin, but this may have been from blood concentration, and not a true numerical gain of cells. The resistance of the blood and general strength under such a high fever was, however, striking. The digestive ability was always good. Leukopenia was a marked and constant feature, the leukocytes were never higher than 6,500, and more often were between 3,000 and 5,000 per cubic millimeter; the polynuclear and lymphocytes were variable, though in normal range proportions; eosinophyles were not present.

The clinical picture was extremely puzzling. Dr. O. D. Kingsley, of White Plains, who first treated him, thought of a tuberculous condition. The fever, night sweats and signs at the right apex at this period of his illness would seem fully convincing, but 8 months later, under the observation of Dr. H. M. King, at the Loomis Sanatorium, the diagnosis of pulmonary tuberculosis was abandoned, and he pointed out the necessity of investigation on other lines, and sent him to St. Luke's Hospital, with the suggestion that the spleen was at the bottom of it, and its removal might be considered. Under our investigation a short chained streptococcus was found once in blood culture, and an autogenous vaccine was employed with prompt remission of his fever, followed by general improvement, but a return of fever after three weeks' remission without subsequent confirmatory blood cultures, led us to believe that our previous positive culture was a contamination.

A few weeks before the patient's death, Dr. S. W. Lambert suggested the possibility of Ebstein's variety of Hodgkin's disease, but prominent clinicians, to the time of his death, were quite in doubt as to the true condition, and considered the probabilities of a cryptogenic septicæmia, chronic miliary tuberculosis and multiple sarcomatosis.

That such cases should be enigmas, is due in the first place, to their infrequency, but chiefly to the poor and meager description of their symptoms found in the text-books, which classify them as a variety of Hodgkin's disease without pointing out their wide divergence from the ordinary clinical picture of that disease. The number of cases reported with long febrile periods with more than one remission, show clearly that we are dealing with a specific affection running a very definite clinical course. In the two cases which I have seen, this one, and one in consultation with Dr. Everett W. Gould, periods of pyrexia and apyrexia alternated, in neither case were the super-

ficial lymphatic glands implicated, but moderate splenic enlargement, with some involvement of the abdominal and thoracic glands, was a feature in both cases.

This case resembles most strikingly, both in its relapsing temperature curve, as well as in the absence of any definable glands, the cases reported by Pel and Ebstein, in 1885 and 1887, one of which had as many as 9 periods of pyrexia, and which were considered by Pel to be pseudo leucemia, and by Ebstein, a new clinical entity.

PATHOLOGICAL REPORT BY F. C. WOOD, M.D.

The body is that of an emaciated young man of small frame. The skin shows a brownish tint. There is a decubitus ulcer over the sacrum. On the anterior wall, over the left costal cartilage, there is a small nodule partially invading the cartilage about 1.5 cm. in diameter. The cut surface is mottled with yellow and white areas. The left pleural cavity contains about 150 c.c. of clear, straw-colored fluid, the right about 100 c.c. There is an old fibrous adhesion between the right lung and the thoracic wall.

The lungs show considerable hyperstatic fluid and are deep red in the posterior portions. Microscopically, there is a little bronchial pneumonia. The right lung shows a scar at the apex, but no other evidences of tuberculosis. The bronchi contain a little thick, mucoid pus.

The lymph nodes of the hyla are enlarged to a considerable mass, the individual nodes measuring 1 to 2 cm. in diameter. They are very dark in color and considerable fluid exudes on section.

The heart shows no lesions except that the leaflets of the anterior and right posterior aortic cusps are united by a fibrous nodule about 5 mm. in diameter.

The spleen is large and soft, weighing 470 grams. The surface is irregular owing to the presence of small nodules underneath the peritoneum. On section nodules are found scattered throughout the organ. They are of opaque yellow color, irregular in outline, and measure from 5 to 10 mm. in diameter. There is one nodule which is much larger, measuring 4 cm., very soft and dark red.

The kidneys show no lesions. The suprarenals, bladder, and other genito-urinary organs are normal.

The mucous membrane of the intestine shows no lymphatic hyperplasia. The stomach mucous membrane is destroyed by post-mortem digestion.

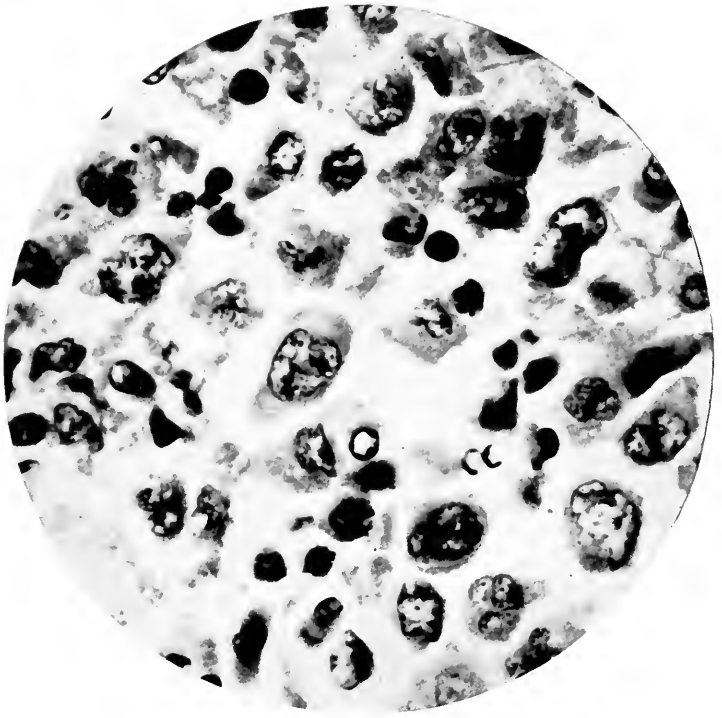


Fig. 1.—Nodule from liver. Case I.
× 1000.

Fig. 2.—Nodule from spleen, showing giant cell. Case I.
× 1000.

The mesenteric lymph nodes are moderately enlarged, measuring 5 to 20 mm. in diameter. They are pale yellow in color, firm and homogeneous on cut section.

The liver weighs 2,240 grams. It is deep red in color, and scattered through the tissue are a large number of yellow, irregular nodules, 5 to 15 mm. in diameter.

Scattered about the thorax and abdomen are a considerable number of enlarged nodes, some lying along the aorta and the esophagus, others under the iliac vessels and in the inguinal region. They are rarely above 1 cm. in diameter. About the trachea, however, the nodes are considerably enlarged, measuring 2 to 4 cm., and form a nodular mass. The largest node measures 2 to 4 cm., and shows on section mottled areas of yellow and deep red color. There were also on the parietal pleura a number of small, yellowish nodules, which are firmly attached to, and in some cases extend into, the substance of the ribs.

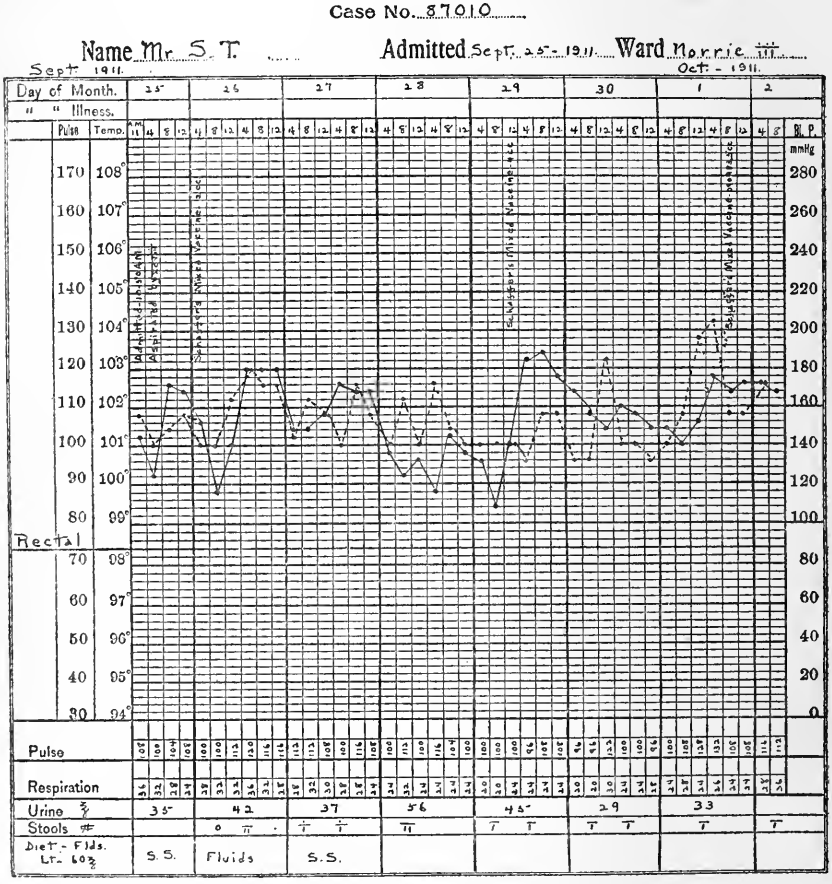
The thyroid shows no lesions. The bone marrow of the femur is a deep red in color in its upper third. The humerus contains red marrow in its middle portion. The marrow of the lumbar vertebræ is increased in amount and very deep red in color. The sternum and ensiform contain a considerable amount of reddish marrow.

Microscopical Examination.—Study of the bone marrow from the femur shows a hyperplasia of all the elements, with many plasma cells and a large number of eosinophiles in the tissue. There are also many areas closely set with normoblasts, such as are seen in the severe anemias. Megakaryocytes are abundant. The whitish nodule in the sternal marrow shows areas of necrosis surrounded by fibrous connective tissue, which contains many large multinuclear cells, plasma cells, and numerous eosinophiles. The lesions in the lymph nodes are typical of those of Hodgkin's disease; in other words, a fibrous hyperplasia with disappearance of the normal lymphoid structure and the growth of many multinucleated cells. A moderate number of eosinophiles are also present. A similar picture is seen in the nodules from the liver. There were no evident nodules in the kidney.

CASE 2.—HODGKIN'S DISEASE COMPLICATED WITH DIABETES MELLITUS CIRRHOSIS OF THE LIVER.

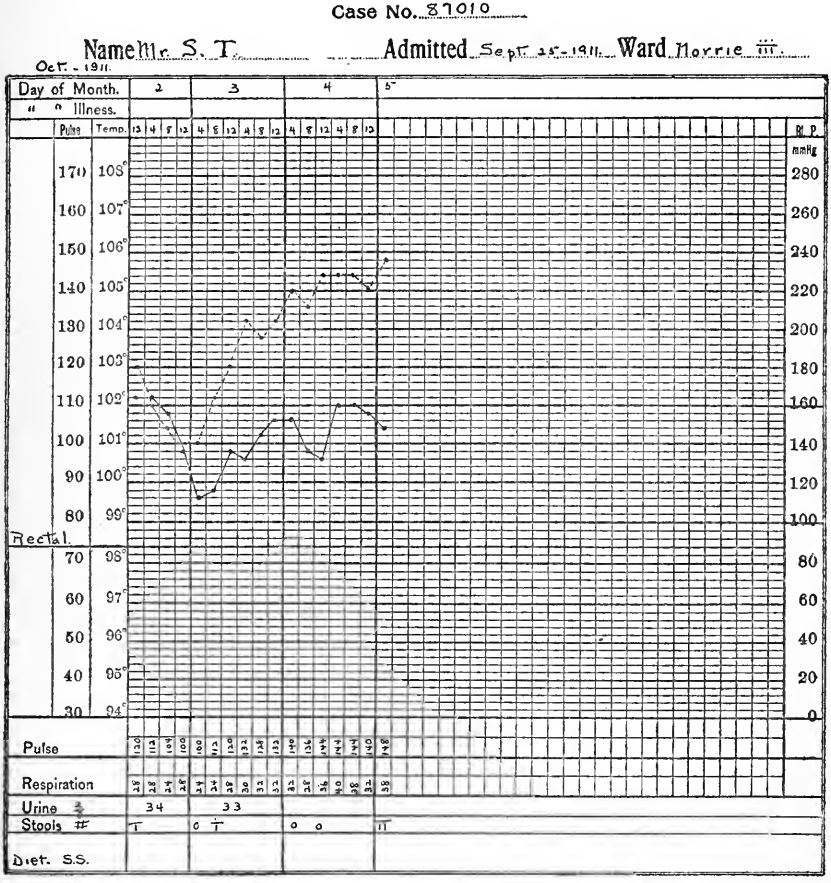
The patient, a male, of 39 years, was under observation in the hospital from September 25, 1911, to October 5, 1911. The reason for the patient's application to the hospital was that he had pain in

the abdomen, swelling of the feet and legs, and cough. Of his family history, the only fact of importance was that his father died at the age of 72, having had diabetes. The patient had been well until 6 years before his admission, at which time he developed diabetes, but improved under treatment. Some 3 years ago the glycosuria re-



turned, sugar being present amounting to about 4 per cent in the urine, but no attention was paid to this fact, and except for having lost weight, he had not suffered from any inconvenience. He had not attempted to keep up a strict diet. Four months ago he had herpes zoster. About this same time he began to have attacks of vomiting after meals, but only rejected the food which he had taken, and never

noticed any blood. He began to be constipated and lost weight. Shortly after it was noticed that he was jaundiced, with clay-colored stools and dark-colored urine. He had at the same time a great deal of abdominal pain, but no acute attacks. The pain also extended into the lumbar region. The symptoms continued until about 6 weeks



ago, when he noticed that his feet and ankles were swollen, and a little later his abdomen began to enlarge, and for the last 10 days he had not been able to walk. He had an annoying cough for about 4 weeks. His shortness of breath had not troubled him much.

Physical examination of the patient shows that he is a fairly well developed, poorly nourished man, with considerable jaundice of skin and sclerotic. He

has moderate dyspnea and is more comfortable sitting up, but he is not cyanotic. The pupils are equal and react to light. There are no conjunctival hemorrhages. A few small lymph nodes are palpable in the neck. The heart shows only a faint systolic murmur at the apex and soft systolic murmur over base. The second pulmonic sound is accentuated. The pulse is regular and of good force, and the vessel walls are not thickened. There is evidence of fluid in the pleural cavity, and a few râles can be heard in the lung. The abdomen is moderately distended, there is edema of the abdominal wall and of the back, and a fluid wave is present. The liver and spleen are not palpably enlarged. There is marked edema of the lower extremities. In the left axilla there is a group of much enlarged lymph nodes.

The day following admission, 8 ounces of fluid were removed from the abdominal cavity, and then the liver was found to be 3 inches below the costal margin. The spleen was just palpable. The urine was 1,024 acid, contained a trace of albumin, 2 per cent of sugar, no acetone, no acetic acid. The red cells were 3,900,000; hemoglobin 75 per cent; polynuclears 72 per cent; lymphocytes 28 per cent. The patient ran an irregular fever. A few days after his admission, a lymph node was removed from the left axilla, measuring 2 x 2 x 1.5 cm. Microscopical examination showed that the capsule of the node was intact, but the reticulum was largely converted into a fibrous tissue mass. The lymph structure was greatly diminished in extent, and many large cells with 5 or 6 nuclei were scattered in greater or smaller numbers throughout the fibrous tissue meshwork. Eosinophiles were not found.

The patient's condition gradually became worse, and he died 10 days after admission to the hospital. An autopsy was not obtainable.

A CASE OF THROMBOSIS OF THE VERTEBRAL ARTERY.

HENRY S. PATTERSON, M.D.

Of recent years, much interest has centered around the vascular accidents of the posterior fossa of the skull in general, and of the vertebral artery in particular. A number of cases of thrombosis of that vessel have been recorded in the literature of the subject, but as yet the symptomatology is sufficiently undefined to warrant a report of the following case:

F. B., 52 years of age, married, housewife. Admitted to the hospital May 20, 1911. Chief complaint, inability to swallow.

Present Illness.—Eight days previous to admission the patient became suddenly faint, and was obliged to sit down. When she attempted to drink, she found that she was unable to swallow, the fluid returning through the nose. She has had some slight numbness of the right side of the face. She has had no pain. She thinks that her hearing has not been good for the last few days. She has been gradually growing weaker, and has been obliged to take to her bed.

Past Illnesses.—None but an attack of swelling of the legs and abdomen, six months ago, lasting seven weeks. Menopause five years ago. Seven children; no miscarriages.

Family History.—Negative.

Physical Examination.—Large-framed, extremely obese woman, not acutely ill. Facies alcoholic. No icterus. Herpes on lips. Patient is conscious and rational, and speaks without difficulty, and distinctly. She is quite deaf in the right ear. She is apparently unable to swallow. The tongue is dry and coated, and does not deviate. The mucous membrane of the mouth is normal. The teeth are in very bad condition. The throat is red and congested. The tonsils are not enlarged. The muscles of the pharynx and soft palate are paralyzed on the right side. On phonating, the uvula and pharyngeal wall move to the left. No anesthesia of the pharynx. On swallowing fluid, it comes out through the nose, especially the left nostril. Very slight flattening of the left naso-labial fold. Patient says that sensation is more marked on the left than the right cheek. The right pupil is contracted; both react to light and accommodation, the left more actively than the right.

Heart.—Apex impulse in the fifth space, 12 cm. to the left of the mid-line. Left border percusses 13 cm. to the left of the mid-line. Right border just

to the right of the sternum. Action irregular in force and frequency. The first sound at the apex is valvular, and varies in quality. The second aortic is louder than the second pulmonic. Reduplication of the second sounds at the base occasionally. No murmurs. Pulse is rapid, and irregular in force and frequency, of poor size and force with increased tension. Vessel wall is palpable.

Lungs.—Clear front and back, except for a few scattered subcrepitant râles. Breathing diminished over the bases behind.

Liver.—Area of dulness not enlarged; edge not felt.

Spleen.—Area of dulness not enlarged; edge not felt.

Abdomen.—Diastasis of the recti, with a large ventral hernia, showing impulse on coughing. No masses nor tenderness; no ascites. Many old striæ.

Extremities.—No paralysis. Varicose veins over the lower part of legs, with scars of old ulcers. Slight œdema of the legs. Knee jerks present.

The temperature was 98° F. on admission, and gradually rose until it reached 105.6° at death, on May 23d.

The blood pressure ranged from 210 to 195 mm. of mercury.

Two leucocyte counts gave the following: 16,000; polymorphonuclears, 83 per cent; red cells, 6,200,000; hæmoglobin, 100 per cent.

Urine: Sp. gr., 1,020 to 1,022; albumen, 15 to 20 per cent. No casts found.

The patient gradually became weaker, lapsed into unconsciousness and coma, the temperature gradually rose, and death occurred on the fourth day of her stay in the hospital. From the sudden development of symptoms, and from the disturbance in the innervation of the right pharyngeal muscles, it was inferred that a hemorrhage had taken place into the nucleus of the right glossopharyngeal nerve.

Extract from the notes taken at the autopsy:

There is considerable œdematous fluid filling the pial spaces of the sulci, which is most marked in the posterior fossa. The right vertebral artery is distended to a diameter of 4 mm. by a thrombus which extends from a point just below the basilar artery to the anterior condyloid foramen, and apparently beyond. It occludes the orifice of the posterior cerebellar artery. On sectioning the medulla, about the level of the olive, an area of softening is seen, which occupies the upper right portion of the section, and is about 7 mm. in diameter. It includes the restiforme body, the upper portion of the olivary nucleus and the intervening structures. It extends up into the inferior cerebellar peduncle. A number of very small branches of the inferior cerebellar artery pass into the medulla at the level of the softened area. The medulla, below this point, and the region supplied by the right inferior cerebellar artery are normal. The cerebrum is normal. The ventricles are free.

REPORT OF A CASE OF ACUTE ENDOCARDITIS WITH INFLUX OF ALL THE CHORDÆ TENDINEÆ OF THE ANTERIOR CURTAIN OF THE MITRAL VALVES.

LEWIS F. FRISSELL, M.D.

T. T., hotel manager, 52 years of age, married, was admitted to the medical ward of St. Luke's Hospital, July 5th, complaining of severe dyspnœa. Until eight weeks before admission, he felt perfectly well, but at that time noticed that his customary mode of life fatigued him and that he became short of breath on exertion. He has not noticed precordial pain or sudden exacerbation of symptoms. Gradually his dyspnœa increased, confining him to bed, and of late has amounted to orthopnœa. He has not been conscious of fever, but his wife thinks there has been some elevation of temperature in the last few weeks.

There has been a little cough without expectoration. Urination frequent. His occupation required no severe physical work, and his habits were inclined to be sedentary.

Until the onset of his present trouble, he had been remarkably free from illness since his childhood, in which he had suffered from measles, scarlet fever, diphtheria and chicken-pox. No history of venereal disease or rheumatic fever is obtainable. His personal habits are bad. He is a constant user of alcohol, five or six whiskeys or beers daily with periodic sprees.

Tobacco is used in moderation. Coffee six cups daily.

The family history is good; his father died of pneumonia and mother of apoplexy.

Physical Examination.—Patient is a fairly well-developed and well-nourished man of 52 years, appearing moderately prostrated. There is some dyspnœa and orthopnœa. No jaundice or cyanosis. Skin and mucous membrane somewhat pale.

Eyes.—Pupils equal and react. Slight icteroid tinge to scleræ.

Tongue.—Moist, not coated.

Throat.—Negative.

Teeth.—In rather poor condition; a few missing.

Neck.—Visible venous pulsation.

Chest.—Well developed, expansion good.

Heart.—Lifting impulse general over precordium. At apex region is a thrill, diastolic in time. Diffuse visible and palpable apex impulse in 5th and 6th spaces 5½ inches from m.l. Left border 6½ inches out. Right border 2½ inches to rt. of m.l. At apex sds. are loud and booming in quality. At apex

and heard over whole precordium and transmitted to back is a loud systolic murmur, almost replacing first sd. This murmur has a maximum intensity at apex. To left of sternum in 5th space, murmur is almost musical in character. Within nipple and to sternum action of heart is gallop rhythm. Over base there is a soft systolic murmur. Both second sds. accentuated.

Pulse.—Regular, small, fair force, vessel wall palpable.

Lungs.—On right side posteriorly, beginning just below scapula, is slight dulness, increasing to base. At extreme base fremitus, voice and breathing sounds much diminished. An occasional râle heard at left base. Otherwise lungs are clear.

Abdomen.—Lax; liver percusses to free border, edge not felt, but liver region is somewhat tender on pressure. Spleen not palpable. No dulness in flanks. No masses.

Extremities.—Knee-jerks present. Considerable edema present.

The day following admission there was noted a presystolic murmur of rather short duration but distinct crescendo character just within and above the apex, and at the apex a very faint diastolic murmur of a transitory nature as it disappeared not to reappear.

The temperature was elevated, remaining between 102-101, the greater part of his stay in the hospital, but toward the end becoming subnormal.

Repeated blood cultures failed to reveal the infecting organism.

The physical signs in the heart did not change, though at one time the conduction time was increased owing to the effects of digitalis, and the systolic murmur became more intense and musical. His hydrothorax and consequent dyspnœa were several times relieved by thoracentesis, but after a long illness the patient died October 5 of a terminal infarction of the lung.

At no time did he complain of sudden, intense precordial pain, nor did his dyspnœa suddenly become aggravated.

Autopsy, October 19, 1911, 2 P. M., by Dr. C. H. Bailey.—Body of well-developed but emaciated adult male. Marked edema of hands, legs, and dependent portions of body. Slight general jaundice. Pupils, 3 mm., normal; conjunctivæ, yellow.

Peritoneum.—About one liter of clear yellow fluid in abdominal cavity. Over anterior surface of liver, especially right lobe, are patches of firmly adherent organized exudate, also over surface of spleen, and a few patches on intestines. Firm adhesions join the omentum to the parietal peritoneum on the left side in the region of the splenic flexure and upper portion of the descending colon.

Pleura.—Pleural cavities contain together 3,300 c.c. clear yellow fluid. Somewhat more in right than left. Left lung firmly adherent at apex.

Lungs.—At the base of the right lung anteriorly is a roughly circular area of consolidation, about 3 cm. in diameter, deep red on section, slightly raised base surrounding surface and with fairly sharply defined outlines.

A branch of the right pulmonary artery leading to the right lower lobe is completely thrombosed. The thrombus extends from the root of the lung, where it protrudes into the auricle as a free tongue-like mass to the posterior

portion of the left base. It completely plugs the vessel, is a little more than 1 cm. in diameter at its upper part, is grayish in color, with red mottling and generally firm, but in places softened. The whole lung, especially the base, is congested and edematous, but the tissue surrounding this vessel shows no sign of infarction. No thrombosed vessel is found leading to the area of consolidation in the anterior portion of right base already described.

Left lung.—At left apex are two or three fibrous scars and one calcareous nodule about 2 mm. in diameter. On anterior edge of upper lobe, about 3 inches below apex, is a roughly wedge-shaped area of consolidation, deep red on section, and with rather sharply defined limits. At its apex is a thrombosed vessel about 3 mm. in diameter. This thrombus is rather firmly attached to the vessel wall in places. A similar area is present in the anterior edge of the lower lobe.

Pericardium.—Contains about 50 c.c. of clear yellow fluid. On surface of heart are numerous large irregular whitish areas of organized exudate which are torn from the wall with some difficulty. The surface of these is generally smooth; one, about 2 cm. in diameter, on anterior surface of left ventricle, has an irregular, ragged surface.

Heart.—Very large, 570 gms. weight. A tough grayish clot is firmly adherent to the musculi pectinati of the right auricle. Tricuspid orifice dilated, 16.5 cm. Cusps normal. Pulmonary, 11 cm., normal.

On opening the left auricle, the anterior cusp of the mitral, and its chordæ tendineæ, is seen protruding into the auricle, the latter having been torn from their attachment to the muscles. On the auricular wall, about 3 cm. above the auriculo-ventricular orifice, are a few small, rough, yellowish vegetations. The mitral orifice measures 14 mm. The anterior cusp, all the chordæ tendineæ of which are torn from their attachment to the heart-wall, contains several nodular thickenings, but no recent process is apparent. The chordæ tendineæ attached to it are of apparently normal length, but enlarged, soft and yellowish in color, and appear to have been recently ruptured. At the summit of the anterior papillary muscle is a small stump which was evidently the point of attachment of one of the broken chordæ. There are two similar nodules in one of the cords of the posterior cusp near its attachment to the posterior papillary muscle. It is impossible to tell in the gross, whether these are vegetations or former points of attachment of the ruptured cords.

The posterior cusp also shows numerous nodular thickenings. Two hard, calcareous nodules, one nearly a centimeter in diameter, are felt in the substance of the cusp at its base. Over the larger of these, at the point of junction of cusp and auricular wall, is a small depressed area with rough surface. Over the other nodules the endocardium is smooth and glistening.

The aortic cusps are normal—9 cm. Heart-muscle appears normal. Right ventricular wall measures 2 cm. Coronaries normal.

Spleen.—255 gms. On surface are several patches of firmly adherent organized exudate. Capsule thickened. Very firm, deep red, trabecula prominent.

Liver.—Greenish-yellow with thickly scattered deep red points ("nutmeg liver").

Gall-Bladder.—Contains large amount of very thick greenish-red bile. Hepatic and common ducts admit passage of probe easily, and on dissection no calculi or other obstruction found.

Pancreas.—Normal.

Kidneys.—L. 195 gms., R. 195 gms. Capsules strip easily. Tissue slightly yellowish and opaque. Otherwise normal.

Stomach.—Intestines normal. Bladder normal.

Anatomical Diagnosis.—Chronic adhesive peritonitis; pericarditis; healed tuberculosis; double hydrothorax; thrombosis of branches of pulmonary artery; infarction of both lungs; cardiac hypertrophy; acute endocarditis; rupture of chordæ of anterior cusp of mitral; chronic passive congestion of liver and spleen.

Bacteriological.—Culture from heart's blood: No growth.

Microscopical.—Liver: Intense congestion about central veins with resulting atrophy of liver cells in center of acini. Many of liver cells about these areas filled with dark pigment.

Pancreas.—Slight increase of interstitial tissue.

Kidneys.—Capillaries congested. A few sclerosed glomeruli.

Pulmonary Artery.—Branch shows occluding thrombus of fibrin. It is adherent to wall in places. Degenerated in center. Undergoing organization at attachment to wall on one side. Another section shows occluding thrombus of large branch, extensively organized, except in center, which consists largely of red blood capsules. Surrounding lung tissue infarcted.

Lung.—Area of infarction consists of extravasated blood and exfoliated cells of alveoli; over a large portion outlines of alveoli only roughly indicated by broken-down connective tissue septa. Many of the exfoliated epithelial cells of the air-vesicles are loaded with pigment. Two small vessels show partial obliteration of lumen by fibrin and red cells, partially organized.

Heart.—Muscle, slight brown atrophy.

Chordæ tendineæ show evidence of old chronic inflammation.

The case presented is of unusual interest on account of the rarity of the lesion, its mode of production and the occurrence of a presystolic murmur in the absence of stenosis of the mitral valve. A review of the literature shows 50 reported cases, including 8 ruptures of papillary muscles, a synopsis of which is appended to the present report. Many of them are old, some in the days before the use of refined methods of physical examination, and even that reported by so great a master as Laennec is difficult of analysis, owing to the incorrect views obtaining as to the production of the second heart tone, so that his statement that "The contraction of the auricle as long as that of the ventricle *donnait le bruit de sufflet*" leaves one in doubt as to his meaning. Presumably, as he regards the second sound of the heart as synchronous in time with auricular systole, the murmur heard was diastolic in time. As regards the cause of the rupture, the

tendency of the early observers is to lay stress on physical effort and trauma, though if the cases be analyzed, in many of them an endocarditis was obviously present, as proven by vegetations or valve change, described in the autopsy reports. These changes were considered by some to be secondary, but precisely on what ground it is difficult to see, except from the absence of symptoms prior to the trauma or strain.

On dividing the cases as reported, it seemed wise to classify as follows:

1. Those cases due to severe traumata, such as fractured ribs from violent compressions, falls from a considerable height, stab wounds, gun-shot wound, in one the kick of a horse. Of this group seven cases were found resulting in a tearing of papillary muscle rather than the chordæ themselves, these latter being due to stab and gun-shot wounds, and a fall from a window, respectively. That trauma of such severity in the region of the precordium could result in rupture of the chordæ will, I think, be admitted without comment.

2. Cases of ruptured chordæ in which the rupture has followed efforts such as straining, lifting, excessive fatigue, severe cough, in which the autopsy disclosed no reported endocardial lesions in the heart. Of these five cases are reported of which two showed blood-vessel lesions, one an aortic aneurism, and the other coronary sclerosis. Two cases also showed papillary muscle rupture, leaving only one case of actual chordæ rupture in hearts apparently free from any other lesion. This is the case of Dickinson. The patient, a young male, twenty-one years of age, had a severe pain under the left nipple while lifting a load of bricks, developed immediate signs of cardiac insufficiency and died in two months. Autopsy carefully describes the valves as normal, except for rupture of the chordæ attached to the posterior cusp of the mitral valve.

3. Cases of rupture said to be due to or preceded by strain, but in which endocardial or myocardial lesions were also found at autopsy. In this class nine cases are found.

4. Cases of rupture of the chordæ, in which endocardial lesion was found, but not following known history of strain or trauma. In this class are nineteen cases. Among these are a number in which no history was given or obtainable, these constituting a sub-class of mere pathological reports.

5. Reported cases of rupture in which data given are insufficient to determine the probable cause in which no autopsy has been made and one ("Gilbin") whose report was not accessible. In this class are ten cases.

Obviously, the cases with pathological change in the heart vastly outnumber the cases without 28 to 12, and if we exclude the severe traumatic cases, we reduce this latter group to 5, which may be still

further reduced to 3 by excluding the 2 cases with vascular disease. Of the remainder, 2 are ruptures of the papillary muscle, leaving but 1 in which the chordæ alone were ruptured. Over-strain alone would therefore seem to be an infrequent cause of this occurrence, unless we regard the rupture itself capable of secondarily causing an endocarditis. This is the position of de Quervain, who reports a case of malignant endocarditis following a sudden muscular exertion. This contention he supports by quoting the production of endocardial lesion in animals by damaging the valves mechanically.

Experimental evidence of the difficulty of rupturing the healthy chordæ is afforded by Barié, who, while able to produce aortic ruptures with pressures of 170-400 mm. of Hg, was able to produce rupture of the chordæ in only one case, and that at 1,085 mm. of Hg., a pressure that is almost inconceivable in the ventricles, and which apparently, under experimental conditions, is more liable to rupture the heart-walls themselves than the chordæ. The ordinary pressure in the ventricle is but slightly higher than the pressure in the aorta, which may be taken roughly as its measure, maximal 3-400 mm. Other authors, as Libman, report in cases of subacute endocarditis such as are caused by his streptococcus viridans, the not infrequent localization of vegetations on the chordæ and occasional rupture, so that the number of ruptured chordæ may be much greater than the reported cases indicate.

It seems fair to assume, then, that the healthy chorda is rarely if ever ruptured by strain or exertion, and that a pre-existent endocarditis is necessary to rupture. That effort may rupture a diseased chorda is obvious. From the examination, it is impossible, in the reported case, to state the date of the rupture of the chordæ. Probably, giving way one by one, the lesion dates from his onset of symptoms, the gradual increase in symptoms being due to the increasing insufficiency of his mitral valve.

The symptoms of such a lesion are, of course, outspoken signs of mitral insufficiency with signs of cardiac insufficiency in proportion to the number of chordæ ruptured and the suddenness of the onset of leakage before the heart can accommodate itself. In a case where the patient is known to have had no cardiac signs, no enlargement of the heart murmurs or symptoms due to insufficiency, and where following a straining effort in which the intraventricular pressure may be assumed to be greatly raised, a sudden severe pain is felt or a feeling as of something having given way, followed by severe dyspnœa and

signs of mitral leakage, a rupture of this sort may properly be suspected. On the other hand, when without history of strain, a mitral leakage is found which gradually becomes worse owing perhaps to the consecutive giving way of the tendons, the lesion is indistinguishable from an ordinary mitral insufficiency, and this will be the case in the vast majority of such ruptures.

The origin of the presystolic murmur is less clear. Apart from complicating mitral stenosis or outspoken aortic insufficiency, but three instances are reported of a murmur occurring in diastole. The doubtful case of Laennec, previously alluded to, makes a fourth. One of these, that of Barié, may be attributed to the perforation in the aortic valve producing a Flint murmur. The other two occurred in cases where the papillary muscle was torn off, leaving the flap with its tendons and muscles free to travel between auricle and ventricle, respectively, in systole and diastole. The murmur was described, in one case, as a systolic and diastolic murmur; in the other, as a murmur in time presystolic, but not having a true presystolic character. "It was not soft, nor was it a squeak."

Our murmur was a fairly localized short murmur inside and above the apex, heard at times as far as the left sternal border, and though not intense, of a clearly rumbling, crescendo character.

As the anterior curtain of the mitral valve swung free it must have traveled from auricle to ventricle with each diastole, and the sound may conceivably have been due to the vibrations set up, particularly by the strong current of blood due to auricular systole. Much as a sail flaps in the wind when a fore and aft vessel comes about in a stiff breeze, so the increased strength of the blood current, at this period of the cardiac cycle, may readily cause a murmur in the heart due to the vibrations of the free flap.

A second possibility is that the abrupt termination of its course from auricle to ventricle brought it up with much the effect of cracking a whip.

SYNOPSIS OF PREVIOUSLY REPORTED CASES.

By Portal:

Observation concerning a case of rupture of two fleshy columns of the mitral valve. The lesion also involved the wall of the left ventricle.

By Corvisart:

Man 39 years of age, abuser of alcohol; transitory attacks of rheumatism; died soon after admission to hospital with symptoms of extreme dyspnœa.

Autopsy findings: Protuberant vegetations on margin of mitral valve and on semilunar aortic valves. The segment of the mitral valve in front

of the aortic orifice was no longer attached by tendinous cords to the fleshy columns. The chordæ tendineæ were ruptured, or detached, and it was barely possible to trace two of these cords at the level of one of the fleshy columns.

By Corvisart:

Man, 34 years of age, injured himself in trying to move, unaided, a barrel of alcohol; suffered from cough and palpitation of the heart, mitral valve studded with soft vegetations. In the examination of the pillars which support the mitral valve, two of them were seen to have been ruptured some time ago. The extremities of these two tendons were soft, smooth, and rounded at the site of rupture. It was not possible to locate on the border of the valve the exact spot where they must have been inserted before the rupture.

By Corvisart:

Courier, 30 years of age, was admitted to the hospital immediately after a horseback ride of one thousand miles, without any rest; he had crossed the Channel after this ride, and while at sea had felt a sudden great oppression, with hæmoptysis. He died soon after admission, under symptoms leading prior to the autopsy to the diagnosis of an acute lesion of the heart, "undoubtedly a rupture of one of its parts." The left ventricle contained one of the large columns which support the mitral valves, floating free in the ventricular cavity. It had ruptured at its base, evidently quite recently, and a small clot was found near the site of the rupture.

By Laennec:

Man, 35 years of age, was admitted to the Neckar Hospital in Paris, with a history of heart trouble dating five months back. Thrill 5, 6, 7 spaces. The contraction of the auricle as long as that of the ventricle "gave the bellows sound." Death soon after admission.

Autopsy findings: The heart was enlarged, especially the left ventricle. One of the tendons which pass from the extremity of the columns to the free border of the mitral valve was ruptured towards its middle. The upper portion was smooth, and was folded under the mitral valve, but without adhesions. There were warty vegetations on mitral valve and left auricle.

By Bertin:

A consumptive girl, 22 years of age: a severe coughing fit led to rupture of one of the muscular columns in which the tendons of the tricuspid valve are inserted; at the autopsy this fleshy column was found to be broken, floating free in midst of the ventricular cavity.

By R. Adams:

Cheyne's case: A musician, 34 years of age, strong and well nourished, of irregular habits, was suddenly attacked with a very severe pain in the left side of the chest, about the precordial region. The condition became steadily worse, with edema of the lower extremities, digestive disturbances, dyspnœa, loss of strength, cerebral symptoms, and so forth. Death about two months after the onset of the symptoms.

Autopsy findings: The most interesting feature consisted in the rupture of the chordæ tendineæ which attach the left auriculo-ventricular valve to the

columnæ carneæ. This rupture concerned variable levels, four of these tendons being found floating by one of their extremities in the interior of the ventricular cavity. Excrescences on mitral and segmoid valves.

By Marat:

A man, 44 years of age, on laboriously rolling a very heavy barrel, suddenly felt something snap in the back, and was attacked by dyspnœa and palpitation. Death twenty months after the accident.

Autopsy findings: One of the columnæ carneæ at which the tendinous cords of the mitral valve are inserted, was entirely ruptured, and pulled out. The patient also had an aneurism of the aorta, but he had never before complained of disturbances, which did not begin until the painful sensation referred to above.

By Nicod:

Autopsy findings, in case of a woman who had suffered from two attacks of suffocation, the last terminating in death: Rupture of two fleshy columns of the heart, at a distance from each other, of unequal length, with a different coloration of the ends.

By Legendre:

Autopsy findings in the case of a man who died with symptoms of dyspnœa, soon after fracture of the ribs, from violent compression: On opening the left ventricle of the heart, a large fleshy column, with tendons passing to the posterior segment of the mitral valve, was seen to be entirely broken and curled up on itself, entangled in two of its tendons.

By Prescott Hewitt:

A boy of twelve years fell from a height and died four hours after the accident. Autopsy findings: No external lesion on thoracic wall. Pericardium intact. Ecchymosis at point corresponding to upper portion of intraventricular septum; this bloody extravasate came from a small tear of the heart-wall which extended to the upper portion of the septum and established a communication between the two ventricles. Two columnæ carneæ in the left ventricle were torn.

By Williams:

Policeman, age 27 years, habitual user of alcohol, lost flesh and strength for two years. Three months ago, on quickly mounting stairs, felt a very sharp pain in epigastrium; some days later, edema of lower limbs, dyspnœa, etc. At time of admission, urine was scanty, blood-tinged, and slightly albuminous. Heart hypertrophied. Auscultation: systolic murmur loudest under left breast. Death a few days after admission.

Autopsy findings: The two mitral cusps were found to be thickened and ossified; the chordæ tendineæ, inserted at the anterior valve, were ruptured at unequal heights, and the fragments were lined with soft vegetations. Posterior cusp was ossified, and its chordæ tendineæ were agglutinated.

By R. B. Todd:

A man, 31 years of age, was admitted to the hospital, with general edema, enlargement of the liver, marked dyspnœa and frequent cough. History of a stab wound, three years previously, in right side of chest, below nipple. Death ten days after admission to hospital. Systolic murmur apex and base.

Autopsy findings: Other valves normal, but the tricuspid presented several interesting lesions. The anterior segment of the valve, namely that which separates the infundibulum from the auricular portion of the ventricle, was suspended free in the ventricular cavity, retaining its connection with the heart only at the level of the fibrous auriculo-ventricular orifice. All the fibrous cords, inserted at the valve, were ruptured at different heights, leaving a fringed valvular border. The fleshy columns in which the cords originate were contracted and showed the rudiments of the broken chordæ tendineæ. The extremities of the latter presented small bulgings, similar to those seen at the end of the nerves in an amputation-stump.

By Gordon:

A woman, 26 years of age, who had been admitted to the Whitworth Hospital with violent hæmoptosis. The diagnosis of rupture of the chordæ tendineæ of the heart was rendered, on the basis of the sudden and violent pain in the region of the heart, followed by intense palpitation and weakness, as well as the decided character of the bruit, and frémissement at the root of the neck. She lived ten days after her admission and then sank; the immediate cause of death was pulmonary apoplexy.

Examination showed very slight disease in the aortic valves; there was a sight deposit in the central valve. Several of the chordæ tendineæ of the anterior portions of the mitral valve were ruptured, and covered with a soft cheesy matter. There seemed to have been slight endocardial inflammation, followed by rupture of the chordæ tendineæ, and this by the effusion of lymph, which lay in great quantities loose in the ventricle.

By Allix:

A prostitute, 25 years of age, was admitted to the St. Jean Hospital in Brussels, having become unconscious a few instants before. Auscultation was impossible; heart sounds were confused and arrhythmic, but accompanied by a distinct vibratory thrill. Patient died one hour after admission. Thrill marked.

Autopsy findings: Trace of an old endopericarditis, marked hypertrophy of the left ventricle. On opening the cavities, it was seen that the chordæ tendineæ, passing from the summit of the principal left columna carnea to the free border of the anterior segment of the mitral valve, were ruptured in the middle; these tendons were very fragile and easily torn; their surface was found to present a large number of small, round, wartlike vegetations.

By Charles A. Lee:

Man, age 65 years, while driving a stake into the ground with a heavy piece of wood, felt something give way suddenly in the region of the heart, and immediately fell to the earth, gasping for breath, and laboring under excessive pain and dyspnœa. He never was well again, but lived for about ten months afterwards, with increasing symptoms of heart disease.

Autopsy: The endocardial membrane was much thickened from chronic inflammation; organized lymph was deposited beneath it. Several of the chordæ tendineæ of the mitral valve had evidently been ruptured, as only their shriveled remains were visible, while others, both of the tricuspid and mitral, were so contracted and adherent to each other as to contract the cir-

cumference of the valves to such an extent as almost to close their orifice, and, of course, to prevent entirely their healthy play.

Blakiston found the chordæ tendineæ shortened in 20 out of 46 cases of tricuspid regurgitation. In one he says, "One of the chords had apparently been broken, and was curled up into a nodule, like a pin's head" (p. 291) (*cit.* Lee).

By Austin Flint:

Woman, 35 years of age, who had suffered for some years from heart disease. "The interesting point connected with the specimen is not the contraction of the mitral orifice, which is common enough, but the presence of two vegetations of considerable size, one as large as a bean, the other somewhat smaller. The larger one is attached to the papillary muscle of the inferior curtain by what appears to be a small pedicle, which is a fractured extremity of one of the tendinous cords. The other concretion is upon another tendinous cord which has not been fractured.

By van Giesen:

Man, aged 24 years, was admitted to U. S. Naval Hospital, and presented hypertrophy of the heart, with tumultuous action, and a murmur with the first sound transmitted along the course of the aorta. Gradual aggravation, death about six months later.

Autopsy findings in heart: On opening the left ventricle, the aortic valves are found to be thickened, contracted and studded with tenacious, fibrinous vegetations. The anterior portion of the mitral valve is also covered with similar vegetations. The chordæ tendineæ of the anterior portion of the mitral valve are all ruptured, shortened, and covered with tenacious, fibrinous effusion. The free extremities, which are expanded into small bean-shaped bodies when drawn with moderate force toward their original muscular attachment, will not meet by about a quarter of an inch. The chordæ tendineæ of the remaining portions of the mitral valve are healthy, presenting no traces of atheroma or ulceration.

Dr. Stokes (*cit.* v. Giesen), in his work upon Diseases of the Heart and Aorta, details a case, extracted from the records of the Pathological Society, which in many respects is similar to the one observed by v. Giesen:

"The cords of the anterior portion of the mitral valve were all broken across near to the fleshy columns; they were thickened, softened, and covered with beads of very soft lymph."

By J. Pollack:

A woman, 42 years old, was admitted to King's College Hospital with severe pain palpitation, dyspnœa, and hæmoptysis. Dr. Johnson diagnosed rupture of one or more of the chordæ tendineæ of the mitral valve.

Post-mortem examination of heart: The pericardium contained four ounces of serum. The heart was large, the left ventricle being hypertrophied more than the right. One of the chordæ tendineæ of the mitral valve was ruptured. Mitral valve was diseased, and the orifice contracted. Aortic valves much thickened.

By Kelly:

Description of specimen derived from a woman aged 49 years. Symp-

toms of heart disease for about two months prior to death. Autopsy: Left ventricle somewhat dilated, aortic valves healthy, slight atheroma of ascending aorta. The anterior curtain of the mitral valve was fringed on the auricular side with some fibrinous beads, and some were found on the tendinous cords also. The posterior curtain was found lying loose; all the chordæ tendineæ were ruptured, and many were much shorter than usual; some had a small bead of fibrin on their free extremity; all broke off close to a fibrinous deposit. It is probable that they were not all ruptured at once, as in some the fracture seemed quite smooth, while in others there was a little fibrin on the free extremity.

By Dickinson:

Patient, male, age 21 years. Perfectly well until four months previously, when he suddenly felt pain under left nipple whilst lifting heavy loads of bricks. Unable to work since, became worse in hospital, and died after two months.

Diagnosis of laceration of chordæ tendineæ in mitral valve was confirmed by autopsy findings. The chordæ tendineæ attached to the lower edge of the posterior flap of the mitral valve were all broken close to their insertion into the fleshy columns, excepting that one or two cords remained entire at one corner of the curtain. A solitary tendinous cord, which was attached at the base of the flap near its center, remained entire. The segment of the valve of which the cords had been broken, appeared to have lost all valvular action, and must have swung uselessly from its base. The broken cords hung with loose ends, which had become somewhat thinned. The free edges of the mitral valve had become somewhat thickened and opaque.

By Bristowe:

Patient, man, age 62 years, died under symptoms of heart disease, which came on almost suddenly about three weeks before his admission to hospital.

Autopsy findings: One of the tendinous cords attached to the posterior flap of the mitral valve was ruptured, the cord was much swollen, and of an opaque yellow tint; this change was most marked in the situation of the rupture. The lower portion of valve was dilated into a pouch and had a deep rugged notch. Mitral was normal in all other respects. Aortic valve was perfectly healthy. Coronary arteries, calcareous.

By Hanot:

The patient, a man, 37 years of age, was admitted with all the signs of mitral insufficiency: systolic murmur at apex, edema of lower limbs, enlarged liver, signs of bronchitis, etc. After three weeks' stay in the hospital, he was suddenly attacked by oppression and breathlessness; the face was livid, the body covered with clammy sweat; irregular heart-action, rapid death.

Autopsy findings: Aorta, intact; valvular lesions of mitral valve; also three valvular tendons, about one centimeter long, whitish and thickened, were found to be ruptured and floating in the ventricular cavity.

By Le Piez:

A woman, 24 years of age, died suddenly (in syncope) on getting up out of bed, a fortnight after the onset of symptoms pointing to heart disease (no abnormal sounds in heart). At the autopsy, the heart-wall was found to be

friable and in a state of fatty degeneration. One of the fleshy columns of the heart was ruptured, cut in two, at the junction of the two lower thirds and the upper third. The segment, where the chordæ tendineæ are inserted, was displaced between the two mitral valves. One rather large-sized tendinous cord was completely ruptured; it did not belong to the ruptured column.

By Gilbin:

Personal observation of a case of rupture of the tendons of the mitral valve. Records not accessible.

By A. W. Foot:

Man, aged 23 years, who had died suddenly while in the act of getting into bed. (All the physical signs of regurgitation through the mitral valve had been present.) The cords, which were found broken across about the middle of their course, were two or three of those attached to the musculus papillaris, which regulates the larger curtain of the mitral valve, and were those nearest to that portion of the curtain which is adjacent to the interventricular septum. The broken cords were studded with warty nodules of fibrin; both the ventricular and auricular surfaces of the principal curtain of the valve were covered with vegetations of a similar character, and continuous with those creeping along the chordæ tendineæ.

By Willard Parker, 1859:

The patient lived several years, suffering with heart disease. There were also fibrinous granulations upon the cords.

By Willard Parker:

Sudden rupture of the chordæ tendineæ, while running to a fire. Death occurred a few months afterwards.

By J. C. Dalton, 1859:

A man, aged 40, whose previous history was unknown, was found in his room comatose and died one hour later.

Autopsy: The mitral valves were covered with several small vegetations. Two of the tendinous cords attached to the inner portion of the anterior curtain of the valve were ruptured.

The rupture was at the point of attachment of the cords to the valve. The free ends of the cords were covered with fibrin, giving them a bulbous shape.

By Alonzo Clark:

There were vegetations on the broken ends of the cords, and upon the valves connected with them. There were vegetations on the cords of three or four other specimens which had been presented to the Society. In one case there was a thinning of the cords, ending in rupture; in other cases, the cords were thickened, but softened. Chordal inflammation is apt to be followed by vegetations, softening, and rupture.

By J. T. Metcalf:

Man, age 23 years, became rheumatic after an injury to the side and exposure to cold; developed dyspnœa, edema of face, ascites, anasarca. Rough systolic murmur over mitral valves, effusion into pericardium and both pleuræ; albuminuria. Death from exhaustion.

Autopsy: Several chordæ tendineæ were ruptured. The aortic valves were studded with fibrin and insufficient; there was a large white clot in the heart.

By Lionville:

Case of an old woman, in whom the columns of the mitral valve were ruptured in consequence of a fall from a window on the third floor.

By Terrillon:

Man, age 48 years, gun-shot wound of chest, penetrating the seventh rib; death twelve hours later. Autopsy showed extensive ecchymoses in the pericardium and myocardium of the anterior wall of the left ventricle; no solution of continuity; rupture of individual trabeculæ and mitral tendons.

By Potain *cit.* Barié. *Revue de Médicine*, 1881, p. 318.

A young woman died rapidly, in the Petié Hospital of Paris, under symptoms which were referred to puerperal endocarditis. At the autopsy, all the tendinous cords of the flaps of the mitral valve were found to be ruptured, so as to produce a true acute insufficiency.

By Barié:

Woman, age 56 years; history of articular rheumatism, followed by symptoms of heart disease; mitral systolic murmur; thrill, death one week after admission to hospital. Autopsy: Heart not enlarged, cavities small, walls of left ventricle slightly thickened. The mitral valve was whitish and somewhat thickened; on testing with water, it was found that the posterior valve did not fit against the anterior valve, but floated in midst of the fluid, in consequence of rupture of four of its tendons. These tendons were broken about the level of their middle portion; they were somewhat thinned, but on examination presented no inflammatory changes. The aortic valves were normal. The tricuspid valve was intact and sufficient.

By Potain:

Rupture des tendons de la valvule mitrale. *L'Union Médicale*, 1891, p. 279.

Man, 72 years of age, who had died with symptoms of mitral insufficiency; the autopsy showed the presence of rupture of one of the tendons of the great mitral valve. The tendon had given way close to its insertion, and either floated in the ventricle, or became interposed between the flaps of the valve, preventing their accurate junction, and giving rise to intermittent mitral insufficiency.

By C. W. Sharples:

Laborer; systolic and presystolic murmur, latter not characteristic in quality.

At the autopsy of a man 45 years of age, there were found lesions of auricular endocarditis, and a rupture of the chordæ tendineæ, which were changed in appearance and character, being all that were attached, most anteriorly and nearest the center of the valve, with only one remaining on the left; thus leaving the valve to flap back and forth without its normal control. The longest chordal fragment on the valve was three-fourths of an inch long. It was softened, thickened, and beaded, smooth over most of its length, with one hanging vegetation. Attached to another broken chorda was a mass half an inch long and one-eighth in diameter, fastened by a

narrow, small, short pedicle. Otherwise it was free to flap about in the ventricle. The other chordæ presented no peculiarities, except that they were thick, soft, and very friable. One of the transverse bands connecting two chordæ near their origin presented a large vegetation.

By Degny Huchard:

Man, 42 years of age, mitral insufficiency, also aortic insufficiency. Autopsy: The tendinous cords inserted at the posterior pillar (angle of septum and ventricular wall) were adherent to the posterior commissure of the great mitral (cardio-aortic) valve. These parts were rigid and calcified, on one arterial surface. Analogous lesions were noted at the summit of the anterior pillar and the anterior commissure of the great mitral valve and the connecting chordæ tendineæ. But here the chordæ tendineæ were ruptured, one large tendon in particular being broken off about one centimeter from its insertion at the valve, so that this anterior portion of the great mitral valve, being free from all tendinous chords, had become displaced upwards, into the left auricle. Only the presence of the rigid calcified tendon stump, which remained adherent to the great valve for about one centimeter, caused the luxation of this portion of the valve to remain stationary. The rigidity of the chordæ tendineæ in front, opposite the small valves, acted like a rigid body placed crosswise, and prevented the great valve from resuming its normal position.

By Hallé:

In the case of a man 63 years of age, who had died from broncho-pneumonia, after suffering for two months from symptoms of heart disease, the autopsy showed the rupture of several tendons and chordæ tendineæ, on the left pillar of the mitral valve; two of these small tendons floated free in the auricle.

By Poupon:

Man, 41 years of age, who had died from a ruptured gastric ulcer; the mitral valve was found to be insufficient and the seat of peculiar changes. A softened vegetation, probably a band attached to the lower border of the anterior pillar, floated free in the cavity, toward the cardiac apex, and therefore in the direction of the blood-stream. The flaps of the mitral valve were extremely thick and hard, with scattered calcified spots. The anterior pillars of the second class were connected by fibrous bands; one of these pillars presented an ulcer, with calcified margins. All the constituents of the mitral valve and its pillars were considerably hypertrophied.

The emboli found at the autopsy were attributed to the rupture of a tendinous band of a pillar of the mitral valve; the band itself was interpreted as the result of an old endocarditis. Murmur at apex. Time not determined.

By Henry:

Rupture of the posterior papillary muscle, 2 cm. in length (one in thickness), of the mitral valve, in a young robust male, known to be in good health two years previously. Death after about eight months, after transitory improvement, following upon traumatism (a kick from a horse) directly on precordium. The clinical picture showed a complicated recent cardiac af-

fection, with the sole symptoms of diastolic-systolic murmurs and dilated heart (ox-heart), which were explained by the free floating papillary muscle; this hung suspended from the chordæ tendineæ, and was necessarily thrown constantly from the ventricle into the auricle, and back again through the blood-current and the cardiac contractions.

By de Quervain:

Man, age 35 years, in good health, experienced a sudden painful sensation in the chest, when holding up a very heavy barrel; this was followed by epistaxis and bloody expectoration. Later, cyanosis, increased frequency of pulse-rate, and cardiac distress. No findings early on auscultation, but three weeks later, a rough systolic murmur was heard at the mitral valve. The general condition became worse, and seven weeks after the accident, the patient died with symptoms of cerebral embolism.

Autopsy findings: Circumscribed thickenings at free margins of anterior mitral flap, 1½ cm. long, 1 cm. wide, irregular and friable. The valve in this area presented no chordæ tendineæ, but there was the stump of one. Microscopically, the thickening was interpreted as a fresh endocarditis proliferation, in part ulcerative in character.

By Schmidt:

A man 85 years old fell out of the window, from the second floor, landing on the left side of the thorax.

Autopsy findings in heart: Posterior aortic valve presented a rupture through its entire thickness. There was also a tear 2 mm. long at the lower surface of the anterior mitral flap. The simultaneous rupture of the mitral valve is explained by the author in such a way that after the aortic rupture, the sudden back-flow of the aortic blood struck the open mitral valve, thrusting it up, and partly tearing it away from its support, the chordæ tendineæ. The rupture had occurred immediately above the insertion of a tendinous cord of the second class, which is inserted at some distance from the free margin, on the surface of the valve. Otherwise the mitral valve and its tendinous cords, as well as the right-sided valves, were unchanged. The heart was slightly atrophic; no degeneration of the myocardium.

By Buchanan:

Male. Symptoms, cardiac insufficiency with fever four years before. Systolic murmur over cardiac area toward axilla over vessel of neck.

Autopsy: Mitral admits 3 fingers; anterior curtain presents an irregular fringe of ruptured chordæ, 8 in number. Free extremities bulbous and granular from endocardial thickening. One papillary muscle, soft, pale, atrophied, is completely severed from tendon. A few vegetations on margin of curtain freed by rupture.

By Hawthorne:

Male. Gave up rowing three years before death on account of "weakness." No definite mitral insufficiency. Presystolic thrill and murmur at apex. Systolic over whole precordium.

Autopsy: Mitral stenosis. Anterior flap projects into and half closes

orifice. Three tendons are torn. Endothelium granular and swollen. Free edge of curtain studded with vegetations.

By Barié:

Case showing presystolic thrill and presystolic and systolic murmur at apex. Also diastolic at base.

Autopsy: No mitral stenosis. Two chordæ of anterior valve ruptured, mitral calcareous. Hole in posterior cusp of the aortic valve.

By Gordon:

Not accessible.

By Jayle:

Systolic murmur at apex.

Autopsy: Rupture of the tendon at anterior flap.

By Norris:

Signs of mitral disease following lifting effort. Examined before; no signs. After systolic murmur at apex. Suspicion of presystolic.

Diagnosis made of ruptured chordæ. No autopsy.

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A REPORT OF TWO UNUSUAL CASES OF SEPSIS.

Lewis F. Frissell, M.D.

S. H., boy, 12 years old, was sent to the hospital July 12th, with a diagnosis of appendicitis. He had been a Boy Scout, and was perfectly well up to July 9th, in camp. On this date he had had some altercation with the other boys and thinks the parts in the neighborhood of the root of the penis were injured. Since that time he has felt weak and has had pain in the right inguinal region, low down. He was nauseated and vomited several times.

There had probably been fever for some time, as patient on admission was irrational and his history is not dependable, that of the trauma being due possibly to a delusion arising from catheterization. His admission temperature was 105 2/4, P. 100, P. 28. When first seen in consultation on the surgical division, at the request of the attending surgeon, the boy looked acutely ill, and presented, on physical examination, no focal symptoms beyond acute local tenderness in the right groin, apparently maximal at a point just to the right of the symphysis pubis. This, with an enlarged spleen and one or two erythematous spots on the abdomen, were all that could be found.

Evidence of wounds, trauma, pus pockets around the teeth, tonsils, or ears were not present.

A blood culture was taken and positive diagnosis withheld.

Two days later, on July 15th, he was admitted to the medical ward. The complete physical examination follows.

The patient is an undersized, fairly well nourished boy of 12 years, appearing acutely ill. There is no dyspnea, cyanosis or jaundice. The skin and mucous membranes are of good color. He is slightly irrational; muscular tremor of hands and arms. On turning in bed, he moves slowly and carefully, as if motion were painful. Patient is generally hyperæsthetic.

Eyes.—Pupils equal and react to light and accommodation. Tongue dry, coated, tremulous.

Throat.—Tonsils are slightly enlarged. Neck, sub-maxillary gland on left

side of jaw palpable; no other glands enlarged. Chest, fair development and expansion.

Heart.—Visible apex beat in fifth space, 3 inches from mid-line. Left border, 3½ inches. Right border, 1 inch to right of mid-line. Sounds of good quality, no murmurs heard. Second sound loud and snapping. Pulse regular, good force, vessel wall not thickened. Lungs clear. Abdomen, even contour, not soft, no rigidity made out. Spleen, sharp edge palpable 1 inch below costal margin in nipple line. No abdominal tenderness. There is marked tenderness to gentle pressure on the rim of pelvis just to right of symphysis. An inguinal gland is palpable on right side, smaller one to left.

Extremities.—K. J. present. Kernig's sign present. Babinski's absent. Abduction of right leg causes pain, but not flexion or rotation.

The elbow of the right arm is swollen, red and tender; there is limitation of motion, due to pain. On the outer edge of hand is a small pustule.

Rectal Examination.—There is tenderness on right side at the line of the pelvis; the sharp edge of rim is not felt as plainly as on the other side.

Eye Grounds.—Normal.

Ears.—Normal.

Urine.—Acid 1,020; alb. trace sugar 0; few granular casts. Leucocytes 13,000, P. 75%. Widal negative.

In 48 hours there had developed an inflamed joint and a pustule; a tentative diagnosis of septicemia was made.

July 16.—Right ankle and left hip involved; another pustule developed on shin. The heart showed a systolic murmur over the pulmonary area. There was some rigidity of neck.

Lumbar puncture; no increase of pressure; fluid clear; few cells; all lymphocytes.

July 18.—Culture from pustules shows staphylococcus aureus. Report of blood culture shows staphylococcus aureus; spinal fluid shows no growth.

The temperature has been high, 101-104°, without chills, perspiration or extreme variations.

The right ankle joint was opened and treated surgically.

On July 28, an extensive urticarial eruption appeared on the chest and abdomen, and the autogenous vaccine treatment stopped in consequence.

July 29.—The urticarial eruption has disappeared, but on right chest and in axillary region and along right costal margin are large hemorrhagic spots.

Heart.—A soft systolic murmur has appeared at the apex, transmitted to the axilla.

Death occurred on August 1.

The entire case presents the appearance of a malignant sepsis, the main interest being the site of the original focus.

The arduous life of a Boy Scout in camp precludes the possibility of an acute septic process before July 9th, and, while the heart valves,

of course, may have been involved from the start, evidence of old valvular trouble was lacking.

If the boy's tale of maltreatment, told in delirium, be true, this may account for the localization of the process in the os pubis, the infection being caused by some wandering staphylococcus.

Case 2.—M. M., 44 years, housekeeper. Patient was admitted to the ward July 23, 1911. At the time of admission she was irrational. Her history, in consequence, was not to be depended on. From members of her family an incomplete anamnesis was obtained. Since infancy she had suffered from some paralysis of the left side, which had caused a limp and a deformity of the fingers of the left hand, which, however, was not functionless.

Otitis media of right ear for years. Date of original trouble unknown. It probably followed an attack of measles, which occurred in childhood.

Six weeks ago the patient complained of severe pain over left gluteal region, running down the posterior aspect of the thigh as far as the knee. The continuation of this pain caused her to go to bed 4 weeks ago, and a diagnosis of "sciatica" was made by her physician. It is not known whether or not there has been fever, but patient has been thirsty. Two days before admission she complained of pain over the other sciatic nerve, and her mental condition became cloudy. Mentality is said to have been good previously. Increased frequency of urination during the past 2 days. The temperature was continuously high, running regularly from 101-105°.

Patient is a poorly developed, emaciated, middle-aged woman, at times irrational, who lies in bed with elbows and knees flexed. Patient mumbles incoherently, but will answer simple questions in a thick voice. The eyes are sunken and the face has an anxious expression. The respirations are rather shallow and somewhat irregular. There is no dyspnea, cyanosis or jaundice. Skin and mucous membranes are pale.

Eyes.—Pupils equal and react.

Tongue.—Dry, coated with brownish material.

Throat.—Dry, coated with brownish material.

Teeth.—Gums and lips covered with sordes. Teeth in poor condition.

Neck.—No glands palpable. No neck rigidity.

Chest.—Poor development and expansion.

Heart.—Apex impulse seen and felt in fifth space, 3½ inches from median line. At the apex there is a very slight thrill, systolic in time. Left border, 4 inches out. Right border, under sternum. At apex the sounds are loud and forcible, first sound somewhat impure. No murmurs heard. Action regular. Over the base there is a soft systolic murmur. The second aortic is slightly accentuated.

Pulse.—Regular, medium size, fair force, vessel wall moderately thickened.

Lungs.—Hyperresonant note everywhere. Breathing sounds are faint on account of shallow respirations. On right side of sternum and left outline of cardiac region are heard short pleuritic râles with inspiration. On an oc-

casional deep breath by patient fine crackling râles are heard in bases of both axillæ, posteriorly and at both bases.

Abdomen.—Muscles are held rigidly. Liver percusses to free border; edge not felt. Spleen not palpable. No pelvic masses or tenderness made out.

Back.—On upper part of left buttock is a red, excoriated area, 2 x 2½ inches, with round, black, central slough. No masses or tenderness found in sacro-iliac joint region.

Extremities (Upper).—There is considerable rigidity of arms, but no joint involvements made out. The left hand is deformed, being flexed at wrist, with extension of first three fingers, and flexion of last two fingers. The grip is weak. There is no atrophy of muscles. Reflexes are active.

Extremities (Lower).—Knees are flexed; extension causes pain. K. J. are active. No edema. The left knee is slightly swollen, red, hot and tender. No fluid made out. There is evidently a partial foot drop on left side. The ankle is slightly red, and causes pain when touched or moved. There is no marked response to pressure over either sciatic nerve, but flexion of legs causes pain, particularly on left side.

Eye grounds normal, except for physiological excavation.

Ears.—Right, acute inflammation on the site of an old purulent otitis. Left, scar in inferior portion; serum thick.

The admission temperature was high, 103°. A leucocytosis of 19,500, with a polynuclear count of 85 per cent, and joint inflammation combined with a suppurating ear, made a septic process the probable diagnosis, the only question being the location of the process. The deformity of the hands and partial foot drop, with a history of a limp and deformed hand, made one fairly confident of an old infantile lesion in the neighborhood of the right internal capsule, and probably not related to the present condition. The following day rigidity of the neck developed, and lumbar puncture was performed to determine the presence or absence of meningitis. A clear sterile fluid was obtained under only slight pressure. Blood cultures proved sterile. The leucocytosis grew more intense, 35,000. The systolic murmur at the base became louder and harsher, and a soft systolic murmur was heard at the apex on July 29th, on which date lumbar puncture was again performed, and 10 c.c. of clear fluid obtained.

Early in August she was transferred to the surgical side, and the right mastoid explored and the dura inspected, but without result, the autopsy showing an acute vegetative endocarditis and old calcareous cerebral lesions, which may have been either old solitary tubercles or inspissated masses of pus with calcification.

Autopsy.—August 6 and 7. Body of much emaciated woman of middle age. Several large pigmented moles on abdomen. Left leg abducted, and pelvis tipped to left in compensation. Left hip enlarged. Two bed sores on this surface. Both forearms wasted. Contractures of left hand. Many teeth missing, others in bad condition. Right mastoid chiseled out and packed with gauze.

Peritoneum normal, except for old, dense adhesions about the spleen.

Pleuræ obliterated by old adhesions, which are very dense posteriorly.

Lungs.—Left apex voluminous and firm, base collapsed and boggy. On section, surface moist and gelatinous. A large amount of edematous fluid can be expressed, leaving some granular areas. Base deep red on section, and contains edema fluid. Right upper lobe voluminous and firm, lower collapsed and boggy. On section, upper presents a moist gray and red surface, quite solid, with yellow clots of thick pus scattered throughout. Further sections show cavities up to 2 cm. in diameter, fixed by their fibrous tissue, and filled with brownish, turbid fluid. Base congested. Bronchial nodes enlarged and caseous.

Heart.—Pericardium normal. Heart small, atrophied; mitral valve has a row of large fibrinous vegetations along line of closure, some projecting 4 mm. into lumen. Aortic cusps show smaller vegetations, ½ mm. in diameter, about the corpora arantii and on folds where the cusps join. The heart muscle is pale and brownish.

Spleen.—Small, very soft, and adherent.

Kidneys.—Normal size; capsule strips readily. Cortex very pale; epithelium opaque. Markings well preserved and regular.

Liver.—Normal size.

Stomach.—Normal.

Intestines.—Normal.

Pancreas.—Normal.

Aorta shows slight atheroma.

Brain.—Very dense and fibrous; moderate edema of pia in temporal fossa. Cerebellum adherent to dura over lateral posterior portion of left lobe. At this point there is a dense calcareous mass, about 2 cm. in diameter, in the cerebellar tissue. Cortex normal.

On section, a cavity is found occupying the position of the head of the caudate nucleus, and partially replacing the lenticular nucleus and anterior limb of the internal capsule on the right side. It measures 18 x 10 x 10 mm. in diameter, and is separated from the lateral ventricle by a delicate wall.

From the floor of the cavity a papillary calcified mass projects upward into the cavity. The cavity is filled with thin, slightly turbid, brownish fluid. Scattered about the sulei, beneath the pia, are a number of spherical nodules 3 cm. in diameter, of about the color and consistence of white matter of lime.

Anatomical Diagnosis.—Acute endocarditis, septic pneumonia of right upper lobe, and broncho-pneumonia of left upper lobe.

Healed tuberculosis of brain and cerebellum. Section and microscopical examination did not prove tuberculosis.

Bacteria.—Smears from heart valves gave + cocci in pairs and short chains, also large, coarse Gram + bacilli and smaller Gram negative bacilli. No tubercle found.

Cultures from lung and heart valves all showed a colon-like bacillus. (Probable post-mortem contamination.)

Section apparently through basal ganglion shows thickening of glia usual about subependymal vessels. There is also an island of glia tissue in the ganglion.

The case is mainly of interest in disassociating the old from her recent symptoms. How much importance to attach to her chronic ear as a site for the portal of infection, whether or no a pus focus lay under the old otitis in the silent area of the lesion, was difficult to make out in the presence of increasing meningeal symptoms such as rigidity of the neck and Kernig's sign, with increasing signs of cerebral irritation.

The site of the active focus was probably in the vegetations on the heart valve, with a probable portal of entry from the ear or the oral cavity.

THE DILATATION TEST FOR CHRONIC APPENDICITIS.*†

W. A. Bastedo, M.D.

That many persistent digestive disturbances are manifestations of a latent or chronic appendicitis has been repeatedly demonstrated by the disappearance of the disturbances after the removal of the appendix. It is also well known to operators that in some of these cases the appendicitis was not recognized for a long time, and that even after long observation there were cases in which there was a large element of uncertainty as to whether the appendix was involved or not. In other words, the appendicitis was latent, and could not be detected by the ordinary means of examination. Hence any measure by which such a latent appendix involvement can be recognized deserves consideration. We would, therefore, again call attention to the usefulness of dilating the colon with air to determine the presence or absence of a latent or chronic appendicitis. Since our first report of the test, we have applied it in a large number of abdominal cases, and have been able in a number of instances to establish the diagnosis of appendicitis when all other methods of examination failed completely or left the examiner in a state of reasonable doubt. In addition, we have received verbal reports from several surgeons who have been employing the test as a routine in their hospital cases. In their experience, as well as our own, the test as checked by operation has proved reliable, failure being reported in only 4 or 5 cases in several hundred.

To make the test a colon tube is passed 11 or 12 inches into the rectum and air injected by means of an atomizer bulb. If, as the colon distends, pain and tenderness to finger-point pressure become apparent at McBurney's point, there is appendicitis. We have compared the test in a number of instances with the Rovsing test and

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find the dilatation test much the more certain; but at times, after moderate dilatation with air, the Rovsing method of forcing the air back into the cecum may be used with advantage. We might sound a warning that if most of the air is not allowed to escape before withdrawal of the tube, colicky pains are likely to ensue.

The test is not needed in an acute case, and in such would be contraindicated; neither is it required in an undoubted chronic case. But the indication for the test is a suspected chronic or latent appendicitis, or any persistent digestive or abdominal disturbance, in which no cause can be found for the trouble. Ordinarily one may entertain doubt about the diagnosis, or at least hesitate about urging operation, when tenderness at McBurney's point can be elicited only on very deep pressure, or is accompanied by a similar tenderness elsewhere in the abdomen. At times, for example, we have thought of appendicitis because of McBurney's point tenderness, but have found in addition puzzling points of tenderness along the transverse colon or at a spot on the left side corresponding with McBurney's. In such cases, dilatation frequently results in the disappearance of all the points of tenderness except that at McBurney's, which it intensifies.

Again, in persistent cases of hyperchlorhydria or gastrosuccorrhea the test should be performed. For just as in the case of a cholecystitis, so a latent appendicitis may have its chief manifestation in stomach derangement, even so marked at times as to simulate an ulcer. And since it has become our routine practice to dilate the colon in all long-standing cases of the kind, we have had the good fortune in a number of instances to discover an unsuspected appendix and to see the gastric symptoms disappear with the removal of the offending vestige.

A further application of the test may be to distinguish between an inflamed appendix and a right-sided pelvic trouble. Pain and tenderness in a right-sided chronic salpingitis or cystic ovary sometimes result from the colon dilatation, but the tenderness is regularly less acute, is low down in the abdomen, and extends toward the middle line. In three instances we have been able to diagnosticate pelvic inflammation in young women in whom appendicitis was suspected and in whom a vaginal examination was impossible except under ether. In each of these the subsequent operation revealed a cystic right ovary and a free uninvolved appendix. We have employed the test in not a few other gynecological cases, and while in some we have been able merely to corroborate the findings of a vaginal examination, in others we have demonstrated appendicitis in addition

to the pelvic lesion. In every such case operated upon the finding of the dilatation test has been found correct.

A few typical case reports may be of interest:

Case 1.—G. L., painter, has had attacks of pain in the abdomen at intervals for 1½ years, without nausea or vomiting. Recently such attacks have become more numerous, and in the last, he had to lie down for one afternoon; he was thought to have painter's colic. He told us that the pain occurred mostly just above the umbilicus or high up beneath the right ribs. He had no lead line on the gums, no polychromatophilia in the blood. On colon dilatation, the gall-bladder was not made out, and no pain appeared in the hepatic region; but in 2 spots there were pain and tenderness, 1 spot just at McBurney's point and another just below the navel. A small umbilical hernia also made its appearance. The patient was advised to have an operation for the hernia and the appendicitis, but as the diagnosis was not confirmed by others he was treated for 3 months for lead poisoning, intestinal indigestion, and rheumatism. The attacks, however, increased and were more localized in the appendix region, so he returned for operation. The dilatation test was again positive, and operation was performed by Dr. H. H. M. Lyle. The chronically inflamed appendix was covered by veil-like adhesions, which extended to the hernial opening; the gall-bladder contained no stones. The appendix and adhesions were removed and the hernia closed. The patient has had no more attacks of the old type, and a little recurrence of the pain beneath the right ribs disappeared quickly under treatment for hyperchlorhydria.

Case 2.—D., a physician, in 2 years had 5 attacks of severe pain in the abdomen, with prostration and vomiting. The pain was always diffuse, never localized, and lasted about one day; the temperature never rose above 99° F., and the pulse was normal or slow. Physicians had suggested appendicitis, but no positive diagnosis was made. Two days after the last attack, which was so severe that morphine had been administered, the patient walked to my office apparently well. Slight tenderness to finger-point pressure at McBurney's point could be elicited only on very deep pressure, but on dilating the colon an acute pain appeared in the appendix region, and tenderness over an area as large as a silver dollar and centering over McBurney's point. Four days later, Dr. J. A. Blake operated and found a chronically inflamed appendix with a constriction close to the cecum, and adhesions extending upward over the cecum. The patient has had no attack since the operation (about 2 years).

Case 3.—Mrs. R., 12 years ago, had an attack of pain in the abdomen, with vomiting, and was in bed 1 day. A surgeon saw her in the attack, and said it was not appendicitis. During the entire 12 years since then she has taken a laxative pill every night and has had no further severe pain, but for the last 6 months has been losing appetite and becoming more costive, and has been irritable and in low spirits. A month ago had a little abdominal pain on the right side for 1 day, but not enough to require treatment. A test breakfast showed free hydrochloric, 48; total acidity, 70. No organic

acid. On dilatation of the colon, pain at McBurney's point with sharply localized tenderness became manifest. It was our belief that hyperchlorhydria treatment would be futile in the presence of a chronic appendicitis, so operation was advised, and Dr. L. W. Hotchkiss removed a retrocecal swollen appendix with 3 marked constrictions and surrounded by adhesions. Since the operation, 8 months ago, the patient has had unusually good digestive and bowel functions, and has been in excellent general health and spirits.

Case 4.—Miss H., aged 24 years, a rather under-developed young woman, with a mitral stenosis, has had in the last year several attacks of cramp-like pain in the right iliac region. Two or 3 times this pain came at the menstrual period, but it occurred also at other times. Vaginal examination was not feasible, so the colon was dilated. At once there was a dull pain over the whole lower right segment of the abdomen, extending from McBurney's point to Poupart's ligament and to the midline. Tenderness was slight, and was most pronounced about half way between McBurney's point and the symphysis pubis. Operation by Dr. H. T. Goodwin showed a right ovarian cyst and a normal appendix.

Case 5.—Miss G., aged 27 years, has had pain in the right side low down for a year or more. It has never been very acute, never caused vomiting, and was most pronounced after the patient had been a long time on her feet. There has been a rather abundant vaginal discharge. Examination per vaginam reveals a tender boggy mass in the right fornix, and much tenderness when the uterus is moved. Out of curiosity, the colon was dilated, and to our surprise an acute pain appeared in the appendix region, and tenderness localized at McBurney's point. Operation by Dr. Frank Markoe showed right salpingitis with tube, ovary, and chronically inflamed appendix bound together in a mass of adhesions.

Case 6.—D. S., has never had any acute attack of appendicitis, but has had some pain in the appendix region when his bowels seemed full of gas. Dilatation was positive for appendicitis. Some time later, in Chicago, he had an acute attack which was diagnosticated appendicitis, and though prostrated, and with fever, he took train immediately for New York. Dr. H. H. M. Lyle operated and found a retrocecal abscess with a sloughed off appendix.

These cases illustrate the positive findings of the dilatation test. In the use of the test during the last four years we have had no case in which the test was positive and the operation findings negative. But in two out of all of our cases the negative finding of the test, after a supposed appendix attack, was followed within six months by a typical attack of appendicitis, and the test was, therefore, presumably at fault. Several times in the early days of the test, surgeons operated for a suspected appendicitis, though the test was negative, and in every such case the appendix was found normal. We have had a verbal

report from one surgeon who has used the test extensively, of two cases which gave positive test but negative findings at operation. With very few exceptions, therefore, the test has proved accurate, and it may well serve as a diagnostic guide in the three classes of cases mentioned, viz., suspected chronic appendicitis, persistent gastro-intestinal or abdominal disturbance with unknown cause, and appendicitis versus ovarian or tubal inflammation.

THE VACCINE TREATMENT OF TYPHOID FEVER.

Austin W. Hollis, M.D., and Norman E. Ditman, M.D.

During the past few years the undoubted success of the preventive inoculation against typhoid fever has been proved. Among the 60,000 men in the United States army who have been inoculated against typhoid fever during the past three years, there have been no deaths from typhoid, and but 12 cases of fever have occurred.

These figures furnish evidence beyond dispute that the use of typhoid vaccine in the amounts now employed, at least in men in health, produces very real immunity.

The practical question of interest which now remains to be solved is, how late in the course of an attack of typhoid fever is it advisable to attempt to aid or increase the immunity which the sick subject is attempting to establish, and what benefits, if any, are to be gained for the attack of illness already in progress.

It has been difficult to predict, on theoretical grounds, what the effect would be of adding bacterial products to a case of illness apparently already overburdened with products of a similar nature; yet, experience is beginning to show that while an attack of typhoid fever of average intensity may seriously impair the activities of the person attacked—producing the picture of severe illness—yet their powers of bacterial resistance through increased immunity may still be greatly augmented.

During the past few years one fact of undoubted value has been clearly proved—rendering the path clear and safe for future work along this line. That is, that the administration of typhoid vaccine to a case of typhoid—unless that case be moribund from an overpowering toxæmia—produces no harm or undesirable symptoms of any kind.

During the past 3 years, on the service of Dr. Austin W. Hollis, typhoid vaccine has been administered uniformly to cases of typhoid

fever. The doses have consisted of 1 c.c. of Parke, Davis & Co. typhoid vaccine every other day—each c.c. containing 50,000,000 dead bacilli.

In the 1909 series, 11 cases were thus treated. No deaths occurred.

In comparison with 21 unvaccinated cases, there were no deaths to 4, 30 per cent of relapses to 10 per cent, 34.3 days duration of fever to 36.7 and an equal number of hemorrhages. In the 1910 series of 40 vaccinated cases, the mortality was 5 per cent, relapses 10 per cent, no hemorrhages and no perforations, with an average fever duration of 30 days.

In the 1911 series of 35 cases, the mortality was 8.5 per cent, there were 2.8 per cent of relapses, 5.7 per cent of hemorrhages, no perforations and an average fever duration of 28 days in the non-fatal cases.

During this same year, in other services of St. Luke's Hospital, in 35 cases where typhoid vaccine was not administered, the mortality was 14.3 per cent, there were 23 per cent of relapses, 2.9 per cent hemorrhages, 2.9 per cent of perforations and an average fever duration of 33.2 days in the non-fatal cases.

Summarizing the available statistics for the 3 years, the following results are obtained:

	Vaccinated cases		Unvaccinated cases	
	Number	Per cent	Number	Per cent
Cases	86	....	56	....
Deaths	5	5.8	9	16.
Relapses	9	10.4	10	17.8
Hemorrhages	2	2.3	1	1.8
Perforations	0	0	1	1.8
Average duration of fever	30.3 days		33.7 days	

Still better results are reported in recent literature as follows:

In 214 vaccinated cases collected by Callison, the mortality was 5.6 per cent, with relapses in 5.1 per cent of the cases.

To realize how vaccination influences the course of typhoid fever, it is of interest to compare these results with those of a very large series of typhoid fever cases collected by Osler, in which the standard methods of treatment were employed. They are as follows:

	Per cent
Mortality	11.2
Relapses	11.4
Hemorrhages	7.0
Perforations	5.7
Average duration of fever	29.4 days

It is therefore apparent that, as far as the present total of statistics goes, there is an appreciable difference in favor of the vaccinated cases. The proper dosage and frequency of administration is yet to be determined.

In some of the recent cases of the St. Luke's series some doses of more than 50,000,000 were given, and in a small number, on alternate days, small doses of Schaeffer's vaccine was given hypodermatically.

In the St. Luke's non-vaccinated cases, tub baths were given, and a diet ranging from 2,000 to 3,200 calories; while in the vaccinated cases the diet did not exceed 1,500 calories and the tub bath was dispensed with—its place being taken by sponges.

In any series of hospital cases which are unselected, a number of cases are found which enter the hospital late, in a more or less moribund or hopeless condition.

Among the fatal cases in the vaccinated series these are frequent. Thus, in the 1910 series, both fatal cases entered the hospital in the fourth week and died 8 and 4 days after admission—the latter from pneumonia, 6 weeks after child-birth.

In the 1911 series, of the 3 fatal cases receiving vaccines, one entered the hospital on the 16th day, in an extremely toxic condition, and died on the 20th day—having received but 1 dose of vaccine. One case was admitted on the 16th day, in an extremely toxic condition, and died from a hemorrhage on the 25th day. The third case was admitted on the 28th day, and died from a hemorrhage on the 34th day.

Therefore, it may be said that while vaccination has little influence on late cases, yet, on the other hand, if vaccination is begun early, good or even brilliant results may be expected. For, among the large number of St. Luke's cases during 3 years of observation, in which vaccination was begun before the 16th day, there were no deaths. On the other hand, it must be remembered that had all the cases of the unvaccinated series entered the hospital before the 16th day, the percentage of mortality in that series would undoubtedly have been much lower.

Observers in general who have seen vaccinated and unvaccinated cases, seem to agree that in the vaccinated cases the "typhoid state is rare, the early toxic symptoms of the disease quickly disappear and the disease in general is better borne."

Major Russell, of the United States army, has shown that in normal subjects the typhoid immunity reaction does not begin until about

7 days after the vaccination. Whether the same holds true for fever cases might be very difficult to determine. A fall in temperature has often been noted within 48 hours after vaccination; but it is seldom that any pronounced improvement begins until after the sixth day from the first vaccination.

If vaccine has been used up to the 25th day of the disease, it is believed that its further use is not likely to be of benefit; while a long continued fever of the septic type is more likely to be benefited by some other form of treatment, and we have undoubtedly seen these cases clear up quickly under combined vaccine.

From the experience of the past 3 years in St. Luke's Hospital, it is believed that it may be well to give an initial dose of 50,000,000 as early in the disease as possible, repeated every other day until the tendency of the fever is downward, when the dose may be doubled at every succeeding injection, provided the fever is still declining. Injections should be continued until there is no danger of a relapse—avoiding, however, a dosage which might be considered excessive in amount.

Whether this maximum dose will prove to be 500 million or 2,000 million, statistics of the next few years will prove.

A CASE OF PAGET'S DISEASE.*

KARL M. VOGEL, M.D.

In spite of the fact that ostitis deformans, or Paget's disease, as it is generally called, is not a remarkably rare condition, at least in its minor grades, the diagnosis is not very often made, and it is only comparatively recently that cases have begun to be reported with any degree of frequency.

Paget, in 1877, first isolated this type of deformity from the general hodge-podge of chronic bone diseases, and in the Medico-Chirurgical Transactions for that year outlined a clinical picture to which subsequent observers have made few additions of moment. As he described it, the disease is one beginning in middle life or later, progressing very slowly during many years, and causing no disturbances other than those due to mechanical changes in the diseased bones. Those most often involved are the long bones of the lower extremities, the cranium, spine, and clavicles. The bones enlarge and soften, and owing to the pressure of the body weight become curved and misshapen, so that finally with the shortening thus produced, as well as through curvature of the spine, the stature steadily decreases. The pain is variable in severity and is most common in the earlier stages of the disease, though it may persist indefinitely.

In a later communication, Paget[1] summed up the most prominent symptoms, as follows:

"It usually affects many bones, most frequently the long bones of the lower extremities, the clavicles, and the vault of the skull. The affected bones become large and heavy, but with such weakening of their structure that those which have to carry weight or to bear much muscular traction become unnaturally curved and misshapen. The disease is very slowly progressive, and is felt only in pain, like that of rheumatism or neuralgia, in the affected limbs, and in increased heat at the tibiæ. But neither the pain nor the heat is

*Read at a meeting of the Section on Medicine of the New York Academy of Medicine, May 16, 1911, and reprinted from the Medical Record July 29, 1911.

[1]Paget: Medico-Chirurgical Transactions, London, vol. lxv, 1882.

constant, nor do they continue during the whole progress of the disease; and pain has not been observed in the head even in the cases in which the skull was greatly thickened. There is not any clear evidence of general disturbance of health. In all the cases traced to the end of life, death has ensued through some coincident, not evidently associating, disease, which has been aggravated by the condition of the bones only in so far as they may have diminished the range of breathing and the general muscular activity.

"In all of the cases I have seen, the general appearance, postures, and the movements of the patients, have been so alike that these alone might often suffice for the diagnosis of the disease. The most characteristic are the loss of height, indicated by the low position of the hands when the arms are hanging down; the low stooping, with very round shoulders and the head far forward, and with the chin raised as if to clear the upper edge of the sternum; the chest sunken toward the pelvis, the abdomen pendulous; the curved lower limbs, held apart, and usually with one advanced in front of the other, and both with knees slightly bent; the ankles overhung by the legs, and the toes turned out. The enlarged cranium, square-looking or bossed, may add distinctiveness to these characters, and they are completed in the slow and awkward gait of the patients and in the shallow costal breathing, compensated by wide movements of the diaphragm and abdominal wall, and in deep breathing by the uplifted shoulders."

In regard to the order of involvement of the bones it may be stated that Packard, Steele, and Kirkbride,[2] from an analysis of a very large number of cases, found that this was as follows: Skull, tibiæ, femur, spine, pelvis, clavicles, ribs, radii, ulnæ. The fibulæ seem to be but rarely affected, but Maier has reported a case in which the disease began in one fibula and in the small bones of the foot.

Paget correctly interpreted the condition as being a chronic inflammatory process, and accordingly suggested that it be known as ostitis deformans. Recklinghausen termed it ostitis fibrosa. The changes in the bone structure may be regarded as the result of two opposing processes, resorption and hyperplasia; that is, a rarefying and a condensing ostitis. Both the spongy and the compact portions of the bone are involved, the destruction of the lamellæ being accompanied by replacement with fatty, gelatinous, or fibrous tissue which frequently shows localized areas of softening and liquefaction, so that cyst-like cavities filled with fluid develop. In addition, calcification occurs and irregular deposits of new bone are formed throughout the entire substance of the bone, resulting in an increase in its size and density. The marrow becomes converted into a more or less fibrous or gelatinous mass containing giant cells, fat cells, and leucocytes.

[2]Packard, Steele and Kirkbride: Am. Jour. Med. Sciences, vol. cxxii, 1901.

Various authors differ somewhat in their views as to whether the process begins subperiosteally or in the marrow, and as to the precise sequence of events; but the essential features are the combination of softening and curvature of the bone, together with an increase in size and density, so that the picture presented by the individual bones may be very variable, and the processes of halisteresis, absorption, and calcification of newly formed osteoid tissue may be going on simultaneously. The surface of the bone may be smooth or rough; the cortex compact or spongy; the cancellous portion dense or porous; the central canal almost obliterated or widely dilated.

In regard to the etiology little more is known than in Paget's time. General arteriosclerosis is constantly present and it has been suggested that the bone lesions are due to sclerosis of the nutrient vessels of the bone. French observers have considered that the disease is a manifestation of hereditary syphilis or a paraluetic condition, and have reported improvement as the result of mixed treatment. Nerve lesions and gout have also been mentioned as possible etiological factors, but without very satisfactory confirmatory evidence. Joint changes occur, but are not very common, although Richard and Ziegler hold that the disease is allied to arthritis deformans. Prince[3], who regarded the disease as a trophic disorder, has laid stress on the possibility of there being changes in the central nervous system, but as yet nothing significant has been discovered. An interesting fact noted by Paget himself, as well as by later writers, is the comparative frequency with which new growths of various sorts, including osteosarcoma, occur in the subjects of the disease. A possible hereditary predisposition has been alleged, since in a number of instances members of the same family have been victims of the disorder. Bockenheimer[4] holds that a congenital anomaly of bone metabolism is an underlying factor.

The direct prognosis, as far as life is concerned, is good, though a subject of the disease, through arteriosclerosis and through local conditions dependent upon the deformity, may be more susceptible to the disorders of old age than an individual not so affected.

The treatment is largely symptomatic and consists chiefly in controlling the pain, when present, though some writers have reported encouraging results from the administration of thyroid extract. Attempts at surgical correction of the deformity of the long bones are

[3]Prince: Am. Jour. Med. Sciences, vol. cxxiv, 1902.

[4]Bockenheimer: Arch. f. klinische Chirurgie, vol. lxxxv, 1908.

contraindicated. In one case of Sonnenberg's[5] in which an osteotomy was performed there was no evidence of callus formation 13 months later.

The question of diagnosis is a more complex one, for while it is not very difficult to recognize the disease when well advanced so that the curvature and enlargement of the bones are apparent, the head is misshapen, the stature shortened, and the patient presents the typical ape-like aspect, in its earlier stages or in mild cases, when, perhaps, only a single bone is involved and the patient complains solely of fleeting pains, it is no doubt often mistaken for sciatica, rheumatism, neuralgia, arteriosclerosis, etc., and many cases are accordingly overlooked. Among the bone diseases that might be confounded with it are osteomalacia, spondylitis deformans, hypertrophic pulmonary osteoarthropathy, and possibly acromegaly, but these all differ in more or less striking particulars from Paget's disease when well developed. One form of disease, however, requires especial mention in this connection, and that is the diffuse enlargement of the skull described by Malpighi in 1697, and to which Virchow gave the name of leontiasis ossea, because, as he said, in looking at representations of such skulls one is reminded of the appearance of the plaster cast of a case of leprous leontiasis. Later authors, however, prefer the designation "diffuse cranial hyperostosis." Most of the writers on bone disease have apparently taken it for granted that this condition and Paget's disease are independent affections, and have given various differential points by the aid of which the two might be distinguished. These relate chiefly to the age at which the disease first appears, and to minor distinctions in regard to the nature of the bony changes, extent of involvement of the fissures and foramina of the skull, the occurrence of nervous disturbances, etc. Recent authors, however, notably Bockenheimer, Prince, and Fitz[6], have suggested that the two conditions are certainly closely allied and probably identical. But the question has remained a somewhat open one because until rather recently information as to the leontiasis skulls was largely derived from more or less ancient museum specimens generally provided only with inadequate clinical histories, so that the possible coexistence of slight changes in other bones could not be altogether excluded. Latterly more definite data have been available, for the microscopic examination of tissue removed at operation on leontiasis patients for the pur-

[5]Glaessner: Wien. klin. Wochenschrift, 1908, p. 1327.

[6]Fitz: Am. Jour. Med. Sciences, vol. cxxiv, 1902.

pose of relieving pressure symptoms has revealed changes precisely similar to those found in the long bones in Paget's disease. An important contribution to the matter was made not long ago by Max Koch[7], who presented before the German Pathological Society the skull of a carefully observed patient clinically suffering from leontiasis, which showed on section the usual appearances of Paget's disease; so that it now seems rational to regard leontiasis ossea as a local occurrence of lesions which when more widely distributed produce the picture of Paget's disease.

The history of Koch's case is briefly as follows:

The patient was a woman of 65 years, who was observed during a year's stay in the hospital. The symptoms began twenty years previously, when she noticed an increase in the size of the head and at the same time tinnitus, vertigo, headache, and impairment of vision developed. Her hearing became impaired only a few months before her entry into the hospital. The circumference of the head was 72 cm., or about 29 inches. There was pronounced enlargement of the temporal arteries, and the ears stood out prominently from the head. Ophthalmoscopic examination was negative, and the visual fields were not restricted. Myopia of —5 D. S. There were hyperostoses of both external auditory meatuses and hypertrophy of the inferior turbinates and nasal septum. The hard palate was so much thickened that the laryngoscope could not be used. The lower jaw was not thickened. The Wassermann reaction was negative. During her stay in the hospital she suffered chiefly from headache, pain in the legs, vertigo, and general weakness. Five days before death she suffered from an apoplectiform attack, with unconsciousness, but no paralysis. Death appeared to be due to a general loss of strength. At the autopsy it was found that there was a synostosis between the axis and third cervical vertebra. There was a moderate kyphosis in the thoracic region, but no abnormality of the other bones. The blood-vessels were markedly sclerotic. On sawing through the skull, it was found that the differences in structure between the external and internal tables and the diploe had disappeared, and the bone was very friable. The thickness of the frontal bone was from 4 to 6 cm., that of the temporal from 3 to 3.5 cm., and of the occiput, 2.5 to 3 cm. There was an abscess cavity in the situation of the frontal sinus on one side, but on the other the sinus had been entirely obliterated. The cut surface was made up of spongy bone, with grayish-red, dense, fibrous tissue, with here and there areas of gelatinous marrow, or cavities filled with fluid. All the foramina and fissures of the base were much narrowed, except the foramen ovale. The meninges were normal, but the brain appeared flattened and diminished in size through pressure. The pituitary body was flattened, but in section showed no abnormalities.

Microscopical examination of the bones showed the changes described by Paget as ostitis deformans, and by von Recklinghausen[8] as ostitis fibrosa.

[7]Koch: "Verhandlungen der Deutsch. patholog. Gesellsch.," 1909.

[8]v. Recklinghausen: "Virchow's Festschrift," 1891.

Fig. 1.—Photograph of patient at the age of 34 years.

Fig. 2.—Photograph of patient at the age of 44 years.

Fig. 3.—Present appearance of the patient.

Fig. 4.—Present appearance of the patient. Note especially the appearance of the ear.

Koch accordingly believes that this case definitely proves the identity of leontiasis ossea with Paget's disease.

The history of the case forming the subject of the present report is as follows:

The patient was a woman, aged 68, born in England, a seamstress. Admitted to St. Luke's Hospital on December 5, 1910, to the service of Dr. Janeway.

Family history negative, except that one sister is said to have died of cardiac trouble, and various members of the family have had "weak hearts." There is no history of bone changes similar to those of the patient, and all other members of the family are well formed, active individuals.

Previous history: When about two years old her head was caught between a clothes mangle and a door. She says that her head has always been large and ill-formed, and believes that this is due to this accident. She has no remembrance of the diseases of childhood. At eleven years of age, after a fright, she had "fits," during which she would become unconscious, but she does not remember falling or hurting herself during these attacks. A short time later she awoke one morning and found that her right side was paralyzed. For a time she had to be fed, and helped in walking, but the paralysis gradually disappeared, and she had no more of the fits after her sixteenth year. About twenty-five years ago she had several abscesses in the left external auditory canal, and at the same time that side of her head became swollen and bumpy. She believes that the present swelling above her ear is due to this cause.

Present illness: Dates back about fifteen years. Previous to this time she had always been a good walker, but she then began to notice a feeling of weakness in her legs, which gradually increased, until about ten years ago, when she was obliged to give up her work, as it took her so long to do anything, and she could not think quickly. If she wished to do anything across the room, it would take her half an hour in thinking about it and in getting up and crossing the room. She had some dull pains in her legs at this time, and also began to grow deaf. During the last five years the pains in her legs have become more severe. Her gait has become very slow and shuffling, and she cannot lift her feet. Two years ago she noticed that the legs were becoming crooked. She has suffered from headache most of her life, and lately her head has felt heavy, so that it drops forward and it is hard for her to lift it. She is afraid to lift her head too high in looking at things, for fear she will fall over backward. She believes that she has become two inches shorter during the past five years. She is of a hysterical nature, and has always cried easily, but she has found that lately she cannot shed a tear, and has also found that the bridge of her nose has grown too large for her glasses. For four or five years her legs have been more or less swollen, and recently her arms also have become edematous. For about a year she has been short of breath, and the veins in her neck and on her forehead have become prominent.

The patient is depressed, realizing fully her slow mental processes and her inability to move or act quickly.

Chief complaints: Shortness of breath and swelling of arms and legs.

Physical Examination.—General condition: Patient is a rather poorly nourished woman, of small frame, past middle age, showing moderate dyspnea, but only slight cyanosis. Her appearance is very striking, on account of the marked disproportion between the size of the head and that of the body. The head is very markedly enlarged, especially in the upper part; it is rather square in shape, with pronounced bony protuberances above each ear. Circumference about the forehead is 65 cm., or 26 inches. In the temporal region and in the neck are numerous markedly distended pulsating veins. The skin of the face appears rather pale and pasty, with numerous brownish pigmented areas. Eyes: Pupils equal, and react to light and accommodation. Tongue clean. Throat normal. Upper teeth artificial; lower in fair condition. Chest poorly developed; slight protuberance of upper part of sternum. Heart: Rather diffuse pulsation over the lower precordium. The apex impulse is fairly well marked in the fifth space, four inches to the left of the median line. The right border is one inch from the mid-line; the left border merges with the dulness of the left chest. At the apex there is a loud, blowing, systolic murmur, transmitted to the axilla, and heard over the entire lower left chest. The second sounds are not accentuated; the action is rapid and irregular. The pulse is rapid, irregular, of poor force and moderate tension. The vessel wall is thickened. Lungs: Clear anteriorly; posteriorly, there is dulness, beginning just above the angle of the scapula on both sides, and becoming flat on approaching the base. Over this area there is diminished breathing, becoming absent at the base, where numerous subcrepitant râles are heard. The abdomen is somewhat distended. The liver percusses three inches below the free border, where its edge can be felt distinctly. It is markedly tender, and pulsates. Extremities: Both legs are markedly edematous, and show moderate curving of the tibiæ.

The patient's height at present is 4 feet 10⅞ inches, whereas she says that previously it was 5 feet 1½ inches, a shortening of a little over 2½ inches. She is very deaf, but examination of the ears shows no occlusion or deformity of the external meatus. The drums show several patches of fibrous thickening.

In conclusion, it may be said that a survey of the literature seems to support the contention that the separation of diffuse cranial hyperostosis as an independent disease is not justified and that it is to be regarded as a manifestation of the same process which is responsible for the symptom complex of Paget's disease. Further evidence in favor of this view is furnished by a case recently reported by Bartlett,[9] in which, as in that described by Koch, during life there was no sign of the involvement of any bones except those of the skull. At the autopsy, however, examination of the femur showed beginning foci of disease.

[9]Bartlett: Yale Medical Journal, 1909, p. 367.

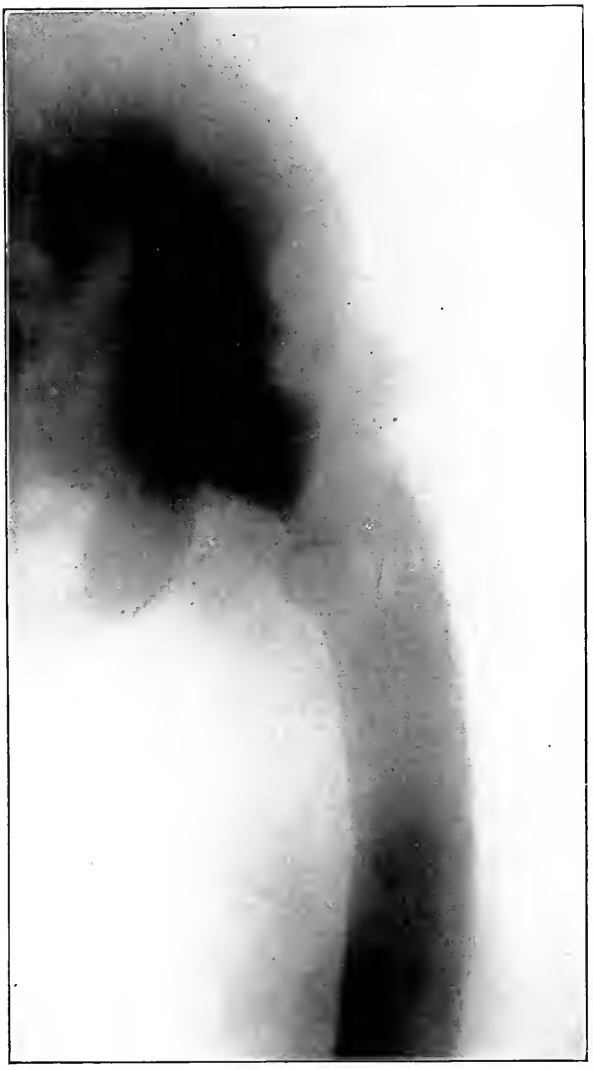

Fig. 5.—Radiograph of the pelvis and femur.

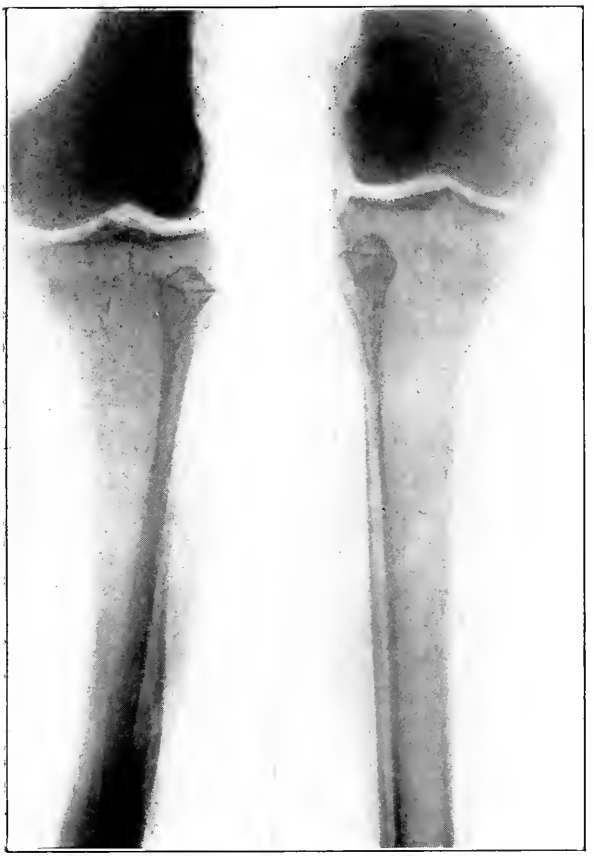

Fig. 6.—Radiograph of the tibiæ.

THE PURIN CONTENT OF FOODSTUFFS.*

KARL M. VOGEL, M.D.

The importance of considering the purin content of the diet in the diagnosis and treatment of certain metabolic disorders has recently been emphasized by numerous writers, for example, by Bessau and Schmid,[1] and by Brugsch and Hesse.[2]

The following determinations of the purin content of some of the commoner foodstuffs were begun in the fall of 1908, in the II Medical Clinic in Munich, at the instance of Prof. Fr. Müller, and were continued in the laboratories of the College of Physicians and Surgeons and of St. Luke's Hospital. In the meanwhile, the publication of Bessau and Schmid's table made it seem unnecessary to continue in this direction, but since then Hesse has reported the results of some analyses made by him. His figures in general are higher than those of Bessau and Schmid, and as mine correspond more closely with the latter, it appears of some interest to record them also.

Hesse, in publishing his figures, calculated the presumptive amount of purin bases corresponding to the nitrogen values found. In order to make his results comparable to those of other authors who have followed the practice of giving the nitrogen content of the purin precipitates, I have calculated the nitrogen equivalent of his values, employing the customary factor 2.65. One column of the table, however, contains his original figures. In the first four analyses of meat and organs I used the method of Burian and Hall;[3] the other determinations were made by means of the copper-bisulfite method.[4] Of the meats, 100 to 250 grams were taken, and of the other articles 250 to 500 grams.

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*Translated from the Münchener medizinische Wochenschrift, No. 46, 1911.

[1]Bessau und Schmid. Therap. Monat., No. 3, 1910.

[2]Brugsch und Hesse. Med. Klinik, No. 16, 1910.

[3]Burian und Hall. Ztschr. f. physiol. Chem., xxxviii, 336.

[4]Krüger und Schittenhelm. Ztschr. f. physiol. Chem., xlv, 15.

	Percentage of purin N				% of purins
	Walker Hall	Bessau and Schmid	Vogel	Hesse	Hesse
Beef: Sirloin	0.0522	0.037	0.059*	0.0666	0.175
				0.0720	0.189
Liver	0.1101	0.093	0.099	0.142	0.372
Sweetbread (thymus)	0.4025	0.330	0.398	0.498	1.308
Spleen	.....	.....	0.196	.....	.....
Codfish	0.0233	0.038	0.040*	0.0499	0.131
Wheat flour	.....	.....	0.001	0.0441	0.116
Rye flour	.....	.....	0.002	0.0365	0.096
Pea flour	0.0156	.....	0.016	0.0411	0.108
Arrowroot	.....	.....	0.001	.....	.....
White bread	0	0	0.008	.....	.....
			0.005*		
Rye bread	.....	trace	0.014	.....	.....
Hominy	.....	.....	0.004*	.....	.....
Oatmeal	0.0211	0	0.030	.....	.....
Rice	0	0	0.0004*	.....	.....
Potato	0.0007	0.002	0.001*	0.0072	0.019
Spinach	.....	0.024	0.022*	.....	.....
Tomato	.....	0	0*	.....	.....
Milk	0.0002	0	0.0002	0.0038	0.010
Swiss cheese	.....	0	0.0004	.....	.....
Eggs	0	0	0	.....	trace

*Refers to analyses made on American material.

ACUTE BICHLORIDE OF MERCURY POISONING—A REPORT OF TWO CASES WITH RECOVERY.

LEFFERTS HUTTON, M.D.

The following two cases are reported on account of the apparently prevailing custom of keeping bichloride of mercury "Blue Tablets" near the fountain syringe to prevent conception. Instead of using a bichloride douche, the tablet (7½ grains) was inserted into the vagina. In looking over the literature on this subject, one is impressed with the small number of cases of acute poisoning resulting from this practice.

In Germany a law was passed in 1897, making all cases of acute bichloride of mercury poisoning reportable. During the next 9 years, 101 cases of mercury poisoning, from the tablet form, were reported, and no record of any case of poisoning from the insertion of the tablet into the vagina. The official report for the past 5 years has not yet been published, but probably the result will be the same as in the preceding 9 years. The sale of mercury in any form is prohibited, except on a physician's order.

In England and her colonies the writer was unable to find any case of poisoning by this method. While in America, where any one is able to buy the "Blue Tablets," 7 cases have been reported in the past 10 years. To this collection of 7 cases the author wishes to add 2 more, as follows:

Patient.—Mrs. B.; 38 years old; born in the United States; occupation, housework. Entered St. Luke's Hospital as a private patient of Dr. Henry S. Patterson, on November 21, 1911, giving the following history:

On morning of admission, at about 2 A. M., patient inserted a 7½-grain tablet of bichloride of mercury in her vagina to avoid conception. Soon after she complained of intense burning sensation in that region. Later, the patient attempted to douche herself, without much success. She then began to realize the gravity of the situation and came to the hospital.

On entrance, she complained less of the pain in the vagina than of peculiar paresthesia and cramp-like sensations in the hands and feet. She was

not salivated. There were no urinary or intestinal symptoms. Physical examination was negative, except for a good deal of redness of the vulva, with some whitish slough and a sero-sanguinous discharge from the vagina.

Vaginal examination, with a bivalve speculum, showed the mucous membrane to be covered with a whitish slough—cervix very red.

Treatment and Subsequent Course.—The treatment consisted of force fluids, alkaline douche 3 times a day, and a colon irrigation 116° twice a day.

Blood.—W. B. B. 16,000. Poly. 73.5. Lymph 26.5. Hgb. 90 per cent. Her urinary excretion ranged from 64 to 144 ounces a day, while her fluid intake varied from 112 to 196 ounces.

Urine Examination.—Alkaline, sp. gr. 1006-1008, very faint trace of albumen, no sugar, a very few hyaline casts.

Stools.—No blood; no increase in number.

Mouth.—No ulcerated areas; no salivation.

The vagina, under the alkaline douches, cleared up very rapidly. She was discharged cured, 7 days after onset.

Mrs. P., age 33, born in Italy, occupation factory hand, entered Dr. S. W. Lambert's service at St. Luke's Hospital, on December 8, 1910, with the following history:

Chief Complaint.—Pain in lower abdomen and a sore mouth.

Present Illness.—Three days ago was seized with sudden pain in lower portion of abdomen. This pain was sharp, non-radiating, worse in daytime, when at work in the shop. Vomited twice with onset. No chills, fever nor cough. Mouth has been sore for the past 3 days, with increased salivation. Teeth not loosened. Some difficulty in eating—no treatment during present illness.

Past history was negative. Menstruated 2 weeks previously; some leucorrhœa.

Physical Examination.—Breath fetid, foul-smelling. Lips dry and cracked. Sordes on teeth and gums. Tongue badly coated—moderate salivation. No evidence of any ulceration in mouth.

Heart normal. Lungs clear. Abdomen—no masses, no tenderness. Liver, spleen, kidneys apparently normal.

Vagina.—Yellow and dark brown sloughs on inner side of each labium majus. Also yellow and white sloughs on both sides of the vaginal canal. The cervix is swollen red, except the places which are covered by slough whitish in appearance. A bimanual examination was not made.

Treatment consisted of bicarbonate of soda throat irrigation. Temperation of 120° every 3 hours. Alkaline vaginal douche. Colon irrigation of hot saline.

On cross-examination, patient confessed to having placed two bichloride of mercury tablets in her vagina 3 days previous to admission and had not taken any treatment previous to coming to hospital.

Subsequent History.—Her vaginal and cervical condition slowly healed. Her urinary excretion ranged from 18-30 ounces a day, which, on repeated examination, showed a high sp. gr. 1030. No albumen. No blood. No sugar. No casts.

Three days after admission patient developed an ulcerative stomatitis, which slowly healed.

On December 23d, 2 weeks after admission, the patient demanded her discharge from the hospital. At this time she had some pyorrhœa along the gums, otherwise her mouth had healed. Vaginally, her cervix was congested and slightly eroded. Vagina congested. No ulcers seen. Although not entirely cured, she insisted upon leaving the hospital, which was reluctantly granted.

A CASE OF LATENT DISSECTING ANEURISM OF THE AORTA AND RUPTURED SACCIFORM ANEURISM.

LEFFERTS HUTTON, M.D., and J. GARDNER HOPKINS, M.D.

The following case is presented on account of the unsuspected findings at autopsy.

Mrs. E., married, age 52, born in the United States, occupation housework, entered the hospital March 25, 1911, on the service of Dr. Samuel W. Lambert. She gave the following history:

Chief Complaint.—Cough, fever, and pain in the right side.

Present Illness.—Eight days ago the patient was suddenly seized with a severe shaking chill, lasting about five minutes. This was followed by fever, a dry, hacking cough, headache, and prostration. She also vomited several times. Twenty-four hours later she had another chill, not so severe as the first. This was followed by fever and a sharp, stabbing pain in the right side, increased by coughing and deep breathing. Her abdomen felt somewhat sore, and was distended. Since onset, the patient has been confined to bed, without much change in her subjective symptoms.

Past History.—She had an attack of pneumonia twelve years ago, which lasted eight weeks. She has had winter cough for several years, with some shortness of breath on slight exertion, especially marked during past year.

Menstruation was regular up to four years ago, when menopause occurred. One child was stillborn at term. No miscarriages. No children living. No history of syphilis obtainable.

Habits.—Takes two cups of coffee daily. Does not use alcohol.

On physical examination, we found a well-nourished woman, who did not appear acutely ill. She had moderate dyspnea, and was very slightly cyanotic. Her cheeks were flushed. She had no herpes and no jaundice.

Eyes.—The pupils were equal, and reacted normally.

Tongue was clean and moist. Throat clear. The mucous membranes were normal. Teeth were in good condition.

Heart.—The apex was palpable in the fifth space, 14 cm. to left of the midline. The right border was beneath sternum. The first sound at the apex was rough and impure; second aortic louder than second pulmonic. No murmurs were heard.

Pulse.—Regular, and of good force. There was a slight increase in tension, and the vessel wall was palpable.

Lungs.—Many fine, crepitant and moist râles were heard all over chest,

front and back. Posteriorly, on the right side, between the scapula and vertebral column, there was a small area of slight dulness with bronchial breathing, voice and whisper. There were many fine, moist râles.

Abdomen.—Slightly distended; no rigidity, masses, or tenderness made out. Liver and spleen did not percuss large. The edges were not felt.

Extremities.—There was moderate edema of legs.

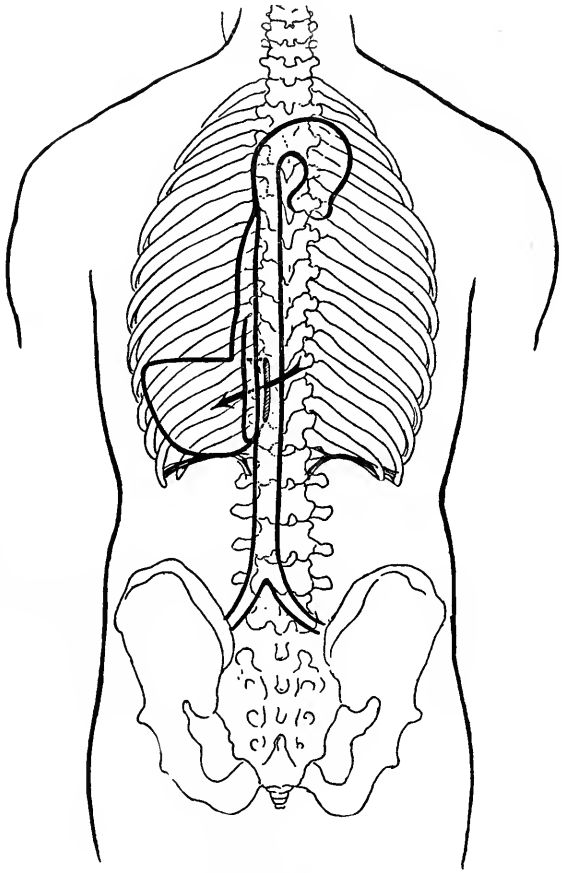

Fig. 1.—Diagram showing position of the aneurisms as seen from behind. The sac of the false aneurism lies in front of the blind branch of the dissecting sac.

On admission, her temperature was 101.3°; pulse 86; respiration 26. White blood cells 21,000; polymorphonuclears 76 per cent; lymphocytes 23 per cent; eosinophiles 1 per cent; hemoglobin 85 per cent.

Urine was acid, specific gravity 1.020. Very faint trace of albumin, no sugar, no indican, and no casts found.

For the next six days her temperature slowly dropped from 102.3° to 99.4°. Her pulse ranged from 96 to 100, and her respirations from 24 to 30. During this time the patient complained several times of "pain as food entered the stomach," although she took fluids very readily and in large quantities. On April 1, seven days after admission, the physical signs had perceptibly changed. The right upper lobe had almost entirely resolved; the right lower and left upper were clear, while below the angle of the left scapula there was an area of dulness with bronchial expiration and voice, and fine, subcrepitant râles over entire lower lobe, indicating a beginning consolidation of this lung. The leucocytes had risen to 25,000, the polymorphonuclears were 80 per cent. Her temperature was 102°; pulse 96, and respiration 24.

During the next five days patient complained of lumbar pain, which was relieved by either the hot-water bottle or codeine in moderate doses, and was thought to be due to the pleurisy. The physical signs of consolidation became more evident. The leucocytes ranged from 20,000 to 15,000, the polymorphonuclears from 80 to 71 per cent; temperature from 100° to 101°, pulse from 86 to 98, and respiration from 24 to 28. On April 7, seven days later, the physical signs consisted of marked dulness, with diminished fremitus and distant bronchial breathing, from angle to base. Above this was an area of increased fremitus, bronchial breathing, and numerous râles. The possibility of an empyema was discussed, but as the temperature was 100.2°, the leucocytes only 11,000 with 68 per cent polynuclears, and the general condition of the patient was considered good, the chest was not explored until thirty-six hours later, when a syringe full of clear fluid was obtained near the angle of the scapula. The cytological examination showed polymorphonuclears 3 per cent, and lymphocytes 97 per cent.

From April 10 to 30, the physical signs of fluid remained the same. The chest was explored at frequent intervals with the same result; namely, a syringe full of clear fluid was obtained, but on substituting a cannula and suction for the needle only a few cubic centimeters more were obtained. The opinion prevailed that there were multiple small cysts, which were aspirated dry at each exploration, surrounded by an immensely thickened pleura.

Three days before her death, the patient began to regurgitate solids,

but managed to retain fluids. This regurgitation remained unaltered. The idea of an interlobar empyema still prevailing, the chest was again explored with the same result: 2 c.c. of clear fluid.

Sixteen hours before death she complained of severe pain in the left side, which was unrelieved by codeine or heat. During the early evening she was restless and suffering from pain, which was relieved by codeine. The patient then slept for about six hours, awakening with severe pain between the ribs on the left side. This was not in-

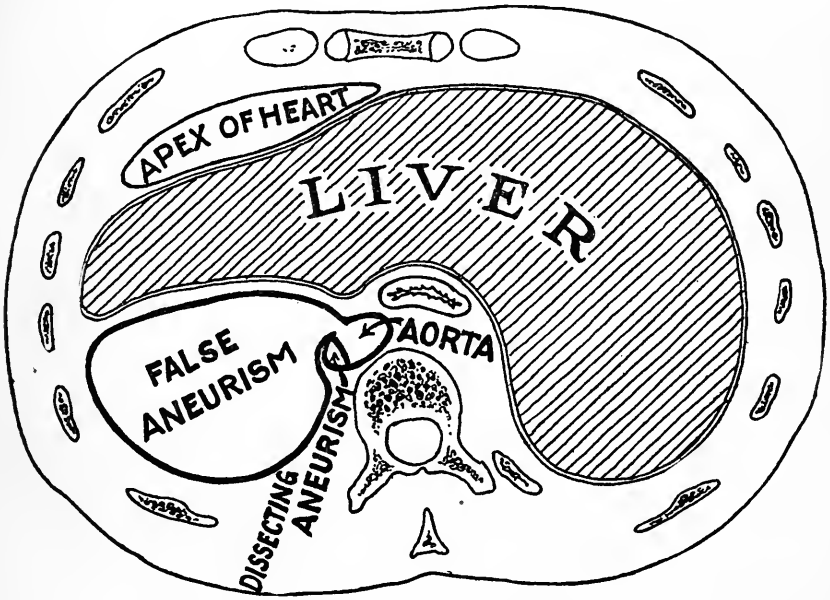

Fig. 2.—Diagram showing the position of the aneurisms as seen in cross section through the trunk. The diaphragm is represented by the double line surrounding the liver.

fluenced by codeine. Thirty minutes later her pulse suddenly became weak and irregular, and her skin cold and clammy. There was no dyspnea, and no air-hunger, patient being conscious until the end. On a hurried examination, breath sounds could not be heard over left lower chest. Patient failed to respond to the usual hypodermic stimulation, and died forty-five minutes after onset of pain.

The autopsy was performed the day after death. On opening the thorax, the anterior portion of the left pleural cavity was found to be filled with blood clot which extended about the root of the lung and

up over the apex, amounting to 600 c.c. in volume. From about the mid-axillary line backward the lung was firmly bound down to the chest wall with a mesh of fibrin 2 cm. or more thick, which was saturated with a turbid yellow fluid. No origin of the hemorrhage could be found about the upper portion of the pleura, where the greater part of the blood was collected. In the left upper quadrant of the abdomen a large mass without definite boundaries was felt behind the peritoneum and above the left kidney. On section, this mass was found to lie behind the posterior portion of the diaphragm, and to consist of a large false aneurism lying between the diaphragm and the diaphragmatic pleura. The aneurism had ruptured upward into the pleural cavity, and on account of the dense adhesions the blood had been forced upward and forward around the root of the lung. The aneurism opened laterally from the aorta by a huge gap measuring 5 cm. vertically by 1 cm. antero-posteriorly. The remnants of the arterial wall could be traced out for a distance of 3 or 4 cm. into the wall of the aneurism. Beyond this point the sac consisted of dense connective tissue, and for the most part was filled with firm thrombus. It extended laterally 11 cm., practically to the lateral chest wall, and measured 11 cm. vertically by 5 cm. antero-posteriorly.

The aorta also presented two other aneurisms. One of these was a fusiform dilatation of the ascending aorta, occupying the region of the fourth sinus. It was 5.5 cm. in diameter.

The lesion of greatest pathological interest was a healed dissecting aneurism which involved the greater part of the descending thoracic aorta. Eleven cm. below the origin of the left subclavian there was a small opening in the left wall of the aorta through which a probe could be passed into an elongated sac in the wall of the vessel. This measured 2 cm. in average diameter, and extended about 2 cm. above the opening into the main vessel. A short distance (3 cm.) below this it branched, one branch communicating by a small opening with the neck of the ruptured aneurism described above, the other lying posterior to the neck of this aneurism and ending blindly a little below the level of the celiac axis. The total length of the sac was 13 cm. The lower portion of the blind sac was filled with a firm thrombus. The upper part of the lumen was free. Some portions of the wall were smooth and glistening, other portions showed atheromatous and calcareous plaques. The caliber of the tube was irregular and the wall showed nodules where it attained a thickness of 14 mm. It did not block the orifices of the intercostal vessels, all of which opened

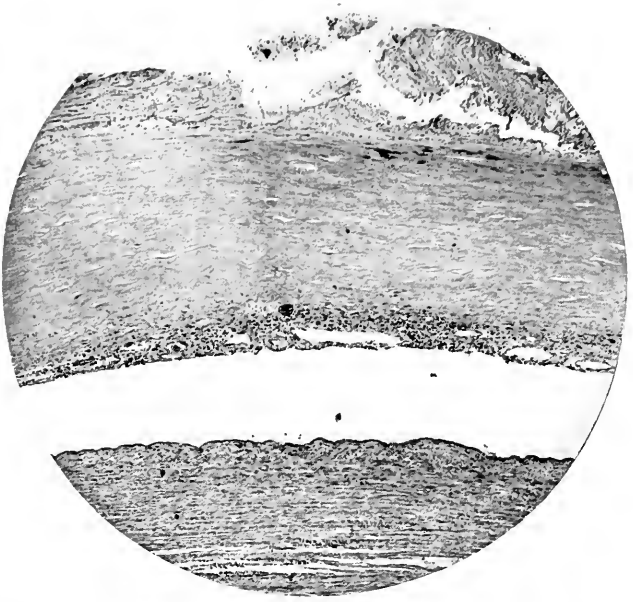

Fig. 3.—Photomicrograph showing cleft in the media of abdominal aorta. Above is seen an atheromatous area in the intima and the lumen of the aorta. 50 diameters.

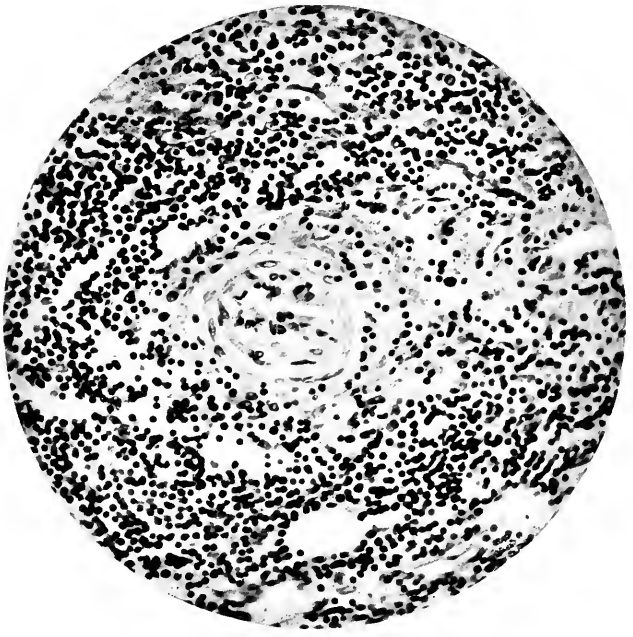

Fig. 4.—Photomicrograph showing almost complete obliteration of one of the vaso vasorum of the aorta. The vessel is surrounded by plasma cells and lymphocytes. 260 diameters.

posterior to it. The aorta itself showed most extreme endarteritis, especially in the lower thoracic and abdominal portions. The changes were much more marked than is usual in cases of aneurism. There were many large calcareous plaques with sharp edges, between which the surface was deeply ulcerated. The orifice of the celiac axis, which lay a little below the main aneurism, was restricted to a diameter of 2.5 mm., while the superior mesenteric just below this was dilated, measuring 7 mm. across. The right renal artery was very large, while the left renal was much contracted, the suprarenal on this side being unusually large, apparently in compensation. These vessels, beyond their origin, showed little evidence of disease, and the atheroma was confined chiefly to the aorta.

The other organs showed little of note. The heart was hypertrophied and the myocardium showed very slight evidence of fibrosis. The aortic ring was thick and calcareous, but not appreciably dilated. The valve leaflets were all slightly thickened, but appeared competent. The coronary trunks were free. The lymph nodes about the aorta were enlarged to a diameter of one to two centimeters. They were soft and homogeneous on section. Many of them were closely attached to the adventitia. The mesenteric nodes were also enlarged to a less degree. The lungs showed edema and emphysema. There were evidences of chronic passive congestion in the liver, spleen and kidneys.

Section through the dissecting aneurism showed that it lay in the media of the aorta between the internal and external elastic lamellæ. The elastic fibers of both these lamellæ were fragmented and partly replaced by hyaline connective tissue. The adventitia was very thick. The walls of the vaso vasorum were thickened by fibrous tissue, and the endothelial cells were swollen. Some of the veins were entirely occluded by masses of pus cells. The vessels were surrounded by collections of plasma cells and lymphocytes. Large collections of these round cells were found elsewhere in the adventitia, but none of the foci contained giant cells or showed central necrosis. Spirochætæ could not be demonstrated by Levaditi's method. The picture was extremely suggestive of syphilis, but did not warrant an absolute diagnosis. The intima of the aorta was greatly thickened by masses of tissue staining faintly with eosin and containing few stainable nuclei. There were areas of calcification. The inner wall of the dissecting aneurism showed similar degenerative changes. The lumen was lined in part with a layer of flat cells resembling endothelium.

Similar lesions were found in the abdominal aorta. On cutting

this vessel after fixation splits were seen in the wall, apparently not artifacts. In section these splits were seen to be in the media between the two elastic lamellæ. The clefts were traversed diagonally by strands of unruptured fibers. There were smaller clefts in the intima.

Changes of this type were first referred to by Rokitansky, with some reserve, as the possible cause of dissecting aneurisms. Recently Babes and Mironescu described a very similar condition, which they termed "dissecting aortitis," in two cases of dissecting aneurism. It seems highly probable that the degeneration of the media in this case, which led to the formation of large splits in the wall, either *intra vitam* or, under very slight stress, *post mortem*, explains the formation of a dissecting aneurism. The perforation of an atheromatous ulcer in the intima would expose this weakened portion of the wall and the blood would then force its way along this zone of the media with very little resistance.

A healed dissecting aneurism is a rare lesion. In 1896, Adami was able to collect thirty clearly described cases and five other probable cases of this lesion. In the literature since that time we have found six cases. There are doubtless many others which have not been reported. Partial rupture of the aorta with the formation of a dissecting aneurism is relatively not uncommon, especially in medico-legal work, but the lesion usually terminates fatally within two or three days. The most common point of rupture is in the neighborhood of the aortic ring, and the sac may extend well down into the iliac vessels. It may end blindly or may establish a secondary communication with the aorta. If the patient survives the first shock, a blood flow may be established through the aneurism. It may carry a fair proportion of the blood stream and some of the large branches of the aorta may originate from it. These healed aneurisms are usually lined with a fairly well developed intima.

Their incidence is somewhat late in life, most cases being between forty-five and fifty-five years of age, and they occur in females about as often as in men. Three factors are concerned in their production, probably in varying degree in different cases: trauma, arterial disease, and hypertension. No one of these factors is constantly present. Cases of rupture have been reported in arteries apparently normal in very powerful individuals during exertion. On the other hand, cases where the heart is small and shows brown atrophy, and where the blood pressure could not have been excessive, may rupture if the arterial disease is marked. Some cases have developed apparently

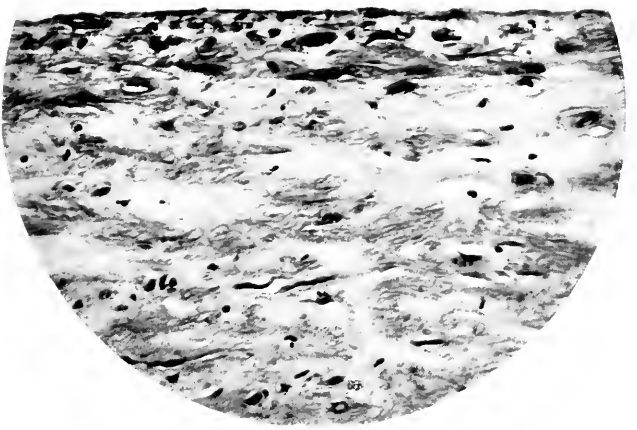

Fig. 5.—Photomicrograph showing endothelium lining the dissecting aneurism. 260 diameters.

while the patient was in bed being treated for some other ailment, so that trauma and exertion are not essential features. The arterial changes are not usually so extreme as in this case. Degeneration of the media is probably the essential feature. It is possible that the "dissecting aortitis" mentioned above may be found to be the underlying cause in most cases.

References.—Adami, Montreal Medical Journal, 1896, xxiv, 945; and xxv, 23. Babes and Mironescu, Beitrage f. path. Anat. (Ziegler), 1910, xlviii, 221. Rokitansky, Lehrb. d. path. Anat., 1855, 3d edition.

REPORT OF A CASE OF CHRONIC ULCERATIVE COLITIS, WITH SIGNS AND SYMPTOMS OF ADDISON'S DISEASE.

EDWARD N. PACKARD, M.D.

Service of AUSTIN W. HOLLIS, M.D.

A. M.—Housemaid, German, aged 51, widow. Admitted August 9, 1911. Died October 25, 1911.

History on Admission.—Chief Complaint: Vomiting, pains in legs, cramps all over body, and loss of strength.

Family History.—Father died, aged 56, of rheumatism; mother, at 57, during menopause. Three brothers and one sister all living and well. Husband was killed in an accident. No tuberculosis in family.

Past History.—Has had no children, no miscarriages. Has always been healthy except for colds, etc. Diseases of childhood not remembered. Menses irregular for 18 months.

Personal Habits.—Drinks about a cup of tea with a meal. No beer or whiskey; always worked fairly hard until present illness.

Present Illness.—About 2 months ago, at time of her menses, patient was very nauseated and vomited a great deal, and this has persisted until present time. About 10 days later, patient began to have cramps in different parts of the body. Then, lately, has been losing strength, and her head and body feel as if they were too heavy for her legs. Her appetite has been very poor all summer, and she has lost a good deal of weight. Patient complains of nervousness, which has been growing more marked. About 3 weeks ago a rash appeared on lower part of extremities; this has gradually extended upward.

Physical Examination.—Patient is a large woman, who has evidently lost some weight, the skin hanging loosely on body. Pt. does not appear acutely ill. No dyspnœa, cyanosis, or jaundice. The skin is of fair color, except following named spots, where skin appears darker than normal: eyelids, neck, armpits, hands and wrists, nipples, navel, inguinal regions, external genitals, and membrane of vagina. There is no pigmentation of mucous membrane of throat or cheeks. On arms, chest and legs is a raised eruption composed of small papules, in places confluent, and in other places partially circinate. This eruption is of a slight reddish tinge and feels lumpy. The eruption itches, and in places the top of the papule is scratched off.

Eyes.—Pupils equal and react.

Tongue.—Clean.

Throat.—Negative.

Teeth and Gums.—In rather poor condition.

Chest.—Good development, fair expansion.

Heart.—No localized apex impulse. Sounds heard best in 5th space, 3½ inches from m. l. Left border 4 inches out. Sds. of good quality; no murmurs or accentuations heard.

Pulse.—Regular, fair size and force; vessel wall not thickened.

Lungs.—At rt. post. base, there are a few sub-crepitant râles heard on deep inspiration.

Abdomen.—Normal.

Extremities.—K. J. not obtained. Very slight edema. Some muscular weakness. No paralysis or atrophy.

After the patient's admission to the hospital, she vomited daily for a week. The test-meal showed a low total acid and no free Hcl. Her gastric symptoms gradually improved. Occasionally, throughout her sickness, she vomited and complained of gastric distress. The patient had blood in the stool almost constantly. The movements were never watery, but were of a brown fluid character containing clots of blood. She never had more than 5 stools a day. Often for days no blood was seen. She had periods of constipation. Her weight for 2 months varied but little, averaging 125 pounds, but for a few days before death, her weight fell to 110 pounds. The blood count was normal, except for 5% eosinophiles. Parasites were not demonstrable. Rectal examinations were negative, and no definite cause for probable ulceration could be found. The urine 1012, trace albumen, no sugar, few hyaline and granular casts. On admission, a trace of indican. Wassermann reaction negative. For 3 days preceding her death the temperature was 94°. The autopsy revealed a chronic ulcerative colitis of an extent not appreciated while she was under observation.

This case also presented the following interesting features: signs and symptoms of Addison's disease, an extensive eruption, and a suppurative skin lesion.

As noted in physical, there were fairly well marked areas of pigmentation. These areas gradually paled out, except in axillæ. The brown skin of hands desquamated during skin eruption described below. The skin of whole body was darker than the average normal individual's, but patient said her skin had always been of dark hue. She had lowered vaso-motor tone with blood pressure in 95 Hg. For a while she was able to sit in a chair, but later her weakness increased rapidly until her death. The gastric contents showed low total acid and no free Hcl, also absence of knee-jerks, found in cases of Addison's disease. The autopsy showed no pathological change in the suprarenals.

The eruption noted in physical gradually spread until the whole body, including the face, was involved. The eruption gave a diffuse, dusky red, slightly raised appearance. Margins indefinite. In places it was lumpy. It itched. No vesicles or crusts formed. No exudation. It gradually faded, the skin desquamating in fine particles.

At about the time the eruption was disappearing, small superficial, painful lumps appeared in axillæ. These contained pus and were opened. The suppuration became extensive and the patient was transferred to the surgical ward. The count was 17,000, P. 87.5, L. 12.5, E. 1. Later, small punched-out ulcers with irregular, overhanging edges, and bases covered with exudation, appeared in pubic region. Also numerous small pustules on eyelids, end of nose, and anterior chest. At one time, 35 abscesses were counted.

Before her death, fine crackling râles were general over both lungs. The blood count was 35,000, P. 92, which was explained at autopsy. The whole suppurative process probably secondary to the ulcerative colitis.

Autopsy Findings: Body of middle-aged woman, appears somewhat emaciated, cheeks sunken. In axillæ are several ulcers ½ cm. in diameter, with raised thickened edges and thick purulent exudate on granulating base. On tip of nose and at inner canthus of left eye are pustules covered with crusts. There are a number of healing ulcers similar to those in axillæ in pubic region, and one or two more over anterior surface of chest. Also two scars on chest of healed ulcers. There is a diffuse brownish pigmentation of the skin most marked in the axillæ.

Peritoneum.—Shows minute black flecks beneath the parietal surface and the omentum is of dull grayish color.

Pleuræ.—Following the line of several intercostal arteries are similar streaks of pigment beneath the pleura. Dome of diaphragm reaches to 3d rib on either side.

Lungs (390 gms., 450 gms.).—Voluminous and rather firm at bases. Apices punctured with old scars but show no active tuberculosis. Bronchi inflamed and contain creamy pus. Bronchial nodes enlarged and black.

On section right lung shows numerous miliary abscesses filled with creamy pus scattered throughout lower lobe. The left shows a few similar abscesses and numerous patches of gray granular consolidation. Except for these patches, the lungs are moist, and considerable fluid is readily expressed from the cut surface.

Heart (330 gms.).—Small, covered with thick layer of yellow fat. Muscle brown. Cavities filled with chicken-fat clot. Valves normal. A ring of atheromatous thickening about base of aorta, and numerous patches in the coronary trunks. Coronaries tortuous.

Spleen (90 gms.).—Normal size, soft. Malpigian bodies distinct.

Kidneys (60 gms., 75 gms.).—Very small capsule strips readily leaving smooth surface. Cortex thick, of pasty, very pale yellow color; markings not well made out; medulla normal.

Suprarenals.—Left softened by post-mortem change. Right appears quite normal.

Bladder.—Normal.

Uterus.—Cervix filled with mucus. Wall contains a few small fibroid nodules.

Adnexa.—Normal.

Liver (1,096 gms.).—Normal size. Pale and mottled, with bright yellowish areas. Gall bladder contains thin turbid bile. Wall not thickened. Small

stone impacted in mouth of cystic duct, but bile may be expressed into duodenum.

Pancreas.—Largely replaced by fatty tissue. Islands of pancreatic tissue appear normal.

Intestines.—Small bowel normal throughout. At ileo-cæcal valve is ulcer with thickened base which throws it upward into the lumen, and along entire ascending colon are similar ulcers about 2 or 4 cm. by 0.5 cm. with long axis running around the gut. They do not appear to penetrate the muscularis, which is greatly thickened so as to throw the ulcer into the lumen like a fibrous ridge. A few thickened spots with beginning ulceration at the center are found in the descending colon. The retroperitoneal nodes, near the cæcum and to right of vertebræ, are enlarged, soft, and uniform deep black on section. The nodes in the mesentery are softened, semi-fluid and brownish. The panniculus was well developed, 4-6 cm. thick, and composed of intensely yellow fat.

Anatomical Diagnosis.—Chronic ulcerative colitis, chronic parenchymatous nephritis, left broncho-pneumonia, miliary abscesses of both lungs, subacute cholecystitis, multiple ulcers of skin.

Bacteriological.—Smears and cultures from lung abscesses showed Gram-positive staphylococci in pure culture.

PNEUMOCOCCUS SEPTICEMIA.

A. E. Neergaard, M.D.

Service of Austin W. Hollis, M.D.

Miss M. D., domestic, age 21. Patient in Minturn III, from November 6, 1911, to November 7, 1911. Diagnosis—Pneumococcus septicemia; congenital pulmonary stenosis. Result—Died.

The patient was admitted at night, sent in with a diagnosis of typhoid fever. She died a few hours later, before complete examination had been made and before the clinical data could be collected. Hence the incompleteness of the following records.

History on Admission.—The only facts obtained from the patient were, that she had been suffering for 8 days with headache and backache, with a fever varying from 101°-103°. She had coughed considerably, at times raising blood.

Physical examination showed a fairly well-developed and well-nourished young woman, acutely ill. Her respirations were rapid and she was markedly cyanotic, but did not suffer from orthopnea.

Her pupils were equal and reacted normally. She had internal strabismus. The tongue was coated. No cervical rigidity. Her chest was well developed, with good expansion.

Heart.—Apex impulse in fifth space, about 5 inches out. No thrills. The sounds were embryonic in character, the heart action irregular. Almost masking the heart sounds, and heard all over the precordium, transmitted to both chests anteriorly and posteriorly, was a loud, harsh systolic murmur, heard with greatest intensity in the pulmonic area. The pulse was irregular, of fair size and poor force. The lungs showed no abnormality other than a few râles at the bases posteriorly. Her abdomen and extremities were normal.

The abnormal findings at the autopsy were as follows:

Pericardium.—Contained 170 c.c. clear yellow fluid. An irregular patch of fibrin about 2 cm. in diameter was firmly adherent to the posterior surface of the right ventricle.

Heart.—Weight 555 gms. The left auricle was very small, the right much dilated, while the right ventricle was greatly hypertrophied, its wall measuring 2.3 cm. in thickness. The tricuspid valve measured 11.5 cm. Its cusps were normal. The pulmonary orifice barely admitted the tip of the little finger, and measured 2¾ cm. One cusp showed 3 small areas, each about

3 mm. in diameter, of reddish color, with rough, irregular surface, due apparently to a recent process. Otherwise, the cusps were normal. The left auricle and ventricle were both small. The mitral valve measured 10 cm., the aortic 7 cm. Their cusps were normal. The left ventricular wall measured 1½ cm. in thickness. The coronaries were normal. Foramen ovale and ductus arteriosus not patent.

Lungs.—Pleuritic adhesions and several small, hard, calcareous nodules at the right apex. At the anterior portion of the right base was a small, firm area (25 cm. in diameter), dark red, and raised above the surrounding surface with fairly sharp demarcation from the adjoining tissue. A vessel leading to this area was apparently occluded by a thrombus. In the upper posterior portion of the right lower lobe and in the anterior portion of the left lower lobe were similar areas, but no thrombosed vessels found.

Liver.—Weight 960 gms. Surface very irregular. Capsule thick. Liver substance very firm on section, generally yellowish, with small red dots, broken up by heavy bands of connective tissue.

Spleen.—Weight 240 gms. Fairly firm, deep red, trabeculæ prominent.

Kidneys.—Weight, right 157 gms., left 180 gms. Capsule stripped with considerable difficulty, tearing away a portion of the tissue. Cut surface, opaque white with red markings.

Uterus.—11.5 x 5.5 x 3 cm. Cavity contained small amount of blood clot.

Findings in other organs insignificant.

Anatomical Diagnosis.—Congenital pulmonary stenosis; acute endocarditis of the pulmonary valve; cardiac hypertrophy and dilatation; hydropericardium; infarction of the lungs; healed pulmonary tuberculosis; chronic primal congestion of liver and spleen; chronic diffuse nephritis.

Bacteriological Diagnosis.—Smears from the pulmonary valves showed Gram + diplococci; smears from the uterus showed Gram + diplococci and Gram + bacilli; culture from the spleen was negative.

Children's Service

CHILDREN'S SERVICE FOR 1911

DISEASES DUE TO MICRO-ORGANISMS	Sex		Results				Totals
INFECTIVE DISEASES	Male	Female	Cured	Improved	Unimproved	Died	
Cerebrospinal meningitis	1	....	....	....	....	1	1
Diphtheria	1	4	4	....	....	1	5
Influenza	1	2	1	2	....	....	3
Gonococcus vaginitis, malnutrition	....	1	....	....	1	....	1
Malaria, anemia	....	1	1	....	....	....	1
Neisser vaginitis, malnutrition	....	1	....	1	....	....	1
Parotitis	....	2	2	....	....	....	2
Pertussis	2	2	....	2	2	....	4
Rheumatism (ac. articular)	1	....	1	....	....	....	1
Scarlet Fever	2	....	2	....	....	....	2
Syphilis (congenital)	....	1	....	1	....	....	1
Syphilis (congen.), mucous patches around anus, rickets	....	1	....	1	....	....	1
Syphilis (congen.), secondary, circinate syphilide of face	1	....	....	....	....	1	1
Tuberculosis of lungs, indigestion, otitis media, furunculosis	1	....	....	....	....	1	1
Tbc. meningitis	2	8	....	....	2	8	10
Tbc. meningitis, general miliary tbc., ruptured appendix, peritonitis	....	1	....	....	....	1	1
Tuberculosis, pulmonary, choroiditis	....	1	....	1	....	....	1
Typhoid fever	3	4	7	....	....	....	7
Typhoid fever, bronchitis	....	1	1	....	....	....	1
	15	30	19	8	5	13	45
ALIMENTARY SYSTEM							
INTESTINES							
Colitis	2	....	....	1	....	1	2
Constipation	....	1	1	....	....	....	1
Constipation, malnutrition	1	....	1	....	....	....	1
Diarrhœa	....	1	1	....	....	....	1
Gastro-enteritis	12	13	17	6	1	1	25
Gastro-enteritis, cervical adenitis	....	1	1	....	....	....	1
Gastro-enteritis, colitis	2	1	....	....	....	3	3
Gastro-enteritis, eczema	1	....	1	....	....	....	1
Gastro-enteritis, malnutrition	5	2	2	1	....	4	7
Gastro-enteritis, otitis media	1	....	....	....	....	1	1
Gastro-enteritis, pertussis	1	....	1	....	....	....	1
Gastro-enteritis, rickets	1	....	....	1	....	....	1
Ileo-colitis	1	....	1	....	....	....	1
Herpetic stomatitis	....	1	1	....	....	....	1
	27	20	27	9	1	10	47
PHARYNX							
Hypertrophy of tonsils, adenoids	....	1	1	....	....	....	1
Retropharyngeal abscess, acute rhinitis, acute pharyngitis	1	....	....	....	....	1	1
Tonsillitis, follicular	1	1	2	....	....	....	2
Tonsillitis, follicular, nephritis	....	1	1	....	....	....	1
	2	3	4	....	....	1	5

ALIMENTARY SYSTEM—Continued	Male	Fem.	C.	Imp.	Un.	Died	Tot.
STOMACH							
Gastritis	1	....	1	....	....	....	1
Gastritis, malnutrition	2	....	2	....	....	....	2
Gastric tetany, convulsions, rickets	....	1	....	....	....	1	1
Indigestion	3	2	5	....	....	....	5
Indigestion, rickets	1	....	1	....	....	....	1
	7	3	9	....	....	1	10
CARDIO-VASCULAR SYSTEM							
BLOOD							
Anemia, following on hemorrhage	1	....	1	....	....	....	1
	1	....	1	....	....	....	1
HEART							
Mitral insufficiency and stenosis, ascites	2	1	....	3	....	....	3
Mitral insufficiency and stenosis, tricuspid insufficiency	1	....	....	....	....	1	1
Pericarditis, mitral insufficiency, ascites	1	....	....	....	....	1	1
Pericarditis, mitral insufficiency, chorea, fibrinous pleurisy	....	1	....	....	....	1	1
Rheumatic endocarditis, aortic and mitral insufficiency	1	....	....	1	....	....	1
Valvular disease, mitral stenosis	....	2	....	2	....	....	2
	5	4	....	6	....	3	9
LYMPH GLANDS							
Lymphadenitis, sub-maxillary	....	1	....	....	1	....	1
	....	1	....	....	1	....	1
NERVOUS SYSTEM							
BRAIN							
Cerebral maldevelopment	1	....	....	....	....	1	1
	1	....	....	....	....	1	1
DISEASES OF THE MIND							
Mental deficiency	1	....	....	....	1	....	1
Mental deficiency, spastic diplegia	1	....	....	....	1	....	1
	2	....	....	....	2	....	2
NERVOUS DISEASES OF UNKNOWN ORIGIN							
Chorea	4	3	4	3	....	....	7
Chorea, herpes zoster	....	1	....	1	....	....	1
	4	4	4	4	....	....	8
SPINAL CORD							
Pollomyelitis (ac. anterior)	1	....	1	....	....	....	1
	1	....	1	....	....	....	1
OSSEOUS SYSTEM							
BONES							
Potts' disease	1	....	....	1	....	....	1
Chondroplasia, inanition	....	1	....	1	....	....	1
Rickets	1	1	....	2	....	....	2
Rickets, malnutrition	1	....	1	....	....	....	1
Rickets, tetany, laryngismus stridulus, gastro-enteritis	....	1	1	....	....	....	1
	3	3	2	4	....	....	6
RESPIRATORY SYSTEM							
BRONCHI							
Bronchitis	5	3	8	....	..	....	8

RESPIRATORY SYSTEM—Continued	Male	Fem.	C.	Imp.	Un.	Died	Tot.
Bronchi—Cont.							
Bronchitis, eczema	....	1	1	....	....	....	1
Bronchitis (acute), inguinal hernia, rickets	1	....	....	1	....	....	1
Laryngitis, pertussis	....	1	....	1	....	....	1
Spasm of larynx, asphyxia, tetany	1	....	....	....	....	1	1
	7	5	9	2	....	1	12
LUNGS							
Abscess of lung	....	1	....	1	....	....	1
Atelectasis, prematurity	1	....	....	....	....	1	1
Fibrosis of lungs	1	....	....	1	....	....	1
Pneumonia (broncho-)	4	2	2	....	1	3	6
Pneumonia (broncho-), ac. colitis	....	1	....	....	....	1	1
Pneumonia (broncho-), inguinal hernia	1	....	....	1	....	....	1
Pneumonia (broncho-), meningitis	1	....	....	....	....	1	1
Pneumonia (broncho), otitis media, conjunctivitis, eczema, inflammation of Meibonian gland	1	....	1	....	....	....	1
Pneumonia (broncho-), pericarditis	1	....	....	....	....	1	1
Pneumonia (broncho-), pertussis	1	....	....	....	....	1	1
Pneumonia (broncho-), pertussis, otitis media	1	....	....	....	....	1	1
Pneumonia (lobar), diphtheria	1	....	....	....	1	....	1
	13	4	3	3	2	9	17
PLEURÆ							
Pleurisy with effusion	1	....	1	....	....	....	1
Pleurisy, suppurative	2	1	....	3	....	....	3
	3	1	1	3	....	....	4
ORGANS OF SENSE							
ORGAN OF HEARING							
Otitis media, ac. mastoiditis, septic meningitis	1	....	....	....	1	....	1
Otitis media, malnutrition	....	2	1	1	....	....	2
Otitis media, malnutrition, broncho-pneumonia	1	....	....	....	....	1	1
Otitis media, nephritis	1	....	1	....	....	....	1
Otitis media, scurvy, pertussis	1	....	....	1	....	....	1
	4	2	2	2	1	1	6
TEGUMENTARY SYSTEM							
Chronic ulcer of neck, malnutrition	1	....	....	....	1	....	1
Eczema	1	1	....	2	....	....	2
Furunculosis	1	....	1	....	....	....	1
Pertussis	1	....	1	....	....	....	1
Scurvy	1	1	2	....	....	....	2
	5	2	4	2	1	....	7
URINARY SYSTEM							
KIDNEY							
Nephritis (exudative)	1	....	1	....	....	....	1
Pyelitis	....	1	1	....	....	....	1
Pyelitis, chronic constipation	....	1	....	1	....	....	1
	1	2	2	1	....	....	3
CONGENITAL MALFORMATIONS							
Congenital hare lip	1	....	....	1	....	....	1
Cong. hydrocephalus	1	....	....	1	....	....	1
Cong. malformation of heart	....	1	....	....	1	....	1
Cong. microcephalus, gastro-enteritis	1	....	....	1	....	....	1
	3	1	....	3	1	....	4
DEFORMITIES							
Flat foot	1	....	....	....	1	....	1
	1	....	....	....	1	....	1

	Male	Fem.	C.	Imp.	Un.	Died	Tot.
INTOXICATIONS AND POISONS							
Intestinal toxemia	1	....	1	....	....	....	1
	1	....	1	....	....	....	1
INJURIES							
Foreign body in œsophagus	1	1	....	....	2	....	2
	1	1	....	....	2	....	2
MISCELLANEOUS CONDITIONS							
Boarder	4	2	4	2	....	....	6
Feeding case	1	1	1	1	....	....	2
Inanition	....	1	....	....	....	1	1
Prematurity	....	1	....	....	....	1	1
Malnutrition	4	4	8	....	....	....	8
Marasmus, prematurity	1	....	....	....	....	1	1
No pathological condition	1	....	1	....	....	....	1
	11	9	14	3	....	3	20

Orthopedic Service

ORTHOPEDIC SERVICE

DISEASES OF THE SPINAL CORD	Cured	Improved	Unimproved	Died	Total
Paralysis (ant. tibial)	....	1	....	....	1
Poliomyelitis (anterior)	....	2	....	....	2
Poliomyelitis (ant.), paralysis	....	1	1	....	2
	....	4	1	....	5
DISEASES OF THE BONES					
Fracture of femur (malunion)	....	1	....	....	1
Fracture of hip (ununited), nephritis	....	1	....	....	1
Fracture of tibia	....	1	....	....	1
Rickets, bow-legs, knock-knees	1	....	....	....	1
Potts' disease	....	1	2	....	3
	1	4	2	....	7
DISEASES OF THE JOINTS					
Osteo-arthritis of ankles, knees, elbows, wrists and fingers	....	1	....	....	1
Pneumococcus arthritis of hip	....	1	....	....	1
Pneumococcus epiphysitis	....	1	....	....	1
Septic arthritis of hip and knee	....	....	1	....	1
	....	3	1	....	4
CONGENITAL MALFORMATIONS					
Dislocation of hip	....	2	1	....	3
Malformation of femur	....	....	1	....	1
Spondylolisthesis	....	....	1	1	2
	....	2	3	1	6
DEFORMITIES					
Genu varum	4	....	....	....	4
Genu valgum	1	....	....	....	1
Hallux valgus	....	1	1	....	2
Muscle-bound feet	1	....	....	....	1
Pes planus	....	1	....	....	1
Stiffness of elbow joint following fracture	....	1	....	....	1
Talipes equino varus	1	1	....	....	2
Talipes equino valgus	1	....	....	....	1
Torticollis	1	....	....	....	1
Ingrowing toe-nail	....	1	....	....	1
	9	5	1	....	15
DISEASES DUE TO MICRO-ORGANISMS					
Tuberculous arthritis of hip	....	2	2	....	4
Tbc. arthritis of knee	....	1	....	....	1
Tbc. osteitis of hip	....	6	....	1	7
Tbc. osteitis of hip, pulmonary tbc	....	1	....	....	1
Tbc. osteitis of knee	1	1	....	....	2
Tbc. osteitis of spine	....	2	....	....	2
Tbc. osteitis of spine, hips and both knees	....	1	....	....	1
	1	14	2	1	18

AN OPERATION FOR SECURING MOTION IN ANKYLOSIS OF THE ELBOW DESIGNED TO PREVENT THE SUBSEQUENT OCCURRENCE OF FLAIL JOINT.

T. Halsted Myers, M.D.

All the older surgeons insisted upon the removal of large amounts of bone, both from the humerus and the ulna and radius if the object was to secure a movable joint after resection of the elbow. One and a half inches was about the distance that should separate the ends of the bones. Since the introduction of the method of interposing between the bones, a flap of fascia and fat, or muscle, or animal membrane, it has not been considered necessary to remove so much bone. For instance:

Goldthwaite, Painter and Osgood, writing in 1909, advise as follows, page 248: Open the joint by the posterior incision. Preserve the attachment of the triceps to the fascial expansion over the upper part of the ulna. Subperiosteal exposure of the condyles of humerus and the olecranon. Condylar surfaces removed by saw, elbow flexed and ends of radius and ulna pushed up into the wound, where they can be reached easily. It is desirable to turn in a flap of fascia or fat, obtained from the neighboring tissues. Arm put up in internal angular splints for two or three weeks. Then gentle passive and active motion permitted. In two months a fairly good functional result may be expected. In some cases there will be too much lateral motion at the false joint, rendering the articulation more or less unstable. To control this a jointed leather splint, permitting flexion and extension, but holding the ends of the humerus and ulna together so that they cannot slip past each other laterally, has been employed.

Kocher, Operative Surgery, 1911, p. 317, pays considerable attention to the conservation of the lateral ligaments. The external lateral ligament, with the attachment of the extensor tendons, and the capsule attached to the external condyle are separated subperiosteally. If complete resection is to be performed, after dislocating the joint, the internal lateral ligament is separated subperiosteally, along with the muscles, from the inner border of the ulna and the internal condyle of the humerus, and the ends of the bones are removed. In separating the lateral ligaments it is better to remove a shell of bone along with them, so as to preserve their attachment to the periosteum. The best results are obtained by interposing the supinator longus.

Binnie, Operative Surgery, 1912, p. 996, prefers operating as follows: With osteotome, separate olecranon from humerus. Remove most of olecranon. Divide bony tissue uniting humerus to ulna and radius. Completely divide

lateral ligaments. Flex elbow acutely. With Gigli saw remove small portion of lower end of humerus. Remove articular surface of ulna, and model a new sigmoid cavity. If necessary, remove part of head of radius. Divide any bony tissue uniting radius to ulna, if possible preserving annular ligament. Smooth and shape opposing surfaces of radius and ulna. Interpose flap of fat, fascia and muscle. Trim edges of humerus. Cover its lower end and one inch of both anterior and posterior surfaces with flap of fat, fascia and muscle (anconeus, extens. carp. Ulnaris, etc.). Stitch this in place. Close wound with drainage.

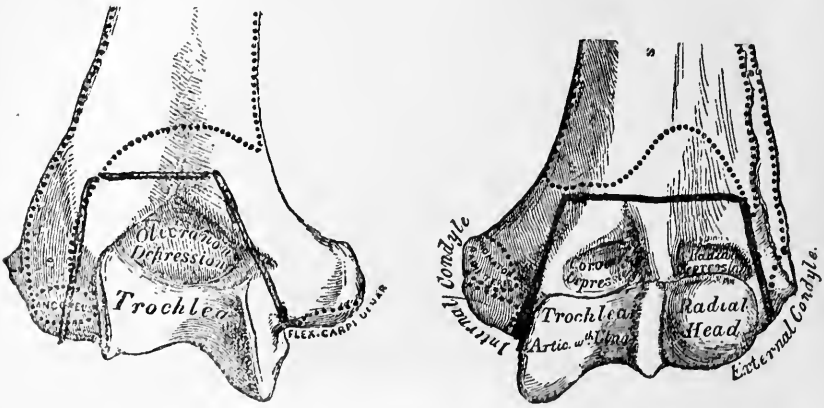

Fig. 1.—Tubby, Deformities, including Diseases of Bones and Joints, 1912, removes rather more bone, including the epicondyle and epithrochlea, the olecranon and its articular surfaces, and part of the head of the radius, making a gap of 1½ inches at least between ends in an adult. He interposes a strip of the anconeus between humerus and radius and ulna, and winds a strip of the extens. carpi rad. long. about radius, between it and the ulna.

As to the best material to interpose between the freshened ends of the bones: While foreign bodies, such as plates of magnesium, ivory, etc., seem to have been generally discarded, many surgeons are using flaps of fat fascia and muscle from the neighboring parts.

Aponeurotic flaps are too feebly nourished with blood to undergo transformation into bursal tissue, which is considered desirable (Huguier, Paris, 1905). Baer, writing in the American Journal Orthopedic Surgery, August, 1909, says:

"In a majority of cases the interposition of living tissues is followed by a constant pain, due to pressure upon its nerve endings. While we may attain a certain degree of motion by the interposition of muscle or fascia, the motion is generally unnatural in character, and quite often results in an unstable joint. The membrane which I use is from the pig's bladder, and is chromicized, so as to remain intact about forty days. This is thin and pliable enough to allow of

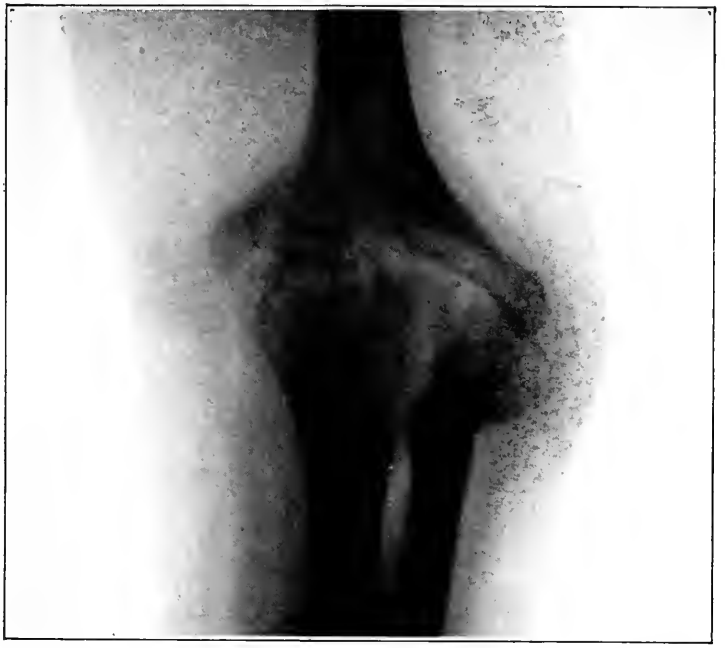

Fig. 2.—Skiagraph of right elbow taken a year after operation.

Fig. 3.—Skiagraphs of left elbow taken a year after operation.

easy adjustment within the joint, and will remain there beyond the period of bone or fibrous formation."

Transplantation of living cartilage (Weglowski, Centralblatt fur Chir. 1907, No. 17) in the treatment of ankylosis, and transplantation of an entire living joint (Buchmann, Centralblatt fur Chir., 1908, No. 19) have not been done sufficiently often to enable one to estimate their value.

The operation I wish to describe is in line with the tendency to sacrifice as little bone as possible, by the interposition of animal membrane, and to especially preserve the leverage of the muscles about the elbow, and prevent too great relaxation of the joint.

In the early part of 1910 I saw a girl, fifteen years of age, most of whose joints were partially or completely ankylosed by an infectious osteoarthritis, which had attacked her ten years previously. The deformities resulting had been corrected, and the joints manipulated, several times, under anæsthesia, by different men, after the active stage of the disease had subsided; but the ankyloses gradually recurred. Nothing had been done for the past five years.

As there were no signs of active disease, and both elbows were ankylosed at 140°, but pronation and supination were fairly good, and as she had a little motion in her fingers, and about half the normal amount in her shoulders, I decided to try to mobilize her elbows, as that would enable her to feed and dress herself.

May 21, 1910. Ether. After Esmarch bandage had been applied, the right elbow was exposed by a vertical incision down to the bone, along the outer edge of triceps tendon and olecranon process. The triceps tendon was freed from the process for about an inch, but its periosteal attachment not divided. All the soft tissues were then retracted en masse, with periosteum, to the outer and inner sides of the joint, but only as far as the condyles. The ulna nerve was displaced inward to the edge of the condyle. This dissection gave free access to the posterior part of the joint, and a truncated wedge-shaped section of the lower middle part of the humerus was removed easily with chisel. The joint was then forced to a right angle position, and the rest of the bone attached to head of radius and to ulna was removed with rongeurs, and all surfaces made smooth. Cargile membrane was then placed in front, under and behind the edges of the humerus. The ulna nerve was replaced, and the wound closed without drainage. A plaster splint was applied from fingers to neck, the elbow being held at 80°, in mid position between pronation and supination.

Eighteen days later, first dressing. Primary union. Elbow passively flexed, without pain, to 40°, and extended to 135°. Elbow then fixed at 40°. Ulna anæsthesia noted at first is less marked. Two weeks later a sling was substituted for cast, and child encouraged to use the arm.

In September the anæsthesia had disappeared entirely, and the elbow had a range of motion from 150° to 30°, and about 15° of both pronation and supination.

October 3, 1910, the same operation was done on the left elbow, with the exception that the incision was carried down the inner side of the olecranon, in order to make the approach to the ulna nerve more direct. This nerve was not displaced at all, but was subjected to rather severe pressure by the retractors, which may account for the anæsthesia in this case The joint was found in about the same condition as the right had been, and was treated in the same way, but instead of Cargile membrane, Johnson and Johnson's chromicised pig's bladder (Baer's membrane)) was used to cover the edges of the humerus. Wound then closed, without drainage, and elbow fixed at 80° by plaster splint extending from fingers to neck.

Nine days later cast was removed. Primary union. Cast reapplied. Oct. 20th, cast permanently removed. Extension to 120°, flexion to 60° possible, but more pain and resistance than in previous case. Ulna anæsthesia. Sling applied; massage and passive motion ordered daily. Position to be changed each day.

Dec. 6, 1910. The child has very good use of the right elbow, and can feed herself and reach all parts of her head. The left elbow can be extended to 140° and flexed to 40°, but is still somewhat tender, though each week less so. The ulna anæsthesia is still present.

March, 1912. Report received from this child states that in the left arm the range of motion is from 145° to 60°, with pronation and supination of 15° each. In the right elbow the range of motion is from 135° to 40°, with an equal amount of pronation and supination. There is still some ulna anæsthesia in left hand. The child can dress herself without assistance.

The object of this method of remodeling the joint is to remove the opposing bone surfaces a considerable distance from each other, and yet not destroy the strength of the articulation, as is generally done when the lateral ligaments are divided, and the condyles, the attachments of the pronators and flexors on the inner side, and the extensors on the outer side are removed. In the usual operation of excision, the posterior support and leverage of the olecranon is also destroyed. In this operation the ligaments on all sides of the joint are preserved.

The operation may not be suitable for tubercular cases which have become ankylosed, but seems to meet the requirements in joints ankylosed from any of the acute infections, or from the various forms of atrophic or hypertrophic osteo-arthritis, and in some cases of ankylosis after fracture about the joint with deformity and excessive callous formation.

The enclosed sketch shows the amount of bone removed; that part included by the heavy lines.

The skiagraphs taken Nov., 1910, show marked changes in the joints, and also how little can be learned from such a picture about the amount of motion possible in that joint.

Otological Division

THE RADICAL OPERATION WITH THE APPLICATION OF THE PRIMARY SKIN-GRAFT, FOR THE RELIEF OF CHRONIC MIDDLE-EAR SUPPURATION—WITH REPORT OF CASES.

EDWARD BRADFORD DENCH, M.D.

I have already written so fully, on previous occasions, upon this subject, that an article of the same character, in the St. Luke's Hospital Reports, may seem rather out of place. The fact, however, remains that in spite of the excellent results obtained in cases of chronic middle-ear suppuration by the radical operation, with the application of a primary skin-graft, many surgeons still hesitate to resort to this procedure.

I beg, therefore, to report two cases which I have operated upon in St. Luke's Hospital during the last few months, which demonstrate clearly the very excellent results which may be secured by this operation:

The first patient was a boy, aged 11, who had suffered from a chronic discharge from the right ear for 7 years. The boy was anæmic, his general condition being much below normal, although no causes other than that of the persistent aural suppuration could be found to account for the impaired general health. An examination of the right ear showed an extensive destruction of the membrana tympani, with granulation tissue present. This granulation tissue evidently had its origin in the tympanic vault. A portion of the internal wall of the middle ear was dermatized. The low whisper, upon the right side, was heard only at 4″. There was no evidence of any labyrinthine involvement. On October 24th the radical operation was performed. The mastoid cells were well developed, and extensive caries was found throughout the entire mastoid. This caries extended posteriorly to the sinus groove, and the lateral sinus was exposed during the operation.

The complete radical operation was performed, all the mastoid cells were obliterated, and the mastoid cells, middle ear and external auditory meatus were thrown into one large cavity by the taking down of the posterior meatal wall. Particular care was given to the obliteration of the hypotympanic space by lowering the level of the floor of the external auditory meatus, while the posterior tympanic space was obliterated by carefully removing

the posterior canal wall, as far backward as possible, without injury to the facial nerve. The nerve was exposed by this procedure, but not injured. The external auditory meatus was enlarged by cutting a tongue-shaped flap from the concha. Cartilage and connective tissue were removed from this flap, and the flap was then folded backward and upward and stitched to the raw area on the posterior aspect of the auricle. The operation cavity was then exsanguinated by firmly packing it with a strip of gauze saturated in a solution of adrenalin chloride, of a strength of 1-1,000. All superficial hemorrhage was controlled by ligatures. The entire cavity, formed by the exenteration of the mastoid and middle ear, was then lined with two Thiersch grafts.

Ordinarily, one graft is used to line this cavity, but it was impossible, owing to the small thigh of the patient, to obtain a single graft large enough for this purpose. The grafts were laid over the bone and made to apply themselves exactly to the irregularities of the surface by introducing a pipette beneath the grafts, and then exhausting the air. This procedure permits the graft to adapt itself to the irregularities of the bony surface. The grafts were held in position by small pledgets of sterile cotton packed into the cavity. The posterior wound was then closed completely, and a third graft was applied to the meato-conchal margin, the graft being held in position by a light packing of sterile gauze. The operation was completed by the application of a sterile dressing. The sutures in the posterior wound were taken out on the second day, and the pledgets holding the graft in position were removed about 5 days after the operation. The grafts adhered perfectly, and the ear was completely dry 3 weeks from the time of operation. Two and a half months after the operation the whispering distance on the right side was 3 feet.

The operation, in this case, was a perfect success, all discharge from the ear having ceased 3 weeks after the operation, and the hearing having been greatly improved.

The second case was that of a young man, 25 years of age. When 2 years old both ears discharged. There was no further aural trouble until 8 years before I saw the patient, when both ears again discharged. For the past 8 years there had been an intermittent discharge from each ear whenever the patient had a severe cold in the head. Two weeks before I saw the patient, the left ear began to discharge rather profusely, and there was some pain in the ear. Upon examination, a large perforation was found in the right drum membrane, with partial dermatization of the mucous membrane of the middle ear. The ear was perfectly dry. Examination of the left side revealed some purulent discharge in the left auditory canal, a large perforation involving the lower portion of the drum membrane and the internal wall of the middle ear was swollen; there was slight sinking of the upper and posterior wall of the external auditory meatus, close to the drum membrane, and a sinus leading into the tympanic vault. The low whisper was heard 23 feet upon the right side and 5 feet upon the left side. The patient had a temperature of about 100° on the afternoon of the day

upon which I first saw him. There was no labyrinthine involvement demonstrable, except that the left labyrinth was slightly hyperæsthetic to the galvanic current. While the patient had no severe pain, there was a continued feeling of discomfort in the ear, and 5 days after I first saw him, the radical operation was performed. The periosteum covering the mastoid was considerably thickened, and there was considerable caries in the mastoid cells. This caries was particularly well marked over the roof of the tympanum and mastoid, and it was necessary to expose the dura in this region before all diseased bone was removed. The dura was slightly congested. The radical cavity was formed in exactly the same manner as in the previous case, the hypotympanic space being obliterated by removal of the floor of the canal, while the posterior tympanic space was also effaced by the careful removal of the posterior canal wall, close to the facial nerve. The meatal flap was formed in the same manner as described in the first case. The entire bony cavity was covered by a single Thiersch graft, held in place by pledgets of sterile cotton. The posterior wound was closed and a meatal graft applied. In 2 weeks' time the middle ear was perfectly dry, and the low whisper was heard at a distance of 15 feet in the operated ear.

These two cases, operated upon within a period of six weeks, show the results that can be obtained in chronic middle-ear suppuration by operative interference. They are simply examples of a series of nearly 200 cases, operated upon by the writer, in the same manner. At the International Otological Congress, held at Bordeaux, in 1904, the author reported 98 cases, operated upon by this method. Since that time, I should say that an equal number of cases had been subjected to operation. With the perfection of technique, the results in later cases have naturally been better than in those cases operated upon at an earlier period, and I believe that now we can promise any patient suffering from a chronic middle-ear suppuration, not only a perfectly satisfactory result, as far as the otorrhœa is concerned, but also a satisfactory result as to the preservation of function of the organ. The only exception which I would make to this latter statement, is in those rare instances where, in spite of an aural discharge, the hearing is exceptionally good. In these cases, the hearing may become somewhat impaired as the result of the operation. In those cases, however, in which, as the result of the suppurative process, the hearing is greatly impaired, we can ordinarily promise the patient an improvement in hearing if he will submit to the operation. This fact is borne out in the two cases already reported.

In a short article of this character, it would hardly be wise for me to discuss the dangers of chronic middle-ear suppuration. It may be well, however, to repeat the statistics which I mentioned in my

paper, read at Bordeaux. These statistics were as follows: The records of the New York Eye and Ear Infirmary, for 8 consecutive years, showed that 19,323 cases of suppurative otitis media were treated in that institution. During this time there were 218 cases of severe intracranial complications. In other words, one patient out of every 88 suffering from middle-ear suppuration, suffered also from some severe intracranial complication demanding operative interference.

These statistics are, I think, sufficient to show how frequently a middle-ear suppuration causes some intracranial complication. The radical operation naturally removes all danger of subsequent intracranial involvement, and if, at the same time, we can promise the patient that the function of the organ will not be seriously impaired as the result of operative interference, we certainly are justified in recommending this procedure in all cases of intractable middle-ear suppuration.

Pathological Department

A NEW ERA IN MEDICINE IN NEW YORK.

F. C. Wood, M.D.

(Address given in Chicago, March, 1911, before the Alumni of Columbia University.)

As most of you are aware, the educational problems before the colleges of this country are many and complex. A growing appreciation of the difficulties to be met is rapidly awakening, not only in the teacher but also in the public, some doubt as to the perfection of our methods and distrust as to the ultimate results as shown in the finished product, the college graduate. The problems are not wholly financial, as many seem to think; they lie far deeper in the innate spiritual qualities of the American race. Never has a people so patiently tried to demonstrate that money will solve all problems of politics, art, or education, as our own. Never has a failure been so complete and absolute. We do not yet fully appreciate that money will buy neither loyalty, scholarship, nor genius, but only industry, no matter with how lavish a hand it be distributed. A faint glimmering of light has occasionally penetrated the darkness when some incomprehensible foreigner has refused to abandon a comfortable teaching position in his native land for twice the salary and one-tenth of the appreciation he now enjoys. Because, with the expenditure of a few millions, a model manufacturing plant can be created in a year or two, people still seem surprised that the loyalty of a teaching body to a university and that intangible thing called tradition may be more valuable than much money; that the poorest paid and least known of the professors within a college's walls may have a world-wide reputation, while the specialist purchased at a high price from a rival institution seems chiefly known to the readers of the illus-

trated editions of the Sunday newspapers, in which he publishes, in popular form, the preliminary reports of investigations, the final results of which rarely appear in print. It is not necessary to cite examples before such an audience. But the fact must not be forgotten that too often we think that a little more money would cure all academic ills, while really a thorough organization of the work of an already existing loyal and harmonious staff of teachers would accomplish quite as much.

But what of Columbia? As graduates of the varied schools of that institution, you may ask what message I bear? Have we mistaken size for greatness or bartered a good name for newspaper notoriety? I can honestly say, No. The growth of the University has been remarkable, but, in general, wholesome, and its efficiency as a teaching institution is in every way better than in the previous decade. The most interesting changes of recent years in any department have been in the Medical School, long famous as the College of Physicians and Surgeons. The educational future of the institution has so recently been assured by an unusual combination of circumstances, coupled with a wise and generous gift of funds, that I shall confine my remarks chiefly to this aspect of the University's growth.

As some of you may know, Columbia, on behalf of its Medical Department, is about to enter into an agreement with the Presbyterian Hospital, one of the largest private hospitals in New York, by which a much closer relationship is to be consummated than has hitherto existed between any of the New York schools and hospitals, an arrangement which permits the nomination by the College of the incumbents of the clinical and laboratory services of the Hospital. In return for this permission, which carries with it the use of the patients in the wards for the teaching of students, the College agrees to care for the scientific work of the hospital, the various heads of the purely laboratory departments becoming ex-officio responsible for the hospital work in their special fields. How great a change this is, and how much it means for the future of Columbia may not, at first sight, be very obvious, but I may safely say it promises a new era in American medicine. It may seem a small thing as compared with the opportunities which have been enjoyed by the English and German schools, by Johns Hopkins, and to a lesser extent by several of the Philadelphia medical colleges. And yet, it is the beginning of what may make New York City, as it should be, but is not, one of the great medical centers in this country. A short statement of the past and

present position of the Medical School may bring more clearly before you what the new arrangement means.

Up to the year 1891, the College of Physicians and Surgeons, though nominally connected with Columbia University, was really a proprietary institution, though, through the generosity of the Vanderbilt family, it had been equipped with buildings which at that time were ample for its needs. Even then, however, it was felt that the school required a closer intellectual relationship with Columbia University, then beginning that remarkable expansion which has culminated in the great educational institution of some 7,000 students now existing in New York. An agreement leading to closer union was therefore carried out, and in 1901 the College was placed practically under the absolute control of Columbia. In the meantime, much new construction had taken place, in order to bring the laboratories up to modern standards. Through the generosity of Mr. and Mrs. W. D. Sloane, the Sloane Maternity Hospital was even then a model institution for the teaching of obstetrics. It has since become, in the past year, by the erection of a new pavilion, a complete Frauenklinik, to use an expressive German term; that is, obstetrics and gynecology are united in this hospital for women. In this phase of its work the school has always had all that it could desire. The Vanderbilt Clinic also has been a model for out-patient work, with a clientele so enormous that it has been difficult even to care for the patients, some 50,000 a year, much less to study each one carefully. Yet these were the only sources of clinical material for instruction absolutely under the school control.

These changes, begun 20 years ago, seemed to place the college in a very strong position, especially as its faculty included most of the abler clinicians visiting the large hospitals. The condition of the scientific department has always been excellent, and the teachers in those subjects are well known the world over. I have only to recall the names of Prudden, Curtis, Cheesman, Hiss, Gies, Huntington, Richards, Herter and MacCallum to your minds. But despite the presence of able men in the departments of medicine and surgery, the feeling has been growing stronger in recent years that they have lacked something that the laboratories possessed, that is a full control of their teaching material. It is only too true that while the laboratory investigators of this generation are justly famous, the clinical teachers in this country, as compared with those of Germany, have contributed but little to the science of medicine. The surgeons, it is true, have

been ingenious, and have devised and perfected many operative methods now generally employed; but surgery is spectacular; it attracts endowments. Surgeons usually can obtain from hospital managers equipment costing many thousands of dollars, when the medical staff can hardly get a microscope, much less a polygraph. Surgery is so definite, so positive, and, one may say, so simple a field, that the surgeon has occupied the foreground in this country to the detriment of the physician. American surgery to-day is technically the best in the world, but medical research is still in its infancy.

The reasons for this are many: First, we do not obtain in medicine the definite results that the surgeon does. We do not so evidently save lives. The general public suspects, and quite justly so, that many of the cures in medicine are due to fresh air, good nursing, and the healing power of nature, and not so much to the drugs administered. On the other hand, it is quite a simple matter for even a mediocre operator to remove an inflamed kidney, or a diseased ovary, or a tumor of the breast, and obtain satisfactory, even brilliant results. The physician works under different conditions. No one can claim to cure chronic Bright's disease. Both kidneys are usually affected, and before a diagnosis is possible and any treatment instituted the organs have undergone serious and permanent changes. The treatment of cardiac lesions is a palliative one. We help the heart to do what it is trying to do naturally. We put the patient in bed and give the hard-worked muscle a needed rest. We regulate the diet, and, if need be, give cardiac stimulants. Nature does the rest. But we do not effect the permanent cure of many forms of heart disease. So, too, with many infectious diseases. Our powers are as yet extremely limited. I may merely mention, as examples, pneumonia and tuberculosis. Our great victories over the latter are those of fresh air, good food, and prevention of the distribution of the virus. This brings us to the second reason why medical research in this country has not prospered. For the investigation of disease in human beings, a laboratory is necessary, and this laboratory is one in which the scientifically trained physician can study patients. Much can be done by means of animal experimentation, but dog medicine will never replace human medicine. The ordinary laboratory animals do not suffer spontaneously from the diseases in which we are most interested. In fact, many of the important conditions cannot be induced in animals with any certainty. Therefore, while sufficient, and in some instances, ample facilities have been given pathologists, chem-

ists, bacteriologists, and even surgeons, the physician has long struggled with poor equipment, insufficient laboratory space, and lack of access to patients whom he can control. In other words, the medical school could offer no facilities for research in medicine, as it had no laboratories for such study; that is, no hospital. A third reason is that in general in this country there is no credit given and no financial reward offered for even the best medical research; the prizes go to the man with a large general practice.

Not a little criticism has been directed for years toward hospital managers for closing the doors of hospitals to those who desired to study disease in the wards as they are studying disease in the laboratory, and for giving appointments on the visiting staff to men who are purely practitioners of medicine, and not investigators; and many comparisons have been made, to the disadvantage of this country, with the great opportunities existing in Germany, which are open not only to the Germans, but to any volunteer who is willing to give a reasonable amount of time in the wards. It is possible for any well equipped young American physician to go to Munich, for instance, and, if he will spend six months, to enter the wards of the great Fr. Müller, and there study patients in a way which he cannot hope to do in America. Even the Johns Hopkins Hospital is more or less closed to outsiders, because of the necessity of using its material for its own students. But the young man comes back from Munich full of enthusiasm and scientific interest, and desirous of the same facilities that he has enjoyed there, only to find the doors of the hospitals closed against him. The great municipal hospitals can offer no advantages to the student of scientific medicine; they are poorly equipped, the scientific staff underpaid and overworked, and the executive staff still too largely under political domination—so the crowded ranks of the practitioners receive another recruit.

And yet there is another side to the question. The managers of a private hospital are given money to be expended in the care of patients. They are trustees of this money, and consequently cannot spend it as freely as they could if the hospital were run on purely business principles. They can try no experiment, risk no cent of their funds. In consequence, the private hospital lags behind even the municipal institution in advancing medical science, and falls far short of what is and always can be accomplished by a private institution not dependent for its future upon donations. Then, too, it is impossible to turn loose in the wards a large number of undergraduate

students. They are, in their enthusiasm, apt to over-examine and annoy a patient. It is difficult, for example, to keep an interesting case of malaria in a ward; every student and interne wishes to have a blood slide for his own collection. The hospital has to protect these people by limiting the number of students to each ward. It is difficult to convince them that they gain weight and strength by repeated punctures of their fingers. So, too, in gynecological work, it is impossible to have a large number of men examine a woman patient. In acute appendicitis the fewer people who palpate the abdomen the better for the patient. The course of a severe pneumonia is not improved by having twenty men listen to the patient's chest. So that the managers have a great deal on their side, and yet, largely due to the agitation and discussion which has been started by the alumni associations of the large New York hospitals, composed as they are of the younger, better trained physicians of the community, most of whom have also studied abroad, one after another of the great New York private hospitals has opened its wards to small numbers of selected fourth-year students. The P. and S., for instance, to-day can send fourth-year undergraduates into the wards of five of the large private hospitals, where they remain for two months, enjoying all the facilities offered to the residents, with the exception that they have no power to administer drugs. Much to the astonishment of the managers, not only has the death rate of the hospitals not increased by this introduction, but it has been found that the attending physicians are apt to give a great deal more time to their ward services than they did under the old régime. The cases are more thoroughly examined, the patients are better satisfied, the histories are more carefully taken, the house staff is relieved of unnecessary routine, and it is now the hospital which is beginning to ask for more teaching. This is as it should be, and the first result of this experiment, begun at St. Luke's Hospital, some three years ago, is the proposal of the Presbyterian Hospital managers to join with the P. and S. as offering the best results in the care of patients. To the managers, of course, scientific study is of less immediate interest, though they also are beginning to feel that the reputation that a hospital gets from the publications and scientific fame of its staff brings it glory, and in that way, larger funds. In Germany—where, as any of you who have studied there know—the patients have less to say about their treatment than they have in this country; where autopsies are universal instead of exceptional; and where the system exists of placing the

patient under the care of eminent men who have made advances in chemistry or bacteriology or pathology, instead of those having merely a large private practice—the conditions are far ahead of what they can be in this country for some years to come. We may never reach the same freedom in handling human beings that now exists in the hospitals of Germany and France. Our attitude toward our patients is quite different, our feeling of responsibility to them is much greater here than it is there. All this makes more difficult the use of patients for thorough scientific study. The semi-military discipline of a European hospital cannot be imitated in America. Patients must voluntarily offer themselves for study. We must ask a patient's permission before we can place him in a respiratory chamber; it is almost necessary to obtain his permission before he can be put upon the somewhat irksome diet which is necessary for the complete chemical investigation of his metabolic peculiarities. These are some of the perfectly obvious and practical difficulties in medical investigation in this country, and there are not a few others patent to every laboratory investigator. We cannot shut our eyes to them, and we must meet them with all possible patience, while at the same time safeguarding our patients from annoyance and injury. This means a far greater supervision by the resident and visiting physicians than exists in Germany, but if such safeguards are offered, I think we can accomplish just as good work here as there, even though at a considerable disadvantage.

The union of the Presbyterian Hospital and the P. and S., the close geographical relationship of the Rockefeller Institute, and the presence of the enormous hospital material now being offered for teaching purposes in New York City, therefore, opens up a new era to the P. and S., which, in the past few years, has been in great difficulties, both financial and clinical. The day of the old-fashioned clinical lecture, when the students sat in an amphitheater and watched the professors operate, or when the students made ward rounds and saw fifty patients without being allowed to examine one, has long since passed. Students must be taught in small numbers; no more than four or six men can be allowed to study a case. It means a great increase in the number of our teachers; it means a great increase in our clinical facilities, before we can reach the ideal. The Presbyterian Hospital, in its new buildings, will construct ample laboratory facilities for such scientific work; it will probably be the center of a large part of the undergraduate teaching of the school, and will offer

opportunities for the best type of medical and surgical research. But that is not the limit of a great hospital school such as must develop in New York, Chicago, and other large cities. For it is in the large cities that opportunities for teaching medicine exist. It is impossible to build a great medical school in a small town. A thousand hospital beds must be available for teaching purposes, if the student is to be thoroughly grounded, not only in medicine and surgery, but also in the important specialties, and such a large material is easily available if Columbia can further extend its hospital affiliations, even if the relationship is not so intimate as that with the Presbyterian. These are the conditions which we are now facing, and many problems must still be solved.

A medical school must, primarily, teach undergraduates to be good practitioners. That is what the public wants; that is what the country needs; well-rounded men who have seen a large series of cases, who are trained in all the fundamental sciences; men who have had at least two years in college, more if possible, so that the curriculum need not be crowded with elementary courses in fundamentals; men who have had real training in biology and not merely a superficial course; men who know something of mathematics, something of experimental physics, and a great deal of organic chemistry, and have a real reading knowledge of German, not only the ability to pick out a few sentences by the aid of a dictionary. Another function of a medical school is said to be to train teachers. I think this is wrong. Teachers are not made, they are born; only a small proportion of the men who study medicine is in any way fitted to teach, and to adapt a school for this special purpose is unnecessary. A still smaller proportion of those obtaining a medical education is fitted for productive research in medicine—the most complicated of all fields. Such men must have all the preliminary training that the future practitioners are given; they must also have opportunities to exert their natural gifts. In other words, the school must offer research opportunities for such undergraduates as show themselves fitted to do such research.

We are too apt to be careless in the use of this term "research." Much of the matter which is published from the foreign universities, much from our own, is not worth the paper it is written on. It is done by immature, poorly trained men, with limited horizon and perspective, and merely encumbers the field for those who come after. Real research ability is very rare. It is well to give the practitioner a chance to see what research means: that he cannot do research without

an enormous sacrifice of time, without giving up many of the rewards that come to one who has many patients. He cannot obtain much more than a living salary—in fact, as a laboratory investigator in this country it is difficult to obtain even that. Research in medicine is also the most expensive possible research, if we except astronomical investigation. It requires not only patients to study, but the facilities of large, well-equipped laboratories. The care of patients in New York City costs over two dollars a day. This expense must be met by the hospitals; it cannot be added to the already overloaded budget of the medical school.

There is also another function of the medical school, and that is the offering to men the opportunity for post-graduate work in various subjects, chiefly in the specialties, but also in the laboratory branches. Most of these men will be practitioners who desire to fit themselves for certain special branches, and this instruction must be disassociated, more or less, from undergraduate teaching. With the diminution which is now going on in the number of men who take up medicine, owing to the overcrowding of the profession, in the first place, and owing to the greatly increased cost of medical education in time and money—for it means a sacrifice of at least ten years' time to become a physician—the number of undergraduate students in the college will probably remain small. We do not desire more than one hundred to one hundred and twenty-five students in a class. We now have about eighty-five. The size of the school is not likely to be increased, therefore, in the undergraduate department, in the near future. Those who direct the future of the college are desirous of seeing extensive development of advanced work and post-graduate teaching—a great expansion of true investigation along the lines of scientific medicine. For this we shall have to have other hospitals than the Presbyterian; hospitals with ample laboratories, with broadly trained clinical teachers in charge of the wards, men who can appreciate the problems which are yet to be solved, and offer the graduate in medicine a chance to develop his special powers of clearing away the obscurities which still surround a large number of the diseases which we so frequently try to treat.

The completion of this ideal scheme will probably require a good many years of patient labor, and implies, primarily, an extensive development of the hospital connections we now enjoy. The final solution lies in the hands of the trustees of hospitals, both municipal and private, and until they realize what is so obviously needed in

medical education, and appreciate the advantages of close union with teaching institutions, it is difficult to see how any real progress can be made, but there can be no question of the final outcome. The hospitals and schools must finally come together to solve their common problems and so to obtain their highest possible development, from both an educational and a philanthropic standpoint.

SELECTING LENSES FOR PHOTO-MICROGRAPHY.

F. C. Wood, M.D.

The drawing of tissues under the microscope is a difficult matter, and but few physicians have the necessary ability or time to produce satisfactory sketches. Even professional illustrators are rarely able to reproduce such material properly without a great deal of supervision, and then only at considerable cost. On the other hand, the production of commercial half-tone plates has now in the best hands reached such a degree of perfection that there is but little loss of detail in reproducing satisfactory prints of photo-micrographs if made on a glossy surface solio or gaslight paper. These facts, together with a desire to reproduce microscopic subjects as documents giving evidence of the correctness of the text descriptions on which a thesis may be based, lie at the bottom of the revival or, if preferred, the more extensive use of photo-micrography in illustrating embryological and histological publications.

The recent commercial introduction of color-sensitive plates and suitable screens has made possible the use of three-color methods for direct reproduction of microscopic objects, if expense of reproduction does not have to be considered, in a beauty and accuracy not possible in the old days of plate making. The employment of the Lumière direct color plate for projection purposes has also revived interest in photo-micrographic methods.

During the last thirty years the elaboration of the mathematical theory of the production of images by lenses, due to the genius of E. Abbe, and the production of glass of special optical qualities by the Jena Glass Works, have also enabled opticians to make many improvements in lenses. The results have been most notable, perhaps, in the production of photographic lenses for general purposes, but very remarkable improvements have also been accomplished in the production of microscopic lenses and oculars, though chiefly of the higher powers.

On the whole, however, these discoveries have not greatly improved the objectives of low or medium magnifying power, from a purely photo-micrographic point of view. Even in the most admirable apochromatic objectives the curvature of the field of vision is often very considerable; so much so that the remarkable 8 and 16 mm. objectives of Zeiss are not especially satisfactory for photographic purposes unless a very small field of view is all that is required. Within such a small field these objectives far surpass almost all lenses hitherto constructed, but their chief value lies in visual use rather than in photographic work, although the fact that they are apochromatic permits focusing them with white light and afterwards inserting a suitable color screen for photographic purposes without danger of altering the focus. With achromatic objectives, on the contrary, this is not a very safe process, and generally it is better to focus with the light with which the photograph is to be taken, for their correction is usually best at about wave length, 550, and is not so good with other colors. Fortunately, this is the yellow-green color most generally useful in the photo-micrography of ordinary stained specimens.

With the higher powers, that is, lenses of 4, 3, and 2 mm. focus, this curvature of the field is less important, because the actual area photographed under any circumstances is very small and the object desired is usually a reproduction of fine details rather than a picture giving extensive topography. Up to 50 diameters, photo-micrography can be admirably done by any one of a considerable series of objectives of the photographic type without using an ocular. These may be the Zeiss tessars or planars, or the well-known miniature photo-objectives of Leitz, Winkel, or Reichert. Above this power the most satisfactory lens is the micro-luminar of Winkel of Göttingen, of 16 mm. focus. This gives a sharp picture over a $6\frac{1}{2} \times 8\frac{1}{2}$-inch plate with a magnification of 75 diameters. With care, it is possible to go a little higher with this objective, but the results are not quite so satisfactory. It is, of course, used without an eye-piece, though with the special "Complanat" oculars of Winkel slightly higher powers can be obtained with some sacrifice of definition.

At this point the possibility of computing lenses of the ordinary photographic type for use without an ocular ceases, and for higher powers we must turn to a form of lens in which the field is never perfectly flat, but in which the possible angular aperture, and consequently the resolving power, rises rapidly with the diminution in

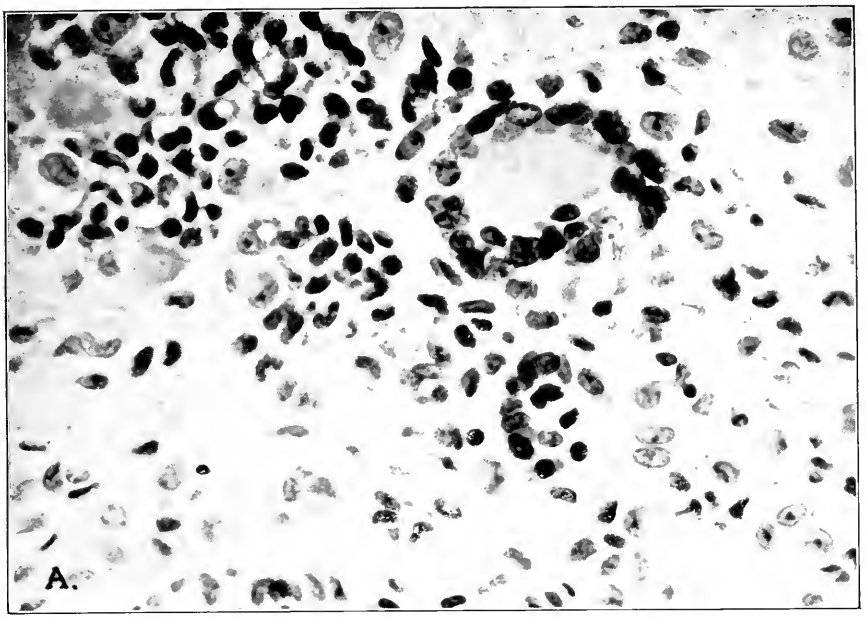

Fig. 1 (A).—Giant cell sarcoma of finger, taken with as large an aperture as lens will bear, and showing a softer effect more closely resembling images seen under the microscope. × 200.

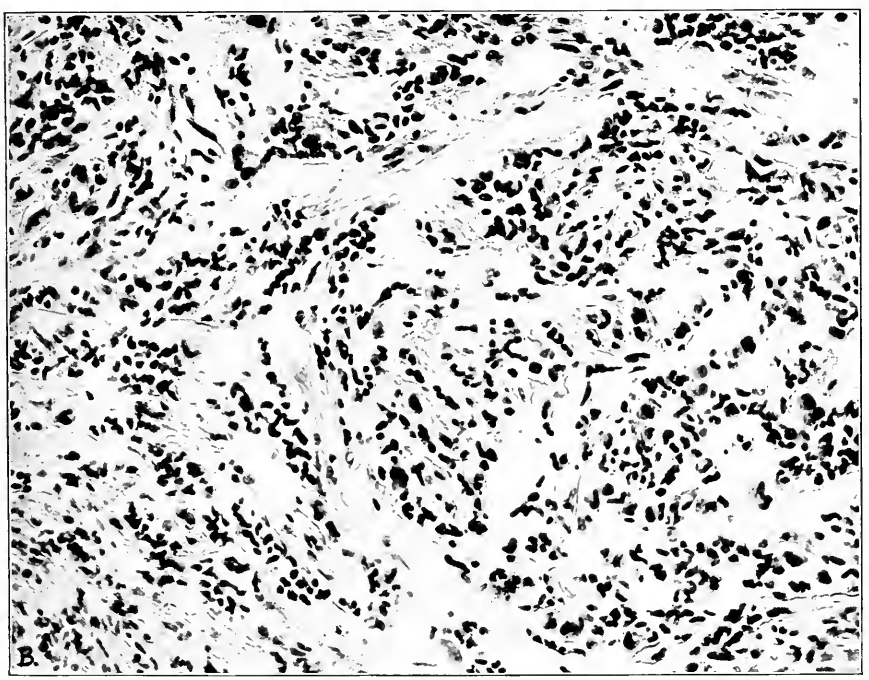

Fig. 2 (B).—Scirrhus carcinoma. The cone of light is too small; hence, the defraction images about the connective tissue. To be compared with A.

focal length. The flatness of the field usually varies inversely with the aperture; that is, the higher the aperture the smaller the area which is in sharp focus at one time. With the eye this makes but little difference, for we are constantly shifting the focus up and down and fusing a succession of pictures. As Nelson says: "Curvature of image is quite an unimportant error in a microscopic objective because all critical observations should be made in the central portion of the field, the rest of the field being used merely as a finder. If it is necessary to view large masses of an object a lower power should be used. Sharp central definition is not always compatible with flatness of field, and this sharp central definition should never be sacrificed for what, at best, is only of small importance."[1] The photographic plate, unfortunately, sees only one plane of an object, and there is no means of getting other planes into focus; consequently, it is of the utmost importance to obtain lenses of sufficient angular aperture to give all details combined with a field large enough to give topography. Usually, extreme apertures should be avoided. Even if the resolution of the details of the object over a small area is extremely sharp, it must be remembered that the only reason for taking a photograph is to produce a print which can be reproduced by a mechanical process. In the last analysis, therefore, we should think chiefly of the method of reproduction, and there is no need of having excessive detail on a plate, because some of the finer points will be inevitably lost in the print and much more in the half-tone by which the image is finally transferred to paper. Of course, this does not mean that any hazy print is sufficient, since the half-tone plate only adds more softness and haze to the original, but it does mean that we must consider the obtaining of a plate with harsh contrasts of light and shade and with moderate sharpness, rather than a thin, exquisitely detailed, smaller field, which, excellent as it may be for lantern slides, is totally inadequate for half-tone reproduction.

The most difficult magnifications to obtain with a sufficient size of field to give topographic relations are those extending from about 100 diameters to 250 diameters. A large proportion of illustrative photographs are taken at about this magnification, lower powers than 75 diameters being employed chiefly for such topographic pictures as are wanted for recording lesions of the spinal cord or the distribution of glandular elements, such as in the endometrium. These, as has been

[1]E. M. Nelson: Jour. Roy. Mic. Soc., 1907, p. 656.

stated, can be taken easily with one of the photographic type of objectives. An example of such a photograph of 75 diameters (see Fig. 4), taken with the Winkel micro-luminar, to show what that lens can accomplish, is given. It will be noted that the field is perfectly flat, covering a 6½ x 8½-inch plate, with sharp detail to the edges.

From 100 diameters on, the most usual combination is a 1-inch or one-half inch objective. Many firms make two-thirds inch or 16 mm. objectives. The older makers in England and America used to produce admirable high angle, four-tenths or one-half inch objectives, sometimes with correction collar. One of these old achromatic one-half or four-tenths inch objectives with the correction collar is a real prize, which nowadays cannot be frequently picked up. Any one who is doing photo-micrography should be on the lookout for such an objective.

The writer, for example, has one such lens, made by Tolles, with a focus of four-tenths of an inch and about 0.65 numerical aperture, which was discarded as useless by the original owner, who did not realize that the lens was corrected, of course, for the tube length in general use at the time when the lens was made; that is, a regular 10-inch "English" tube. Consequently, he found that the lens was very unsatisfactory when used on a short "Continental" stand. Of course, the images are brilliant when used on a proper length tube, and when the correction collar is screwed to its highest point the lens works splendidly at 160 mm. tube length; the field is very flat, the color correction is good. The lenses are as clear as on the day they were made, and the whole objective is a testimonial to the magnificent work that came from the hands of that great master of lens making.

It might not be uninteresting to note, in passing, that the writer has been offered one hundred dollars for this supposedly worthless lens by one who appreciates its optical qualities—a change in value almost as remarkable as some stories told of finds of first editions of old books.[2]

Such objectives, of course, are not frequently offered for sale at the present time, because they are all made for the old long-tube microscope stands now chiefly used in England, but they can usually be obtained for a small sum when they do appear in the stock of second-hand dealers.

[2]For similar records of a fine old Powell lens made in 1850, N.A. 0.385, which is practically equal to a Zeiss 16 mm., N.A. 0.35, see paper by A. A. C. Eliot Merlin, Jour. Roy. Mic. Soc., 1907, p. 646.

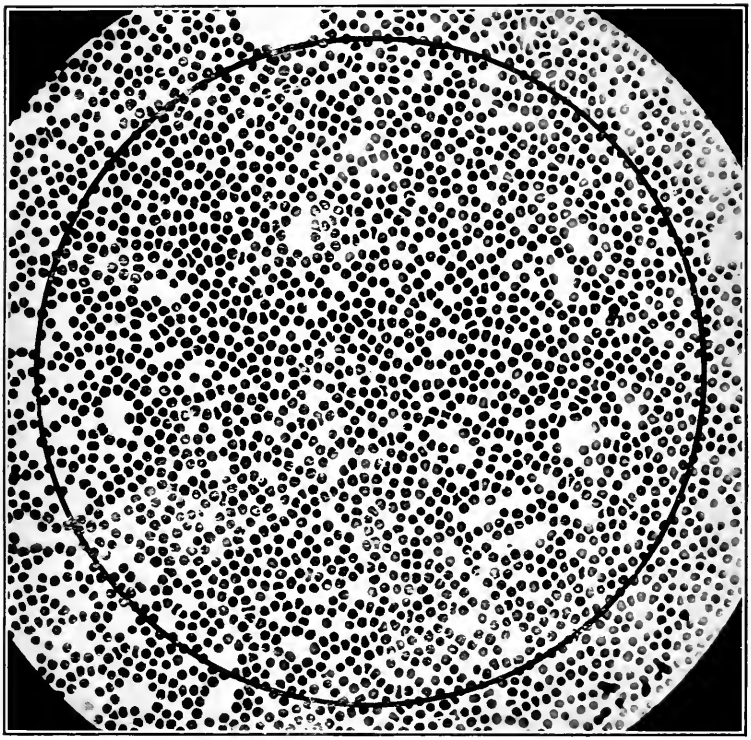

Fig. 3.—Tolles 4-10 inch, used as long tube objective. × 200. The ink ring shows the limit of the sharp field.

METHODS OF TESTING LENSES.

The best method of testing the flatness of field and the optical correction of low and medium-power objectives is, not by the eye, which continually accommodates to focal differences, but by photographing a black and white object with very sharp edges to the black lines. The most satisfactory way to obtain an object of sufficient fineness and quality is to silver one side of a cover-glass of measured thickness and then scratch fine lines through the silver coating. The cover-glass is then mounted in balsam, silver side down, and if examined will be found to show clear spaces alternating with black areas, the edges being perfectly clear cut. Such a grating for testing objectives is sold by Zeiss under the name of Abbe test plate. The ruling in this case is covered with a wedge-shaped piece of glass, from 0.10 to 0.20 mm. in thickness, so that corrections for different thicknesses of cover glasses can be obtained if the objective has a correction collar. It is not, however, necessary to purchase such a special testing apparatus, as one can easily be made as follows: A number of cover-glasses of suitable thickness are first obtained. Most dealers have measuring calipers and will select a set of cover-glasses 0.17 to 0.18 mm. thick. This is the usual thickness for which objectives are corrected. A series of such cover-glasses should be cleaned by moistening them with strong ethyl alcohol, draining off the surplus, and then pouring over the cover-glasses a few c.c. of strong nitric acid. The beaker should be immediately placed in the open air or under a fume hood, as a strong reaction will occur, very offensive fumes of nitric peroxide being given from the acid. In a few minutes, after the boiling of the acid has ceased, the surplus should be poured off and the covers rinsed repeatedly in distilled water until the water no longer reacts acid to the litmus paper. The covers should then be lifted out of the water with clean forceps and dried between two layers of filter paper, without touching them with the fingers. After blowing off any lint, they should be dropped flat on the surface of a silvering mixture so as to float. A convenient solution for this purpose is the following:[3]

One gram of silver nitrate is dissolved in 20 c.c. distilled water, and strong ammonia (0.880 sp. gr.) is added until the precipitate formed is just redissolved. A solution of 1.5 grams potassium hy-

[3]Edser and Stansfield. Nature, lvi, 504, 1897.

droxide in 40 c.c. water, and again ammonia until the precipitate redissolves; 80 c.c. distilled water are next added, and then silver nitrate solution (any strength), until there is a faint permanent precipitate. Make up to 300 c.c.

For the reducing solution, 1.8 grams of milk sugar are dissolved with the aid of heat in 20 c.c. of distilled water. The two solutions are mixed in a flat dish and the cover-glasses immediately dropped on the surface of the fluid so that they fall flat and float. The dish is covered and left quiet for an hour; at the end of that time, the silver deposit is usually thick enough, the covers are lifted out, rinsed in distilled water and dried.

Perfectly satisfactory rulings can be made by taking a fine sewing-needle (No. 11), and, making a series of light scratches through the silver in various directions, examining from time to time with a hand-lens to see that a small area, about 2 or 3 mm., is thoroughly scratched up. A more satisfactory preparation, which gives regularly spaced rulings, can be made by the use of an ordinary rotating paraffin microtome and a microscope with a mechanical stage. A strip of stiff spring brass about 25 cm. long, 1 cm. wide, and about 2 mm. thick is taken and a fine needle is fastened to the tip with a mass of sealing wax. The needle should be perpendicular to the surface of the metal. The strip is then clamped to the jaws of the holder ordinarily used for carrying the mounted paraffin blocks for cutting, and the feed is adjusted to give any convenient number of microns. The most satisfactory spacing is 50 microns, which in the ordinary paraffin microtome requires two turns of the wheel. A silvered cover-glass is fastened on a slide with some sealing wax, silver side up, and clamped to the mechanical stage, and the microscope and microtome are clamped to the table so that they do not move in relation to each other, and are so arranged that the slide is movable at right angles to the line of feed of the microtome. After the preliminary adjustments have been made, the needle is lowered into contact with the silvered surface, the springiness of the brass strip equalizing any excess pressure, and a scratch about 10 mm. long is made in the silver by moving one of the screws of the mechanical stage. The needle is lifted by rotating the microtome slightly and the cover-glass is moved out of the way; then the microtome is rotated completely, so as to feed the needle forward 50 microns, the point of the needle is again brought into contact with the silvered surface, and by moving the microtome stage parallel to the first cut and 50 microns from it, an-

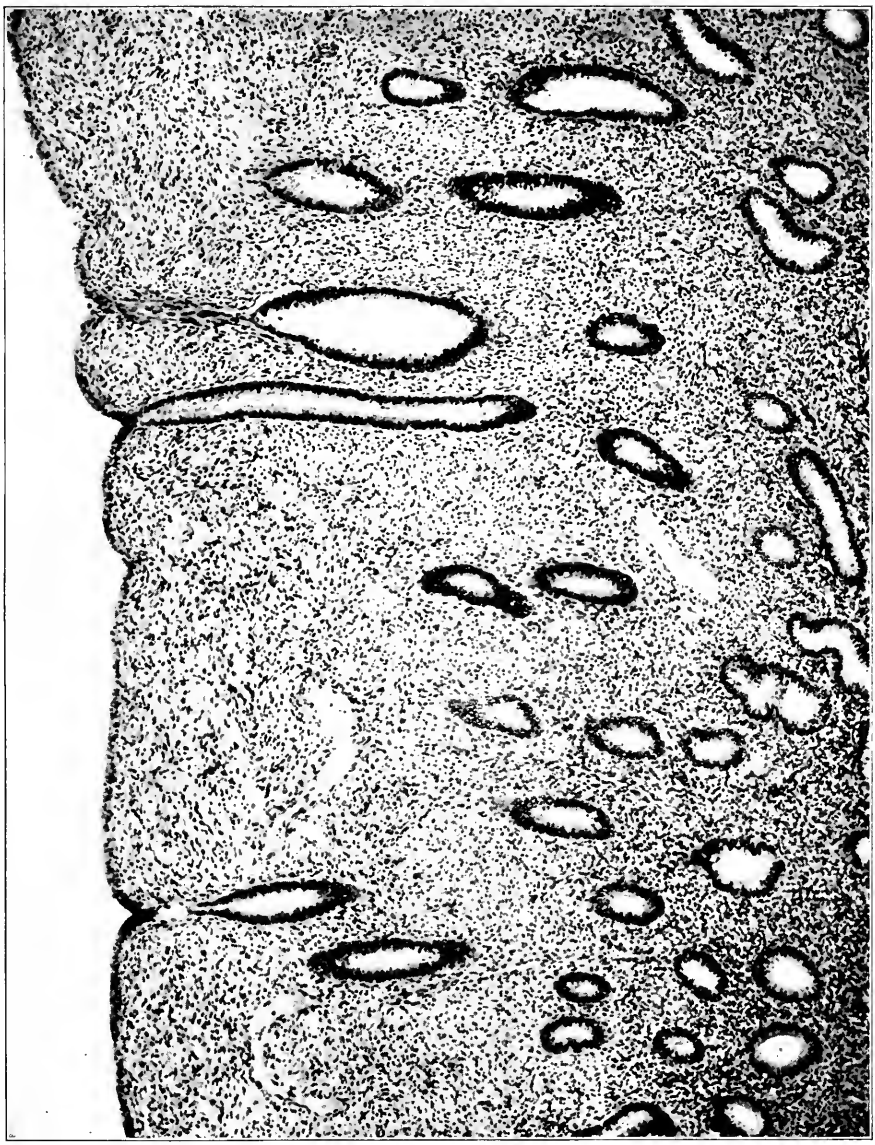

Fig. 4.—Normal post-menstrual endometrium. Winkel 16-mm. micro-luminar, with no eyepiece. × 75. The entire plate is sharp to the edges, but as it was impossible to reproduce all, an area 15 x 13 cm. was selected.

other scratch will be made in the silver. This is to be repeated until a considerable ruled area is obtained. The slide is then turned at right angles to its first position and a series of cross rulings made. The cover-glass is then freed from the surface of the slide by softening the sealing wax, and mounted in balsam. As soon as the balsam is hard, the sealing wax is cleaned off with some strong alcohol and the slide examined with a half-inch lens in order to see if the rulings are satisfactory. This will usually be so, if a very fine needle has been employed. The very finest sewing-needles (No. 11) are the best for the purpose. In order to prevent bending, the needle should be set a very short distance from its tip, in sealing wax. Not all needles have a good point, so before using one, it should be examined with a hand magnifying lens or under a low-power objective to see that the point is not turned over, as is frequently the case in finer grade needles.

If it is impossible to obtain a ruled test plate as described, an excellent object to determine the flatness of field of an objective, though not its resolving powers, is a smear of normal blood, or, for short focus immersion objectives, a slide of diphtheria or tubercle bacilli, thinly spread and faintly stained. The spread of blood should be very carefully made, if possible, on a carefully selected plate-glass slide, though the ordinary cheap slides will do if one is picked out which is flat and free from rough points on the surface. To test the flatness, hold the slide so as to get a reflection of a window-frame on its surface and see whether the lines are straight and do not become curved when the slide is rotated. Several slides should be cleaned by boiling in strong nitric acid, then washed in distilled water and dried with a cloth or filter paper, free from grease. Normal blood is then smeared over the surface of the slide, using any of the methods regularly employed in preparing specimens for diagnostic work. The smears should be thin and perfectly even, and the corpuscles separated from each other by a space equal to about their own diameter. The slide is dried and fixed in strong methyl alcohol and stained very intensely with a 1/100 solution of water soluble eosin. If desired, the leucocytes may be stained after pouring off most of the eosin by the addition of a few drops of a 1/400 methylene azure. The blood should be then mounted in balsam, using a measured cover. In order to get photographs with a satisfactory contrast, it is necessary to use a yellow-green screen and a color-sensitive plate, but as this is the light which is necessarily used with all achromatic objectives when photo-

graphing stained tissues, it does not in the least interfere with the test.

As soon as a suitable mount is obtained, the slide should be set up in the microscope, the objective inserted with a suitable projection or other eye-piece, and then the lines of the grating or the borders of the red cells carefully focused on the ground glass of the camera by the use of a hand-lens. It is necessary to see that the condenser is in proper adjustment for the lens; very few lenses will stand a cone of light filling more than one-third to one-half of the aperture of the back lens. This is best noted by focusing, then removing the eye-piece and adjusting the condenser while looking down the tube. If a Nernst light, or electric arc, or similar strong source of illumination is employed, it is necessary to reduce the intensity of the light by a piece of dark glass or a fragment of a photographic dry plate which has been exposed to daylight for a second and then developed and fixed. This will usually give a neutral tint film sufficiently opaque to prevent injury to the eye. With a Zeiss photographic apparatus such a dark glass is provided in a cap which fits into the end of the draw tube. If the photographer is fortunate enough to possess one of the old-fashioned four-tenths or one-half inch achromatic objectives made by Powell & Lealand, Tolles, H. R. Spencer, or Wales, which are provided with a correction collar, great care should be taken in seeing that this collar is turned until the best correction is obtained for spherical and chromatic aberration. The colored fringes seen at the edge of the black lines are present, to some extent, with all achromatic objectives, especially at the periphery of the field, and their complete removal, except at the center, is not so important as the perfect correction of the spherical aberration, as is shown by the perfect sharpness of the edges of the silver bands or the blood cells. Most objectives are now corrected for a shorter tube length, usually either 160 or 170 mm., and marked, as a rule, with the proper length for the draw tube. It is necessary, if the objective is not screwed directly into the nose-piece, to allow for either 10 mm., in case of a revolving nose-piece, or 22 mm., in the case of the Zeiss sliding objective changers. The old-fashioned achromatic objectives with correction collar, even when computed for the long tube, often work admirably on the modern short tube-stand if there is a sufficient range of collar adjustment.

As it is not always easy to judge the point at which definition begins to fall off, it is usually better to make a photograph rather than

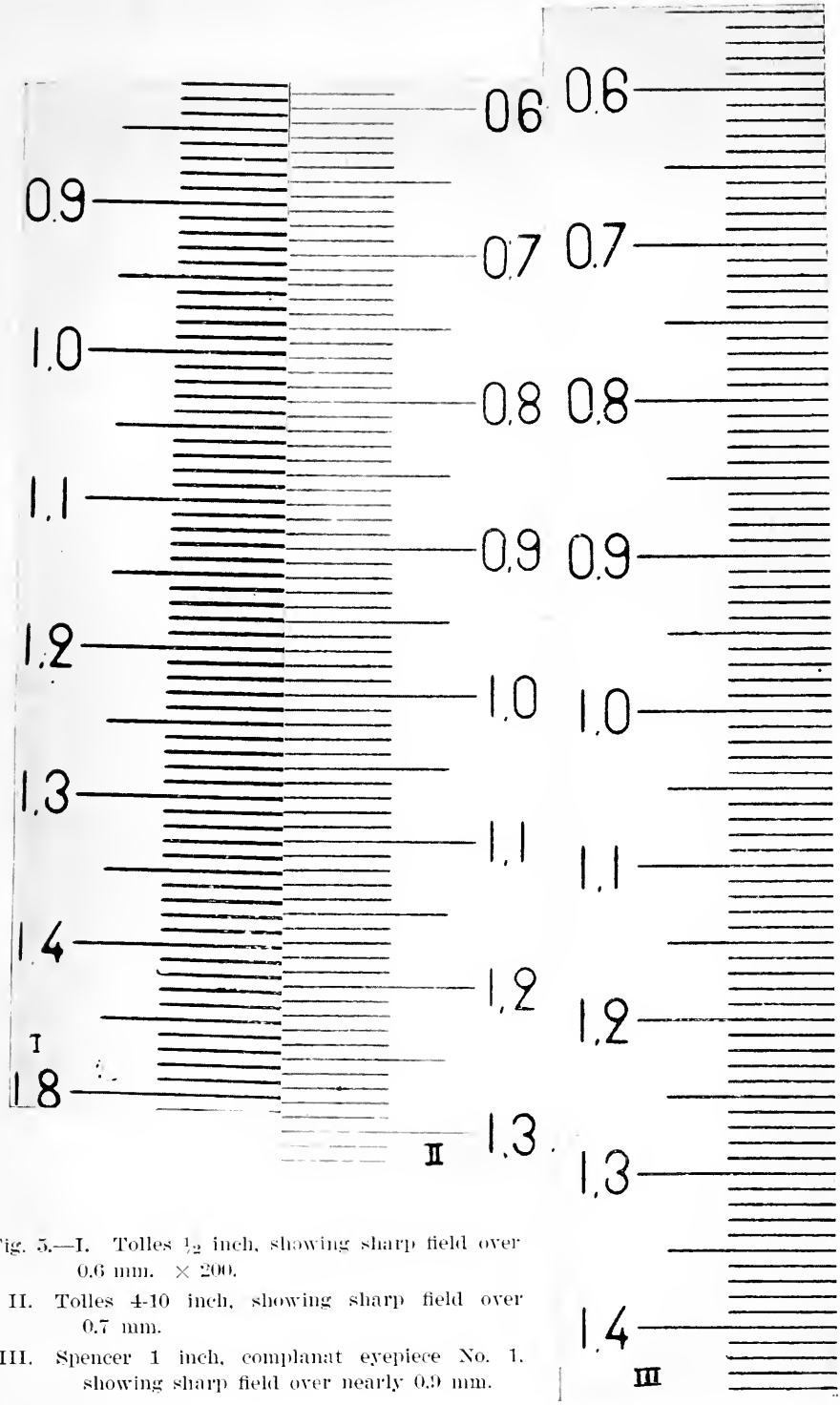

Fig. 5.—I. Tolles ½ inch, showing sharp field over 0.6 mm. × 200.

II. Tolles 4-10 inch, showing sharp field over 0.7 mm.

III. Spencer 1 inch, complanat eyepiece No. 1, showing sharp field over nearly 0.9 mm.

to rely entirely upon the eye. The most suitable plate for this purpose is the Cramer isochromatic double-coated plate, which has great latitude of exposure and stands prolonged development without fogging, and yet is sensitive to the yellow-green color used in tissue photography.[4] The exposure should be short and the development prolonged in order to bring up all possible contrast. When the negative is dry a print can be made from it if desired, and the quality of the image judged from the print, but to any one who is experienced in looking at negatives, it will be easy to determine the approximate point at which the sharpness of image ceases, and that, of course, is the size of the useful field of the objective. It is generally somewhat easier to tell the exact point from a blood smear photograph than it is from the photograph of the ruled screen. The screen, however, affords a better test of the optical qualities of the objective than the corpuscles, because the edges of the silver bands are extremely sharp and the slightest haziness or color is shown in the photograph as a lack of sharpness to the edge. Another definite way of getting the diameter of the field, though it is not a satisfactory one for the resolving power of the objective, is to photograph a stage micrometer. If the lines are black or the micrometer used is one of the photographic reproductions, the diameter of the useful field can be fairly easily told; but, as just stated, it is not a satisfactory test for resolution, because by varying the time of exposure, the screen and the development, the apparent sharpness of the lines can be varied and a poor objective will give, on a thoroughly developed plate, a much better picture than a good objective on a thin plate.

In order to get the best definition the lens should be used with an achromatic condenser of approximately the same focus as the objective. If such a condenser is not available, a lens of about the same aperture and focus can be used in the substage as a condenser with the most satisfactory results. On looking down the tube of the microscope, the illuminated area of the condenser diaphragm should be central, and the maximum amount of light used which the objective will bear without the image becoming hazy. If the light is not centered or if the diaphragm is reduced to too low a point, the diffraction lines will appear at the borders of the red corpuscles or at the edges of the rulings of the Abbe test plate. It is, unfortunately, only too

[4]It is not necessary to go into further details concerning plates or developer here; possibly in a subsequent number of this report the subject of plates, screens, developers, etc., will be fully treated.

common to see photo-micrographs in which the diameter of the sharp field of the objective has been increased by the process of reducing the cone of light thrown by the condenser to a very small diameter. The result is that, while the field covered by the objective is slightly larger, diffraction lines are present about the borders of the nuclei, and the bodies of the cells and all finer details are lost. The advantage of the use of lenses of large aperture is that they stand a good deal of light without the image becoming hazy, and therefore the exposure may be shortened with equal or better definition. Influenced, perhaps, by the constant habit of looking at or making drawings of histological material with a pen, the average person thinks that a photo-micrograph showing diffraction lines represents more accurately the appearances usually present under the microscope than a much softer picture obtained by the use of a high-angle immersion lens, but a little study of stained sections under the microscope with high-grade lenses and a suitable condenser and light will show that, on the contrary, few cells have a sharp outline, and that in well preserved material each shades into the next contiguous cell without any great contrast. The routine fixation and hasty paraffin embedding of much of the material ordinarily examined has also contributed not a little to the sharp outline attitude, for the inevitable shrinkage following such procedures tends to isolate cells or groups of cells and thus leave clear spaces about them.

AREA OF USEFUL FIELD.

The results of the examination of photographs of ruled silvered plates, of blood slides, and of stage micrometers is shown in the appended table. The magnification chosen was a constant one; that is, 200 diameters, this being the maximum magnification likely to be used with medium power objectives; above that point, 6 or 4 mm. lenses are to be preferred, though an exceptional 8, 10, or 12 mm. lens may permit a useful magnification of 250 to 300. This is much lower than the theoretical "useful magnification," which is usually given as 100 times the numerical aperture; in other words, a lens of N.A. 0.20 should give a good image at 200 diameters, one of 0.30 at 300 diameters, one of 0.65 at 650 diameters; but in practice but few objectives will give more than half this, and the ordinary cheap commercial achromats not more than a third or even a fourth. The test is therefore much more severe on the 16 to 25 mm. lenses than on those of shorter focus. Possibly all that can be expected of inch

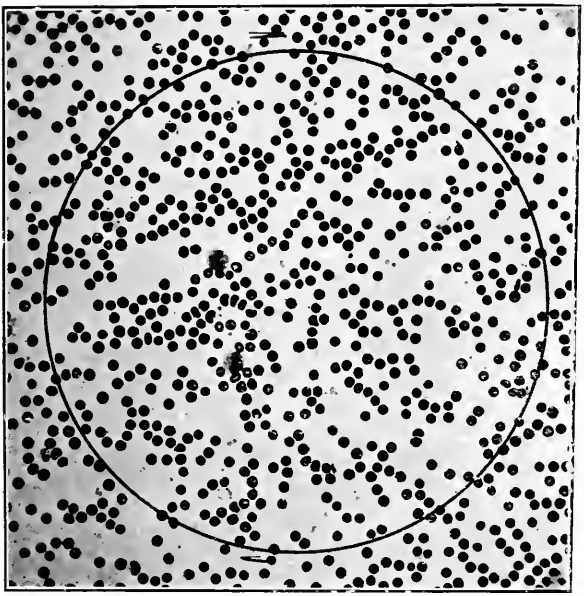

Fig. 6.—Zeiss 8-mm. apochromat. with No. 3 projection eyepiece. × 200. The ink ring shows the limit of the sharp field.

objectives is a maximum of 125 to 150. A few lenses, even though their aperture is relatively low, will give good pictures at 200 diameters, the best example the writer has seen being the Winkel apochromat of 25 mm. focus with a N.A. of 0.22. This exceptional lens stands 200 diameters better than many of much shorter focus and larger aperture, with a sharp field covering a 6½ x 8½ plate.

The objectives, concerning which further details are given in Table II, fall naturally into two groups; the first 8 of 12 mm. or less in focus, the last 5 of between 16 and 25 mm. focus. Of the half-inch lenses, the Tolles one-half is the superior though its low aperture of 0.42 requires more careful handling of the illumination than the three which follow. The Spencer Lens Company's 8 mm. apochromat is one of the best for photography now being made. The Watson lens is only fair. The Powell and Lealand, though still made, is not to be recommended. The particular type of Bausch and Lomb half-inch is no longer listed by that firm, but is a very fine lens. Last of all comes the 8 mm. Zeiss, with so small a field that its usefulness is much restricted, though within that field it gives the best and sharpest images of any objective examined. In the second group, the maker of the Spencer lens is long since dead, but the lens is a remarkable one. Close to it is the Winkel apochromat, which is the best lens now obtainable for low-power photography; that is, between 75 and 200 diameters. The Zeiss and Leitz 16 mm. are very good for within the sharp field, but this is much too limited for photographic purposes.

A long series of tests have also been made with low-power achromatic lenses of both foreign and domestic makers, but though, with patience and great care, fair results can be obtained, they are not wholly satisfactory. The optical requirements for photographic work are much more severe than for purely visual effects, for which these lenses are intended. In the higher powers, however, some excellent lenses are obtainable, especially the "flourite" objectives of Leitz and Reichert, but better work can be done with the 4 and 6 mm. dry apochromats of Zeiss, and still better with Powell and Lealand's quarter-inch apochromatic immersion. For the highest magnifications, such as are required for photographing bacteria, the improved achromatic 1/12-inch oil immersions are very satisfactory, though nothing quite equals the Zeiss 3 mm., N.A. 1.40.

I.—TABLE OF LENSES ARRANGED ACCORDING TO DIAMETER OF USEFUL FLAT FIELD AT A MAGNIFICATION OF 200 DIAMETERS.

Lens	Diameter of Field in Millimeters with Zeiss II. Projection Eye-piece	
Tolles ½ inch.	110 mm.	Definition satisfactory.
Tolles $^4/_{10}$ inch.	100 mm.	Used with 160 mm. tube.
	85 mm.	Used with 250 mm. tube. Definition about the same, but better than the ½ inch.
Wales $^4/_{10}$ inch.	90 mm.	
Powell and Lealand ½ inch achromatic immersion.	90 mm.	
Spencer Lens Co. 8 mm. apochromat.	75 mm.	Very good definition.
Watson ½ inch holostigmat.	70 mm.	
Powell and Lealand ½ inch apochromat 160 mm. tube.	75 mm.	Fair definition; much better than would be expected from resolution tests.
Bausch and Lomb ½ inch, Series III.	60 mm.	Very good definition.
Zeiss 8 mm. apochromat.	60 mm.	Definition better over this area than any of the above lenses, except the immersion P. & L.
Leitz 8 mm. apochromat.	60 mm.	Definition even better than the Zeiss in the lens examined, but not equal to the immersion.
H. R. Spencer 1 inch 250 mm. tube.	115 mm.	With Winkel Complanat Eye-piece No. 1 field is 200 mm., with excellent definition.
Winkel 25 mm. apochromat.	160 mm.	With Complanat Eye-piece No. 2, 180 mm. and very good definition.
Zeiss 17 mm. achromat AA.	180 mm.	Very fair definition.
Watson 24 mm.	170 mm.	Field, but very poor definition all over; will not give good picture at over 150 diameters.
Zeiss 16 mm. apochromat.	70 mm.	Good definition.
Leitz 16 mm. apochromat.	75 mm.	Good definition.

II.—TABLE OF RESOLUTIONS OF SERIES OF LENSES.

The diatoms employed were Nitschia scalaris (abbreviated N.S.), 26,000 lines to the inch, mounted in styrax, and Pleurosigma angulatum (abbreviated P.A), 44,000 lines to the inch, mounted in realgar.

Grade	Lens	Object	Resolution Central Light	Resolution Oblique Light
100	Tolles achromatic $^4/_{10}$ inch or 10 mm. N.A.=0.65 for 250 mm. tube.	N.S.	Easily into dots, even better than Zeiss 8 mm.	Very easily into dots.
		P.A.	Easily into dots.	Easily into dots.
100	Zeiss or Leitz 8 mm. apochromatic. N.A.=0.65 for 160 mm. tube.	N.S.	Easily into dots.	Easily into dots.
		P.A.	Easily into dots.	Easily into dots.
90	Wales achromatic $^4/_{10}$ inch or 10 mm. N.A.=0.60 for 160 mm. tube.	N.S.	Dots.	Dots.
		P.A.	Easily into dots.	Easily into dots.
75	Spencer Lens Co. 8 mm. apochromatic. N.A.=0.60 for 160 mm. tube.	N.S.	Dots.	Dots poorly.
		P.A.	Dots.	Dots.
70	Watson holostigmat 12 mm. N.A.=0.45 for 170 mm. tube.	N.S.	Good resolution, but only lines.	Fairly into dots.
		P.A.	Not resolved.	Fairly into dots.
70	Bausch and Lomb, Series III, 12 mm. N.A.=0.54 for 210 mm. tube.	N.S.	Fairly into lines.	Fairly into dots.
		P.A.	Fairly into dots.	Fairly into dots.
65	Tolles ½ inch. N.A.=0.42 for 250 mm. tube achromat.	N.S.	Easily into lines.	Fairly into dots.
		P.A.	Not resolved.	Poor resolution in dots.
65	Powell and Lealand ½ inch or 12 mm. apochromatic. N.A.=0.64 for 250 mm. tube.	N.S.	Lines only.	Dots poorly.
		P.A.	Not resolved.	Not resolved.
60	Herbert R. Spencer 1 inch or 25 mm. N.A.=0.35 achromat.	N.S.	Lines only.	Lines only.
		P.A.	Not resolved.	Not resolved.
60	Zeiss 16 mm. N.A.=0.30 apochromatic for 160 mm. tube.	N.S.	Lines only.	Lines only.
		P.A.	Not resolved.	Not resolved.
50	Zeiss 17 mm. A.A. achromat. N.A.=0.30.	N.S.	Lines only.	Lines only.
		P.A.	Not resolved.	Not resolved.
45	Watson 24 mm. N.A.=0.24 holostigmat.	N.S.	Just shows lines.	Just shows lines.
		P.A.	Not resolved.	Not resolved.
45	Winkel 25 mm. N.A.=0.22 apochromat.	N.S.	Barely shows lines.	Barely shows lines.
		P.A.	Not resolved.	Not resolved.

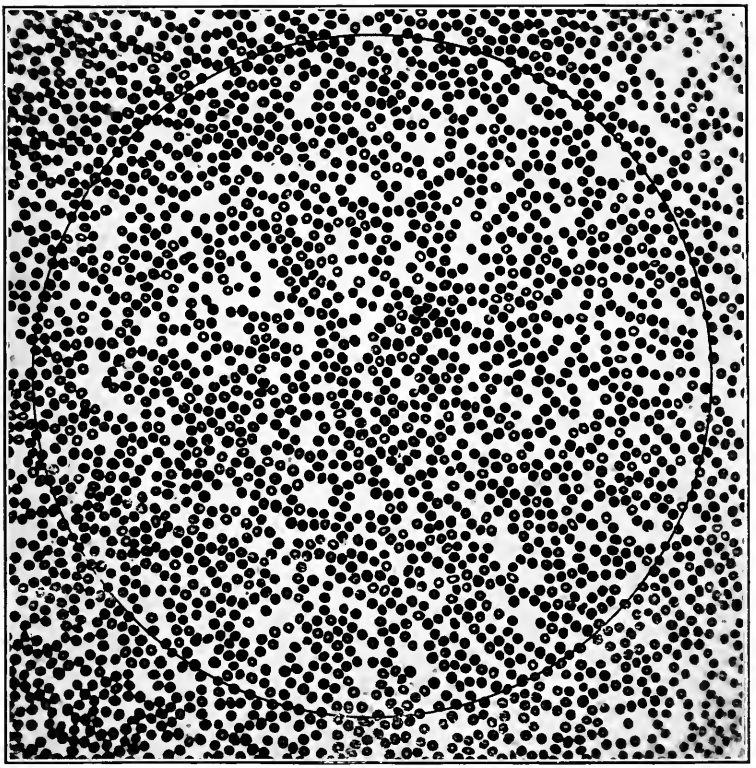

Fig. 7.—Powell and Lealand ½-inch apochromat × 200. The ink ring shows the limit of the sharp field.

The resolution, or ability to reproduce fine details, also varies directly with the numerical aperture of the lens, but a lens of high aperture may not be so satisfactory as one of slightly smaller opening if its spherical and chromatic corrections are not also perfect.

An example of this is seen in the Powell and Lealand half-inch apochromatic, which will be discussed later. Theoretically, this lens should be an excellent one, but practically it is worthless.

Resolution being equal, the best lens in every respect is the one giving the largest field. With the low powers it is possible that the entire field may not be employed for reproduction, but it is very convenient to have a little leeway so that the exact centering of the image to be obtained is not necessarily carried out under the microscope, but a general field selected from which a suitable part may be cut for final use as an illustration. The selecting and absolute centering of an exact field on the ground glass of the camera focus is time-consuming, and it is difficult, without a great deal of experience, to judge the final effect of a tissue photograph, either in the camera or from a negative; the print is the best criterion.

The most accurate means of testing the resolution alone, because the mere marking of the angular aperture on the objective does not guarantee that the measurement is correct, is to use a diatom, suitably mounted. The objectives under consideration range from 25 to 8 mm., with a numerical aperture of from 0.22 to 0.65. There are two diatoms which offer convenient standards. These are the Pleurosigma angulatum and the Nitschia scalaris. They should be mounted in a highly refracting medium, such as realgar, or, if such preparations cannot be obtained, dry. The light used must be intense; direct sunlight, if properly screened, is good; or the direct light from a Nernst filament or miniature arc lamp, or even a flat-wick kerosene lamp is preferable to daylight. The condenser must focus the light on the object. Under such conditions it is remarkable what a good lens will do. For instance, the writer has been able to resolve Amphipleura pellucida in realgar with the Zeiss 4 mm. short-tube apochromatic lens and a dry Watson parachromatic condenser, using a suitable blue screen. Usually, nothing but an immersion lens will show the lines on this most difficult object.

The table on page 238 shows the results of the examination of a series of lenses in the possession of the writer—a rough estimate in percentage is given to aid in classification, but has no claim to accuracy. It will be seen that the resolution of Pleurosigma in dots by

central light immediately divides the objectives into two classes, those above N.A. 0.50 and those below, though a Powell and Lealand one-half N.A. 0.64, falls, for some unknown reason, into the lower class. The Nitschia scalaris gives another dividing line when resolved into dots by oblique light at N.A. 0.40. All of the objectives thus tested respond pretty closely to the theoretical limits, those usually set being a numerical aperture of at least 0.65 for the resolution of the dots of P. angulatum with an axial light, slightly less for lines, and at least 0.30 for N. scalaris in styrax; the only exception being the Powell and Lealand half-inch, N.A. 0.64, which falls way below its class, being surpassed by the Watson and Bausch and Lomb half-inch objectives of considerably lower aperture and equalled by a Tolles half-inch achromatic, N.A. 0.42. The flat field of this lens is almost twice that of the Powell and Lealand, and for photographic work the objective is much superior, though made some 40 years ago, before the discovery of the Jena glasses and the computations of Abbe, permitting apochromatic lens construction.

In the same way the Tolles 4/10-inch is equal to, and possibly surpasses in resolving power, the best modern product of Zeiss, the 8 mm. apochromat, while its field is much larger. Close behind is an old achromatic 4/10-inch Wales, with slightly lower aperture, but with a large, flat field and exquisite definition. Either of these old lenses are much superior to the modern achromatic lenses of any maker for the purposes of photography, if we except a Powell and Lealand half-inch immersion, specially made for the writer, with a numerical aperture of 1.30. This has a flat field of 90 mm. and gives better definition and greater resolution than any dry lens. It is, however, purely a photographic objective, and not useful for other purposes.

Some improvement in the flattening of the field can no doubt be obtained by improvement in the eye-pieces employed in photographic work. A step in this direction has been taken by Winkel, whose so-called "complanat" eye-piece gives a somewhat flatter field with his objectives than with the projection type of eye-piece made by Zeiss, and, in fact, the performance of the old achromatic objectives is often better with a complanat than with a projection eye-piece.

CONCLUSIONS.

A complete outfit of lenses suitable for the highest class of photomicrography is expensive, and, though fair results can be obtained by

the use of the cheaper grades of achromatic lenses generally fitted to microscopes, quite satisfactory for ordinary visual work as they may be, yet good photographs can only be made with the finest lenses, for defects which the eye will entirely overlook will become most apparent when a lens is used for photography. An ideal outfit would be the following:

1. Zeiss planar 75 mm. focus for very low powers.
2. Winkel micro-luminar, 16 mm. focus, for magnification from 25 to 75 diameters.
3. Winkel 25 mm. apochromat with complanat eye-pieces I and II, for 75 to 150 diameters.
4. Tolles, Spencer, or Wales $^1/_{10}$ or ½-inch for from 150 to 200 diameters. In lieu of these a Spencer Lens Company's 8 mm. apochromat is the best now on the market as regards flatness of field. The Zeiss or Leitz 8 mm. apochromat is more expensive and has a smaller field, though giving superior definition.
5. A Powell and Lealand ¼-inch apochromatic immersion, N.A. 1.30, for 200 to 500 diameters. As this lens is expensive, a dry apochromat 4 or 6 mm. of Zeiss, or any other standard make, such as Winkel, Leitz, or Reichert, may be substituted, but the flat field is smaller than in the immersion and the lenses more difficult to handle because of the necessity for careful adjustment of the light and their sensitiveness to varying thicknesses of cover-glass. The Powell and Lealand immersion is the most satisfactory lens made for medium powers.
6. A Zeiss 3 mm. apochromat, N.A. 1.40, for the long tube. This is one of the most remarkable lenses made. It gives a range of from 500 to 1,400 diameters with the Zeiss III and VI projection eye-pieces, which are also to be employed with lenses IV and V. It is less easily injured than the 2 mm. Zeiss apochromat, N.A. 1.40, the front lens of which may be dismounted by the slightest touch to a cover-glass, and has a larger field. The long tube, 3 mm., is a shade better than the short tube lens of the same aperture and focus. In fact, all of the long tube objectives give better results than the short tube ones, as the same magnification can be obtained with a lower eye-piece.

CASE OF INCOMPLETE RUPTURE OF THE HEART DUE TO CORONARY HEMORRHAGE.

J. Gardner Hopkins, M.D.

The patient was a woman of fifty years, a designer by occupation. Except for the diseases of childhood, she had always been well up to four days before admission to the hospital, when she was suddenly seized with a feeling of suffocation while at work and had to be taken home. This attack was followed by rather severe constant pain in the precordium, which increased on deep breathing. She had no other symptoms. As the pain continued, she came to the hospital and was admitted on Dr. Janeway's service. On examination, the apex beat was not made out. The heart dulness was apparently increased; the sounds were distant but normal; no murmurs were heard. The pulse was regular and of good force. After rest in bed, the pain disappeared, and on the third day the patient was allowed to sit up, with the expectation of discharge in a few days. While being wheeled to her bed after defecation, she became deeply cyanotic, fell forward in her chair, and apparently died instantly.

At autopsy, the pericardium contained coagulated blood which formed a thick layer about the anterior, posterior, and right surfaces of the ventricles and extended up about the aorta and pulmonary artery. The clot was thickest at the apex posteriorly, where it measured 2.5 cm. The ventricles and right auricle were in systole and left auricle in diastole (Fig. 1). The leaflets of all the valves were thickened and those of the mitral showed atheromatous plaques. There was no evidence of endocarditis. In the apex of the left ventricle was a blood clot about 2 cm. in diameter, and in the anterior wall near the septum was a cleft filled with blood clot continuous with that in the ventricle (Fig. 2). This cleft extended downward and to the left, following the course of the muscle fibers. At the left border of the heart it reached the subpericardial fat and extended through the fat, communicating with the pericardium apparently at the apex posteriorly, though the precise point of communication was not made

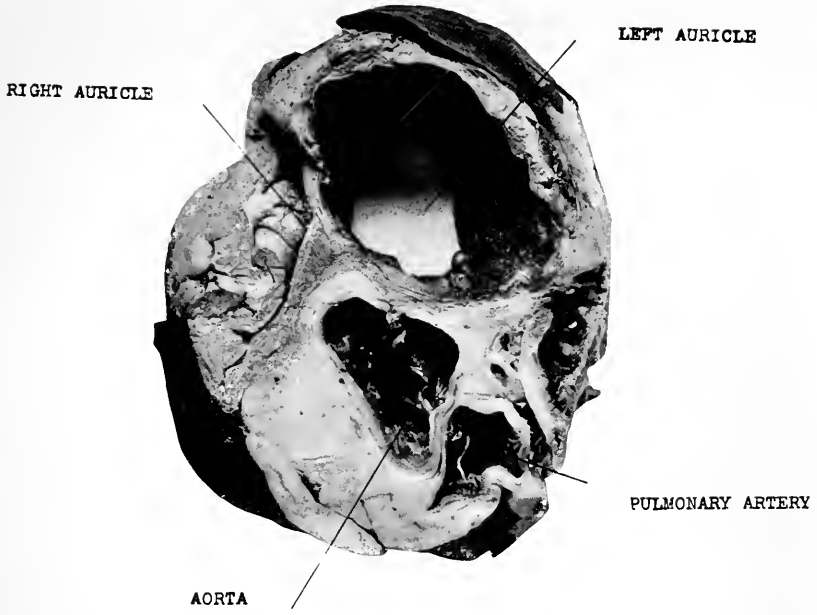

Fig. 1.—Section through base of heart, viewed from above, showing compression of right auricle and dilatation of left auricle.

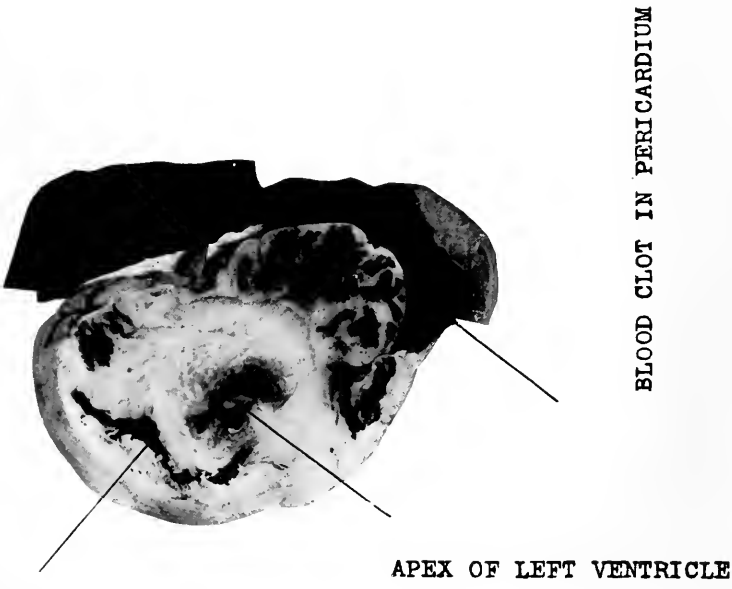

Fig. 2.—Section through heart, near apex, viewed from below, showing rupture in the wall.

out. There was also hemorrhage in the fat below and about the apex of the right ventricle. The blood clot in the ventricle was covered by a delicate membrane, which, on section, consisted of a fibro-cellular membrane covered with endothelium, and was evidently the remains of the endocardium, showing that the blood had lain beneath the endocardium and not actually in the ventricular cavity. The coronaries showed extensive sclerosis, and the descending branch of the left coronary was much thickened and diffusely calcareous. The external diameter of this vessel was about 4 mm., but its lumen was very small. About 4 cm. from its origin the lumen was practically occluded by the thickening; but could be traced, on section, 1 cm. or more below this point (Fig. 3). The anterior wall of the left ventricle in the region supplied by this artery was pale yellow and glistening and translucent on section. There were other smaller tears in the muscle, also filled with thrombi, which did not extend to the pericardium. Microscopically, the muscle cells in this area were shrunken and hyaline in appearance. In some areas they showed no nuclei; in others they had small, deeply staining nuclei. Between the necrotic cells there were rows of polymorphonuclear leucocytes, among which were a few small, round cells. In places the leucocytes were collected in masses resembling small abscesses (Fig. 4), in which a few partly calcified fragments of muscle cells could be seen. No bacteria were seen in these areas. There were also many leucocytes about the tears in the muscular wall. The small arteries in the wall were thickened and some were filled with organized thrombi (Fig. 5). The pericardium was thickened, due chiefly to infiltration with small round cells and large cells of endothelial type.

The aorta showed extensive arteriosclerosis with calcification, and there was marked interstitial nephritis.

The anatomical diagnosis was: Arteriosclerosis of aorta and coronary arteries. Myomalacia of anterior wall of left ventricle. Incomplete rupture of the heart due to hemorrhage from a coronary vessel. Hemopericardium. Chronic interstitial nephritis. Adenoma of renal cortex. Chronic passive congestion and fatty degeneration of liver. Emphysema, congestion, edema, and healed tuberculosis of lungs.

Rupture of the heart is among the rarer causes of sudden death and has aroused much interest since the first case described by Harvey. Morgagni described a number of cases from his own experience, but the lesion appears to be much less frequent in recent times. Elleaume collected sixty-one cases, thirty-seven of which were in men

and twenty-four in women. The rupture is usually very minute, as in this case, and usually larger externally than internally. The cleft, as a rule, follows the course of the muscle fibers. Occasionally there is a long tear, in one case from the base to the apex; and from three to five multiple tears have been reported. Forty-three of fifty-five cases involved the left ventricle, and the usual point is in the anterior wall, near the apex. The rupture may follow embolic or sclerotic occlusion of the coronary artery with subsequent softening of the wall. Abscesses in the myocardium, gummata, and tumors have also led to rupture. Ten of Elleaume's cases were due to rupture of an aneurism of the heart, which is a relatively frequent cause. Quain suggested that diffuse fatty change might lead to rupture, but this seems unlikely as it lessens the force of the heart action and would rather tend to prevent rupture.

Rupture of the heart occurs in old age and usually after severe exertion. It sometimes occurs without any apparent occasion, and even while the patient is asleep. In other cases it may follow psychical excitement, as in the case of Philip the Second of Spain, who died of rupture of the heart when told of the defeat of his armies.

In the case reported here, rupture was evidently due to degeneration of the myocardium in the region supplied by the descending branch of the right coronary artery. The sections of the heart muscle present a typical picture of acute suppurative myocarditis, but the history of the case is very much against the supposition that the process was infectious. There was no evidence of endocarditis and pericarditis, or any other condition which would account for the origin of an infectious myocarditis. It is probable that the collections of leucocytes were not due to bacterial infection, but to reaction about the necrotic tissue. These collections of leucocytes are commonly found in softening of the myocardium. The fact that the inner blood clot lay beneath apparently intact endocardium makes it seem probable that the rupture was due to hemorrhage from some coronary vessel which penetrated internally beneath the endocardium and externally into the pericardial sac. There was no escape of blood from the ventricle. From the amount of reaction about the clot, the first hemorrhage into the wall probably occurred when the patient had the first attack of dyspnea, and the terminal event was rupture of this intramural hematoma into the pericardium.

The most interesting feature of the case is the condition of the auricles. In a section through the base of the heart (Fig. 1) the left

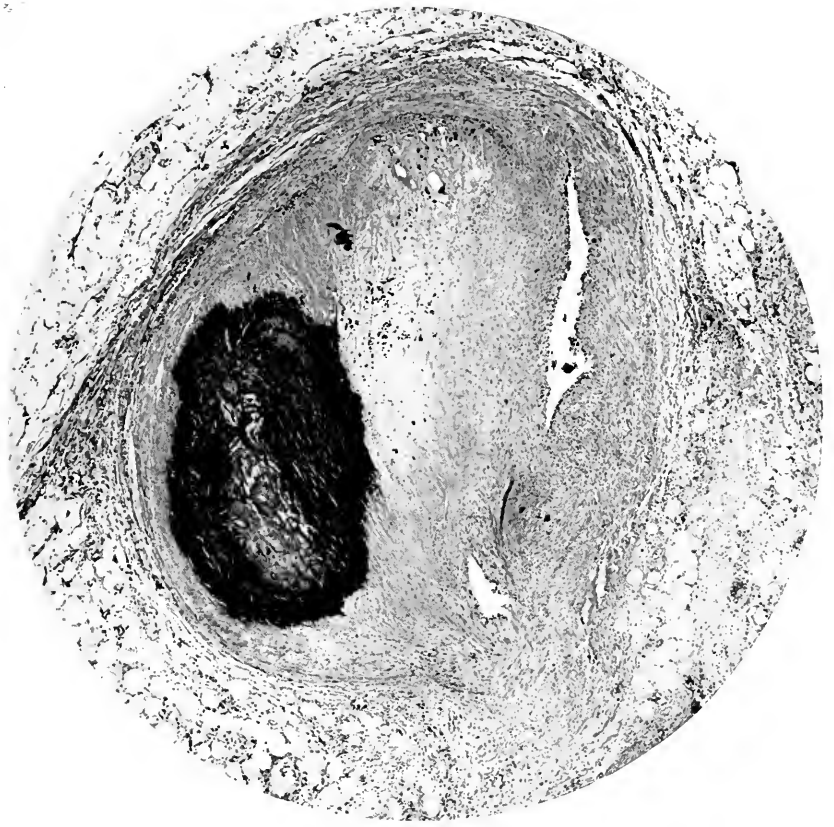

Fig. 3.—Photomicrograph showing cross section of the descending branch of the left coronary artery. The lumen is obliterated, except for two narrow slits at the right. To the left is a calcified area. 20 diameters.

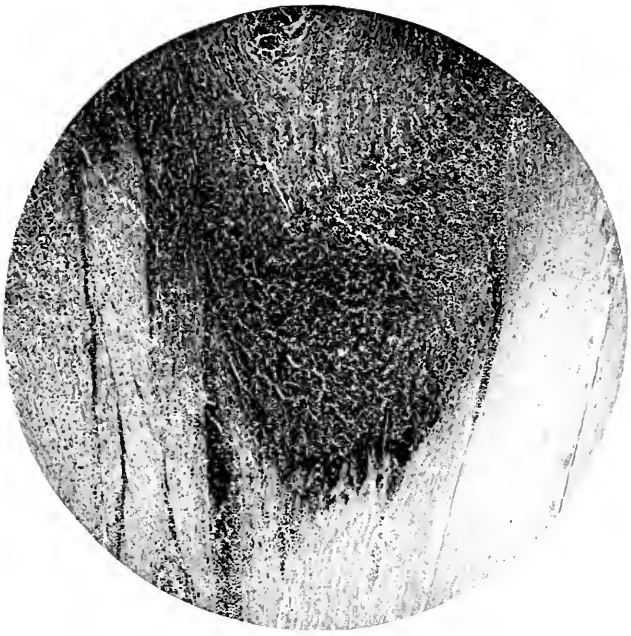

Fig. 4.—Photomicrograph showing collection of leucocytes in necrotic heart muscle. 45 diameters.

Fig. 5.—Photomicrograph showing remains of endocardium covering the blood clot in the ventricle. 225 diameters.

Fig. 6.—Photomicrograph of the necrotic heart muscle, showing small thrombosed vessel, surrounded by leucocytes. 260 diameters.

auricle is seen to be in diastole, while the right auricle is compressed to a mere slit. This illustrates clearly the theory of Cohnheim that death in hemopericardium is due to compression of the right auricle, the chamber in which the blood pressure is the lowest. He injected fluid into the pericardial sacs of dogs and showed that the sudden injection of 150 c.c. to 200 c.c. was sufficient to cause death, whereas, a much larger amount of fluid might accumulate gradually in the pericardium without a fatal result. The effect depends upon the tension of the fluid rather than upon its amount. As the pericardial pressure approaches the pressure in the right auricle it interferes with the entry of the blood into the heart from the systemic veins, causing a rise in venous pressure and a fall in arterial pressure. The pressure in the left auricle is considerably greater than in the right, and consequently the entry of blood from the lungs is not interfered with until the pericardial pressure is increased considerably above the point necessary to compress the right auricle.

References.—Cohnheim, Allegemeine Pathologie. Trans. by McKee, vol. i, p. 30. Elleaume, Essai sur les ruptures de cœur. Thèse de Paris, 1857. Friedreich, Virchow's Handbuch f. spezielle Pathologie u. Therapie, vol. v, sec. 2, p. 183. Morgagni, De sedibus et causis morborum, book ii, letter 27. Quain, Medical and Chirurgical Transactions, London, vol. xxxiii.

REPORT OF THE WASSERMANN REACTIONS DONE BY THE PATHOLOGICAL DEPARTMENT DURING THE YEAR 1911.*

C. H. Bailey, M.D.

During the past year 597 Wassermann reactions have been done by the Pathological Department. The results of the reaction on the 423 cases whose histories were accessible were as shown in the following table:

	Positive	Doubtful positive	Doubtful negative	Negative
Syphilis:				
Primary	6	1	0	1
Secondary, untreated	20	0	0	1
Secondary, treated	7	0	1	4
Tertiary	54	11	1	10
Latent	24	9	1	26
Congenital	2	1	0	2
General paresis	1	0	0	0
Tabes	5	2	1	2
Diseases possibly of syphilitic origin:				
Aneurism	6	2	0	0
Aortic insufficiency	9	0	0	5
Facial paralysis	1	0	0	2
Chronic inflammations of the eye	7	2	0	13
Diseases not diagnosed clinically as syphilitic	1	2	3	177

Those cases are classed as positive which gave complete inhibition in the tube containing 0.2 c.c. of the patient's serum and antigen, with complete hemolysis in the control tube containing 0.4 c.c. of the patient's serum without antigen. Those in which there was a slight trace of hemolysis in the tube with antigen and complete hemolysis in the control, and those which, with complete inhibition in the tube with antigen, showed a trace of inhibition in the control, are classed as doubtful positive. Those with partial hemolysis with antigen or

*A portion of this article appeared in the Archives of Internal Medicine, May, 1912.

partial inhibition in the control are classed as doubtful negative; all others as negative.

The three cases classed under "Diseases not diagnosed clinically as syphilitic" which gave positive or doubtful positive reactions require special mention.

Rheumatoid Arthritis.—Wassermann positive. No history of syphilis obtainable. Patient complained of pain and swelling in her right great toe of three weeks' duration. Pain in right ankle and left arm for one week. On entrance, toe was swollen, somewhat red, and tender. Slight tenderness over inner aspect of left tibia. Temperature normal. During her five weeks in hospital both elbows and several phalangeal joints were involved. Patient was on mixed treatment eleven days, potassium iodide being continued nineteen days longer, without improvement.

Gelatinous Carcinoma of Rectum.—Wassermann doubtful positive. The patient denied lues, and the past history was not suggestive. Diagnosis was made from section of excised portion of tumor.

Lymphosarcoma of Tonsil.—Wassermann doubtful positive. This woman gave a history of one miscarriage, one child born dead at term, one child dead at 22 months (cause not known). She had two living children. Otherwise, there was nothing suggestive in the past history. Diagnosis was made from section of excised portion of tumor.

The cases on which Wassermann reactions have been done subsequent to the injection of Ehrlich's 606 are but fourteen in number. These cases are, however, of sufficient interest to report individually.

Case 1.—Oct. 25, 1910. Chancre of lip. Spirocheta pallida present. Wassermann positive.
Oct. 26. Intramuscular injection of 0.6 gm. 606.
Nov. 8. Roseola present.
Nov. 15. Intramuscular injection of 0.6 gm. 606.
Nov. 19. Spirocheta pallida present. Wassermann positive.
Dec. 7. Wassermann positive.
Dec. 21. Wassermann positive.
Feb. 1, 1911. Wassermann positive.

Case 2.—Primary lesion six years ago. Came in for stricture of urethra, one month duration.
July 1, 1911. Intravenous injection 606.
July 12. Wassermann negative.

Case 3.—Primary lesion four months previously, followed by secondaries.
Dec. 4, 1910. Intramuscular injection 0.5 gm. 606.
Jan. 4, 1911. Wassermann positive.

Case 4.—Secondaries in February, 1911. Then six months pregnant.
Mar. 8, 1911. Wassermann positive. Intramuscular injection of 606, followed by mixed treatment.
June 28. Wassermann negative. Baby said to be well.

Case 5.—Primary lesion April, 1910. Secondaries about one month later. From August up to the time of admission to hospital had numerous ulcerating lesions on various parts of body. Treated continuously, since primary lesion, with mercury, by inunction and injection, without effect.

Nov. 26, 1910.	Entered hospital. Condition: multiple gummata and serpiginous syphilides. Wassermann positive.
Nov. 27.	Intramuscular injection of 0.4 gm. 606.
Jan. 4, 1911.	Wassermann positive. Lesions healing rapidly.
Apr. 26.	Wassermann negative. Lesions healed.

Case 6.—Infant, age two months.

Oct. 16.	General eruption, snuffles, hoarseness. Large liver and spleen. Wassermann positive. 0.025 gm. 606 administered subcutaneously.
Oct. 18.	Wassermann positive.
Oct. 25.	Wassermann positive.
Nov. 2.	Wassermann positive. Condition much improved.

Case 7.—June 7, 1911. Fading roseola, and mucous patches. Wassermann positive.

June 10.	0.6 gm. 606 intravenously.
June 24.	0.6 gm. 606 intravenously.
June 26.	Wassermann positive.
July 29.	0.6 gm. 606 intravenously.
Aug. 30.	Wassermann positive.
Nov. 15.	Wassermann positive.
	Has had no symptoms since first injection.

Case 8.—Jan. —, 1910. Primary lesion, followed by secondaries. Treated with mercury.

Sept. 15, 1911.	Wassermann positive. Has no symptoms.
Sept. 22.	0.5 gm. 606 intravenously.
Oct. 17.	Wassermann positive.
Oct. 27.	0.5 gm. 606 intravenously.
Nov. 28.	Wassermann doubtful positive.
Dec. 7.	0.6 gm. 606 intravenously.
Dec. 27.	Wassermann doubtful positive.
Feb. 15, 1912.	Wassermann negative.

Case 9.—Mar. —, 1909. Primary lesion. Treated ten months with mercury.

Mar. 29, 1911.	Wassermann positive. No symptoms at present.
Jan. 2, 1912.	Wassermann negative.
	Had two doses of 606 in the interval.

Case 10.—Primary lesion six months ago; 606 four months ago.

June 21, 1910.	No symptoms at present. Wassermann negative.

Case 11.—Primary lesion six years ago.

Dec. 7, 1910.	Orchitis, dactylitis. Wassermann positive.
Dec. 8.	0.9 gm. 606 intramuscularly.
Dec. 18.	Wassermann positive.
Mar. 2, 1911.	Wassermann positive.

	Mar. 15.	Wassermann positive.
	Sept. 20.	Wassermann doubtful positive.
Case 12.—	Sept. —, 1910.	Primary. Treated with mercury for seven months.
	July —, 1911.	606.
	Oct. 18.	Wassermann negative.
Case 13.—	Sept. —, 1910.	Primary lesion.
	Mar. —, 1911.	Mixed treatment.
	Mar. 26.	606 subcutaneously.
	May 27.	606 intravenously.
	June 28.	Wassermann negative.
Case 14.—	Denies syphilis.	Had yaws thirty-five years ago.
	Oct. 16, 1911.	Wassermann positive. Palpable tumor of liver. Diagnosis: Gumma of liver.
		0.5 gm. 606 intravenously, followed by mixed treatment.
	Jan. 23, 1912.	Tumor not felt. General condition greatly improved. Wassermann positive.

Four of the above cases, on whom no test was done before injection, gave a negative reaction after injection. Four, on whom the reaction was positive before injection, gave a negative reaction after an interval of three and one-half to nine months. Six cases gave a positive or doubtful positive reaction after an interval of seventeen days to nine months.

It is conceded by most observers that in working with a hemolytic system it is advisable to use known amounts of both amboceptor and cells. Wassermann's original method for the diagnosis of syphilis makes use of 1 c.c. of a 5 per cent suspension of sheep corpuscles with just twice the amount of amboceptor necessary to hemolyze these cells. Since the discovery of the existence of an anti-sheep amboceptor in some human sera it has been a question whether this additional amount of amboceptor might not be sufficient to produce hemolysis in conjunction with a small residue of complement not fixed in the first stage of the reaction. If this should occur, negative results would thus be obtained in syphilitic cases.

The recognition of this possibility has given rise to several modifications of the Wassermann reaction. The best known of these is probably that of Noguchi. He claimed[1] that: "Wassermann's original method is subject to an error arising from the presence in human serum of a varying amount of natural amboceptor capable of being reactivated by guinea-pig's complement." He found experimentally

[1]Noguchi. Jour. Exp. Med., 1909, xi, 392.

that four units of anti-sheep amboceptor prevent entirely the detection of one unit of syphilitic antibody. The modification of the Wassermann technique devised by him has, among other advantages, that of avoiding this danger by the use of a hemolytic system consisting of human blood cells and the serum of a rabbit immunized against them.

Several other methods of obviating this source of error have been suggested which still make use of sheep corpuscles as in the original Wassermann method. That of Bauer[2], in which each serum is tested for anti-sheep amboceptor and artificial immune serum added only to those which show an insufficient amount of natural anti-sheep amboceptor to give complete hemolysis with the amounts of sheep cells and complement used in the Wassermann reaction, will of course be efficient in those cases in which the human serum contains just enough amboceptor to give complete hemolysis. As, however, some sera contain many times this amount of natural amboceptor, with these the source of error still remains.

Jacobaeus[3] proposed absorbing the sheep amboceptor from human serum by incubating the serum, after the addition of sheep cells, at 37° for one-half hour; then centrifuging off the cells and proceeding with the Wassermann reaction according to the regular technique. In a series of 257 cases he obtained about 10 per cent more positives by this method than without absorption. He claims that complementoid is also removed by this method, thus giving it the advantage of a modification introduced by Wechselmann,[4] in which complementoid is removed by digesting the inactivated human serum with barium sulphate. Bauer[5] had previously tried the same procedure which Jacobaeus employed, but discarded it on account of its making the serum anti-hemolytic. He claimed that this property was much increased by the addition of liver extract, thus causing negative sera to give positive reactions.

S. Mintz[6], using this method in a series of 38 cases, obtained 30 positive reactions against 25 without absorption. The sera which reacted positively were all syphilitic.

This method of amboceptor absorption has been tried by the author

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[2]Bauer. Sem. méd., 1908, xxviii, 429.

[3]Jacobaeus. Ztschr. f. Immunitätsforsch., Orig., 1911, viii, 615.

[4]Wechselmann. Ztschr. f. Immunitätsforsch., Orig., 1909, iii, 525.

[5]Bauer. Berlin klin. Woch., 1908, xiv, 834.

[6]S. Mintz. Ztschr. f. Immunitätsforsch., Orig., 1911, ix, 29.

on 305 sera, regardless of the amount of natural anti-sheep amboceptor present. The result of the Wassermann reaction on each of these sera has been compared with the result of the reaction on the same serum with the natural anti-sheep amboceptor present.

Only 53 of the 305 sera contained one or more units of natural anti-sheep amboceptor in 0.2 c.c., *i.e.*, sufficient to hemolyze completely 1 c.c. of a 5 per cent suspension of sheep corpuscles in the presence of one unit of complement. In 70 sera there was no trace of anti-sheep amboceptor in 0.2 c.c. In the remaining 182, anti-sheep amboceptor was present, but in a quantity not sufficient to produce complete hemolysis.

The technique employed to remove the anti-sheep amboceptor from the human serum was as follows: To 0.5 c.c. of the patient's serum, after inactivation, was added 2 c.c. of 0.85 per cent salt solution and 0.1 c.c. of sheep cells. After shaking, the mixture was incubated at 37° for 20 minutes. The cells were then centrifuged off and the supernatant fluid used in the Wassermann reaction, 0.5 c.c. of the diluted serum being used in the tube with antigen and 1 c.c. in the control tube without antigen, the amount of antigen, complement, etc., being correspondingly reduced to one-half the usual quantity. The remainder of the fluid was used to test the completeness of the amboceptor absorption. In about one-quarter of the cases a sufficient amount of the patient's serum was used to test for remaining sheep amboceptor in a full c.c.

In all but three of the 305 sera, removal of the sheep amboceptor was complete. 0.2 c.c. of each of these showed a faint trace of hemolytic power for sheep cells still present. One additional serum showed very slight hemolytic power remaining in a full c.c., but none was demonstrated in 0.2 c.c.

So far as the removal of anti-sheep amboceptor from human serum is concerned, we may conclude that the method is practically always efficient. The objection to the method is that inhibitory bodies are in some way produced by this process (''Sachs-Friedberger phenomenon'') which considerably slow hemolysis, there sometimes being a trace of inhibition at the end of an hour in the control tubes and in negative sera. The difference in reaction between positive and negative sera is, however, clear-cut, the inhibitory action not being sufficiently marked to render the method impracticable as a means of avoiding any error which may be due to the presence of natural anti-sheep amboceptor.

Rossi[7] claims that incubation at 0° for 20 minutes is as efficient in absorbing the amboceptor as incubation at 37°, while by this method the inhibitory phenomenon does not appear. His method is to add 0.5 c.c. of sheep red blood corpuscles to 1.5 c.c. of the patient's serum, both having been previously cooled to 0°. The mixture is kept at this temperature for 20 to 30 minutes, then rapidly centrifuged, and the serum drawn off with a pipetté. In a series of 60 syphilitic cases he obtained 50 positives by the Wassermann reaction and 56 positives after absorption.

This method of absorption, as well as that at 37°, was tried on 195 of the above 305 sera. In a portion of these the Rossi technique was followed in detail. With the remainder, the technique was the same, except that serum and cells were mixed in the proportions used for absorption at 37°. This method was found equally efficient in absorbing the anti-sheep amboceptor. So far, however, as the avoidance of inhibitory action is concerned, it was unsuccessful, there being little if any difference in this regard between the two methods.

The results of the Wassermann reactions on the 305 sera with natural anti-sheep amboceptor still present and on the same sera after the amboceptor has been completely removed, are shown in the following table. The results of the Wassermann reaction following absorption at 0° did not differ in any particular from those following absorption at 37°.

Wassermann reaction	Positive	Doubtful positive	Doubtful negative	Negative
On sera with natural anti-sheep amboceptor present	103	16	2	184
On sera after removal of anti-sheep amboceptor	104	18	1	182

As is shown by the above table, there was a difference in results in but three of the 305 cases. The cases in which the reaction differed were as follows:

1. Diagnosis: Pyorrhea Alveolaris. No history of syphilis obtainable. The serum contained five units of anti-sheep amboceptor. Wassermann reaction: with anti-sheep amboceptor present, negative; after removal of anti-sheep amboceptor, doubtful positive.

2. Diagnosis: Syphilitic Laryngitis. Primary lesion fourteen years ago. Treatment previous to Wassermann reaction not known. The serum contained over three units of anti-sheep amboceptor, the exact amount not being de-

[7]Rossi. Ztschr. f. Immunitätsforsch., Orig., 1911, x, 321.

termined. Wassermann reaction: with anti-sheep amboceptor present, doubtful negative; after removal of anti-sheep amboceptor, positive.

3. Diagnosis: Tabes Dorsalis. No history of syphilis. Serum contained two units of anti-sheep amboceptor. Wassermann reaction: with anti-sheep amboceptor present, negative; after removal of anti-sheep amboceptor, doubtful positive.

It will be noted that in none of these cases was the difference in results a difference between a frank negative and a frank positive reaction.

In reporting results of the Wassermann reaction it is unnecessary to give in detail the technique employed, the method being so well known. There are, however, certain points which it seems to us should be mentioned. In the reactions reported here, the guinea-pig serum was always titrated and care taken never to use as much as two units, as will frequently be done if 0.1 c.c. is used. The antigen used in nearly all of the 305 cases was an acetone-insoluble fraction of beef heart, prepared as recommended by Noguchi[8]. The quantity used in the reaction was 0.01 c.c. This antigen was not hemolytic or anti-complementary in four times this amount, and had high antigenic properties. Titrated against four positive sera it gave with one complete inhibition in one-tenth the quantity used; with a second, complete inhibition in one-thirteenth, and with the other two complete inhibition in one-twentieth the quantity used.

As the results obtained by the amboceptor absorption methods vary considerably from those reported by Wechselmann and other observers, experiments were done to test the effect of the introduction of artificial amboceptor on the Wassermann reaction done with the above antigen.

Three sera were selected which were frankly positive with this antigen and Wassermann reactions done on each of these after the addition of five, ten, and fifteen units of artificial anti-sheep amboceptor. Reactions were also done on the same sera without the addition of artificial amboceptor and after the addition of five, ten, and fifteen units, using an amount of antigen which by titration with each serum contained two units of antigen for that serum. Each serum contained natural anti-sheep amboceptor, but in an amount less than one unit. This was not removed.

Serum 1.—Contained two units of syphilitic antibody. Wassermann reactions were positive with both strong and weak antigens. With the addition of

[8]Noguchi. Serum Diagnosis of Syphilis, 2d Edition.

five, ten and fifteen units of amboceptor, the reactions were doubtful or negative with both antigens, but the inhibition was greater with the stronger antigen.

Serum 2.—Contained eighteen units of syphilitic antibody. Wassermann reactions were frankly positive with the strong antigen, even with the addition of five, ten and fifteen units of amboceptor. With the weak antigen the serum gave a frankly positive reaction when artificial amboceptor was not added. With five units of amboceptor the reaction was doubtful; with ten and fifteen units, negative.

Serum 3.—Contained more than twenty-five units of syphilitic antibody. Wassermann reactions were frankly positive with both antigens, with fifteen units of anti-sheep amboceptor present.

CONCLUSIONS.

From the above cases and experiments, we conclude that it is possible for anti-sheep amboceptor in human serum to affect the Wassermann reaction, but that when an antigen of high titer is used this is possible only with sera of very low antibody content and several units of anti-sheep amboceptor. As these two conditions, in our experience, occur but rarely in practical work, we feel that, when a strong antigen is used, the importance of anti-sheep amboceptor in human serum as a cause of negative reactions in syphilitic cases is not great. As a routine procedure, the absorption of amboceptor is unnecessary. Its removal is advisable, however, from sera which give a negative or doubtful reaction, and which contain a large amount of anti-sheep amboceptor. This is easily accomplished by digestion with sheep cells. It is immaterial whether this is done at 0°, 37°, or room temperature.

COMPLEMENT IN HUMAN SERUM.*

C. H. Bailey, M.D.

C. C. Bass[1], in "A New Conception of Immunity," draws interesting conclusions from several statements, the experimental proof of which, unfortunately, he does not give. The broad application made of the principles stated, and the fact that, as stated, they are contrary to generally accepted ideas, encouraged the following brief experiments.

The statements referred to are as follows:

"Human complement capable of acting with human amboceptor to produce lysis . . . is destroyed by any temperature above normal body temperature. . . . A temperature of 40° C. (104° F.) destroys complement in human serum in from fifteen to thirty minutes and prevents lysis regardless of the amount of amboceptor employed."

"Freshly drawn human blood contains little or no complement capable of acting with human amboceptors. . . . No human specific complement develops at ordinary fever heat, 38° to 40° C. (101° to 104° F.), such as obtains locally and often generally in most inflammations."

"In the event that human complement has developed in a blood . . . it again disappears in from thirty to seventy-two hours. . . ."

EXPERIMENTS.

Experiment 1.—Five human serums, all of which had been on the clot for over seventy-two hours, were withdrawn, and tested for complement, before and after heating at 40.5° C. for thirty minutes.

To tubes, each of which contained 0.25 c.c. of a 2 per cent suspension of washed calf-cells and 0.25 c.c. (two units) of a 1-600 dilution of inactivated serum of a rabbit immunized to calf-cells, was added the human serum in the following amounts: 0.8, 0.4, 0.2, 0.05 and 0 c.c. A similar series was set up for

*Reprinted from the Journal of the American Medical Assn., 1911, lvii, Dec. 23.

[1]Bass, C. C.: Jour. A. M. A., Nov. 4, 1911, p. 1534.

each patient, with like amounts of serum, heated for thirty minutes at 40.5° C.

Patients A and B gave no hemolysis with either heated or unheated serum. Patient C gave complete hemolysis with 0.4 c.c. of serum, both heated and unheated. Patient D gave slight hemolysis with 0.8 c.c. of serum, heated and unheated. Patient E gave complete hemolysis with 0.8 c.c. of serum, and almost complete with 0.4 c.c., heated and unheated.

This experiment shows that complement may still be present in serums over seventy-two hours old, capable of completing a lytic system, and that such complement is not destroyed by thirty minutes' heating at 40.5° C. It would be remarkable if human complement capable of acting with a calf-immune system were not destroyed by thirty minutes' heating at 40° C., while "human complement capable of acting with human amboceptor to produce lysis" were thus destroyed. As, however, the article in question specifies the latter, the following experiment was performed to determine this point, as well as the truth of the statement that "freshly drawn human blood contains little or no complement capable of acting with human amboceptors."

Experiment 2.—A normal individual was bled directly into an equal amount of citrate solution in a water bath at 41° C. The cells were immediately centrifuged off, and varying amounts of the supernatant fluid added to a 5 per cent suspension of sheep cells, previously sensitized with human serum containing natural sheep amboceptor, and set up in the bath at 41° C. Incubation was at 41° to 43° C., for thirty minutes.

The remainder of the supernatant fluid was retained in the bath at 41° to 43° C., for thirty minutes, and then added to sensitized cells.

To tubes, each of which contained 0.25 c.c. of a 5 per cent suspension of washed sheep-cells and 0.125 c.c. (= 2 units) of human serum containing anti-sheep amboceptor, inactivated one-half hour at 56° C., was added the citrated plasma, prepared as above described, in the amounts given in Table I.

TABLE I.—COMPLEMENT CONTENT OF PLASMA OF NORMAL INDIVIDUAL.

	Result	
	With fresh plasma	With plasma heated 30 min. at 41° C.
Citrated plasma	Hemolysis	Hemolysis
0.6	Complete	Complete
0.3	Complete	Complete
0.15	Almost complete	Almost complete
0.075	Partial	Partial
0.0375	Slight	Slight
.0	None	None

Experiment 3.—Two patients, one with a temperature of 104° F., the other 104.5° F., were bled directly into equal amounts of citrate solution, in a water bath, at a temperature of 42° C. The cells were at once centrifuged off, and the plasma, thus diluted, added to sheep cells sensitized with human serum, as in Experiment 2, already in the bath at 42° C. Incubation was at 42° C., for thirty minutes.

TABLE II.—COMPLEMENT CONTENT OF PLASMA OF FEBRILE PATIENT.

Fresh citrated plasma from febrile patients	Result	
	Patient A	Patient B
	Hemolysis	Hemolysis
0.6	Complete	Complete
0.3	Complete	Complete
0.15	Complete	Almost complete
0.075	Partial	Partial
0.0375	Slight	Slight
.0	None	None

From the above experiments the following conclusions seem justified:

1. Human complement capable of acting with human amboceptor to produce hemolysis is not destroyed by a temperature of 41° C. for thirty minutes.

2. Freshly drawn human blood contains a considerable amount of complement capable of acting with human hemolytic amboceptor, and such complement is not destroyed by heating at 41° C. for thirty minutes.

3. The blood of a patient with a temperature of 40° C. contains complement capable of acting with human hemolytic amboceptor.

4. Complement does not necessarily disappear from human serums in seventy-two hours after withdrawal from the body.

EFFECTS ON TITRATIONS OF INEQUALITY OF SENSITIZATION OF CORPUSCLES.*

C. H. Bailey, M. D.

It is well known that corpuscles will absorb many times the amount of specific amboceptor necessary to produce hemolysis, and that such absorption takes place with considerable rapidity. We believe, however, that the importance of these facts in quantitative serum work, as titrations for lytic or complementary power, has not yet been recognized. The author has frequently noted that duplicate titrations of the same serum gave results that differed beyond reasonable limits of experimental error, and that the reading obtained from a serum titration could be influenced considerably by slight variations in the method of activating the corpuscles. These results appear to be due to the fact that when corpuscles are added to an amboceptor dilution they are not at once evenly distributed through the fluid, and thus, owing to the rapidity with which amboceptor is absorbed, an opportunity is afforded for certain corpuscles to take up more of the amboceptor than others, and unequal sensitization results. If this be the case, it will be seen that slight variations in the method of activating the corpuscles will produce variations in the distribution of amboceptor and consequently in the hemolysis obtained.

The following complement titrations illustrate the difference in results which are produced by different methods of activating the corpuscles:

Experiment I.—The tubes contained a 1-10 dilution of guinea-pig serum, in the amounts shown below, with sufficient salt solution to make the final total in each tube 1.25 c.c.

A. To each tube was added 0.25 c.c. of a 1-2000 dilution of amboceptor (= 1 unit) and 0.25 c.c. of a 5 per cent suspension of sheep corpuscles, separately.

B. Into 4 c.c. of a 1-2000 dilution of amboceptor were dropped, rapidly, but one drop at a time, 4 c.c. of a 5 per cent suspension of sheep corpuscles, the

*Reprinted from the Journal of Experimental Medicine, May, 1912, xv.

receptacle being shaken in the meantime. Of this mixture, 0.5 c.c. was added to each tube.

C. Like B, except that the process was reversed, the amboceptor being added to the corpuscles; 0.5 c.c. was added to each tube.

D. Into 4 c.c. of the 5 per cent suspension of corpuscles was quickly poured 4 c.c. of the amboceptor dilution, and the mixture immediately shaken; 0.5 c.c. was added to each tube.

The results, after incubation for one hour at 37°, were as follows:

Guinea-pig serum, 1-10 dilution...	.375	.3	.25	.225	.2	.175	.15	.125	.1	.075	.05	.025	0
A	C	C	C	C	C	AC	AC	AC	P	P	VS	VS	0
B	VS	VS	VS	VS	VS	VS	VS	VS	VS	VS	VS	VS	0
C	C	C	C	C	C	C	C	AC	AC	P	VS	VS	0
D	C	C	C	C	C	C	C	C	AC	P	VS	VS	0

Note.—In this and the following experiments C = complete hemolysis, AC = almost complete hemolysis, P = partial hemolysis, S = slight hemolysis, VS = very slight hemolysis, and 0 = no hemolysis.

It is to be noted that although the amounts of amboceptor, complement, and cells, and the dilutions, are the same in corresponding tubes of the different series, the results differ considerably. B and C are methods of activation which would hardly be used in practical work. They serve, however, to illustrate the extreme variation in results which may be obtained. It is evident in B that practically the entire amount of amboceptor was taken up by the first few corpuscles added, consequently they were the only ones to hemolyze; and there is little difference in the amount of hemolysis resulting between the highest and lowest tubes. C, on the other hand, as might be expected, approaches closely to an even sensitization. A and D are methods which are often used in complement titration, as for the Wassermann reaction, A probably more commonly than D. When a cell suspension is added to small tubes containing amboceptor, as was done in A, it may be noted that frequently a few cells will be distributed through the liquid, the greater bulk, however, remaining on the surface, along the side of the tube, or sinking to the bottom, thus affording an opportunity for a few cells to absorb more than their share of the amboceptor, the amount depending, of course, on the interval elapsing between the introduction of the cells and the shaking of the tube.

It is evident that to cause this inequality, the absorption of amboceptor by corpuscles must be very rapid. The following experiment was done to obtain some idea of the amount of amboceptor absorbed by corpuscles in a given time:

Experiment II.—Amboceptor absorption in A, with a 5 per cent suspension of sheep corpuscles, and corresponding immune rabbit serum; in B, with a 2 per cent suspension of sheep corpuscles and corresponding immune rabbit serum. The amboceptor was so diluted as to contain 1, 2 and 5 units of amboceptor for equal quantities of the 5 per cent and 2 per cent suspension of cells.

a. To 4 c.c. of diluted amboceptor, in a centrifuge tube, was added 4 c.c. of corpuscle suspension, and the mixture immediately centrifuged, at a speed of about 2,000 revolutions.

b. Like a, except centrifuged after an interval of 2 minutes.

c. Like a, except centrifuged after an interval of 5 minutes.

From each was taken 0.5, 1, 1.5 and 2 c.c. of the diluted amboceptor, 0.5 being approximately equivalent to 0.25 of the original amboceptor dilution, and to each of these amounts was added 0.2 c.c. of a 1-10 dilution of guinea-pig serum (= 1 unit by previous titration) and 0.25 c.c. of corpuscle suspension, each tube being immediately shaken after the addition of the corpuscles. For the control tubes the amboceptor was diluted with equal parts of salt solution. After incubation at 37° for one hour the results were as follows:

Amboceptor	0.5	1	1.5	2
A. Five per cent suspension of sheep corpuscle				
1 unit of amboceptor				
Control	C	C	C	C
a	0	0	0	VS
b	0	0	0	0
c	0	0	0	0
2 units of amboceptor				
a	VS	P	P	AC
b	0	VS	P	AC
c	0	VS	VS	S
5 units of amboceptor				
a	C	C	C	C
b	P	C	C	C
c	S	P	AC	AC
B. Two per cent suspension of sheep corpuscle				
1 unit of amboceptor				
Control	C	C	C	C
a	0	S	P	AC
b	0	0	0	VS
c	0	0	0	0
2 units of amboceptor				
a	P	C	C	C
b	0	P	AC	AC
c	0	VS	S	P
5 units of amboceptor				
a	C	C	C	C
b	C	C	C	C
c	P	C	C	C

It appears from the above experiment that a 5 per cent suspension of corpuscles almost completely absorbed one unit of amboceptor in the brief time necessary to centrifuge off the cells. About three-quarters of the 2 units of amboceptor appear to have been absorbed in the same length of time, while of the 5 unit amboceptor 1 unit at least remained. Absorption by the 2 per cent suspension was apparently not so rapid. About three-quarters of the 1 unit and about one-half of the 2 unit amboceptor were absorbed in the time taken to centrifuge, while of the 5 units at least 1 unit remained even after absorption for 2 minutes plus the time taken to centrifuge.

Definite conclusions, however, cannot be drawn from such an experiment as to the exact amount of amboceptor absorbed or the relative speed of absorption by the 5 per cent and 2 per cent suspension of corpuscles, as it is not known to what extent the failure of hemolysis may be due to inhibitory bodies produced by the addition of corpuscles for the purpose of amboceptor absorption ("Sachs-Friedberger phenomenon"). Such inhibitory bodies probably do not greatly influence the results obtained in this experiment, but if present it is natural to suppose that they are in larger amount after absorption with a 5 per cent suspension of corpuscles than after absorption with a 2 per cent suspension of corpuscles.

Though the cells were centrifuged from the fluid as quickly as possible, an interval of between 1 and 2 minutes probably elapsed before their complete removal. The following experiment was done to show that an appreciable amount of amboceptor is absorbed in a considerably shorter time than this.

Experiment III.—A 1-10 dilution of guinea-pig serum was used in the amounts given below, with sufficient salt solution in each tube to make the final total 1.25 c.c. A 5 per cent and a 2 per cent suspension of sheep corpuscles, and a 5 per cent and a 2 per cent suspension of calf corpuscles were used with one unit of the respective amboceptor; 0.5 c.c. of the corpuscle suspension, activated as follows, was added in series:

A. 3 c.c. of the corpuscle suspension was poured into 3 c.c. of amboceptor dilution, and quickly shaken.

B. 0.5 c.c. of the corpuscle suspension was poured into 3 c.c. of amboceptor dilution, shaken, and, 15 seconds later, 2.5 c.c. of the corpuscle suspension added.

C. Like B, except with 50 seconds interval.

D. Like B, except with 1 minute interval.

E. Like B, except with 2 minutes interval.

After incubation at 37° for one hour the results were as follows:

Guinea-pig serum diluted 1-10....	.5	.4	.3	.25	.2	.125	.062	.031	.015
5% suspension of sheep corpuscles									
A.....	C	C	C	C	C	C	AC	S	VS
B.....	C	C	C	C	AC	AC	P	S	0
C.....	C	C	C	AC	AC	P	S	VS	0
D.....	AC	AC	AC	P	P	P	S	VS	0
2% suspension of sheep corpuscles									
A.....	C	C	C	C	C	AC	VS	0	0
B.....	C	C	C	C	C	P	VS	0	0
C.....	C	C	C	C	AC	P	VS	0	0
D.....	C	C	AC	AC	P	S	VS	0	0
E.....	AC	P	P	S	S	VS	VS	0	0
5% suspension of calf corpuscles									
A.....	C	C	C	C	C	C	AC	VS	0
B.....	C	C	C	C	C	AC	P	VS	0
C.....	C	C	AC	AC	AC	AC	P	VS	0
D.....	C	C	AC	AC	AC	P	VS	VS	0
E.....	AC	AC	P	P	S	S	VS	VS	0
2% suspension of calf corpuscles									
A.....	C	C	C	C	AC	P	VS	VS	0
B.....	C	C	C	C	AC	P	VS	0	0
C.....	C	C	C	AC	P	VS	VS	0	0
D.....	C	C	AC	P	VS	VS	VS	0	0
E.....	AC	AC	AC	P	VS	VS	VS	0	0

It will be seen by the foregoing experiment that a considerable effect is produced on the degree of hemolysis obtained by a contact of even 15 seconds of a portion of the corpuscles with the amboceptor before the introduction of the remainder. The effect is somewhat less marked with the 2 per cent than with the 5 per cent suspension. This, we believe, is what takes place in greater or less degree in any serum titration when an even distribution of the corpuscles throughout the amboceptor dilution is not at once obtained. It is probably impossible to obtain an absolutely even sensitization. The nearest approach to this is produced by quickly pouring the diluted immune serum into an equal volume of the corpuscle suspension, which is shaken during and for a short period after the mixing (Exp. I, D). If the corpuscles are poured slowly into the amboceptor or introduced in separate lots, as with a 5 or 10 c.c. pipette, unequal sensitization will result. This is illustrated in an extreme degree by the method of sensitization used in Exp. I, B, that of dropping the cells into the amboceptor. It is to be noted that the reading is not only higher with an even sensitization, but is also sharper; that is, the

change from complete hemolysis to entire lack of hemolysis, instead of being gradual, is quite sudden (Exp. I, A and D).

The importance in practical work of obtaining as uniform a distribution of amboceptor as possible is obvious, as well as the necessity in any comparative titration of using suspensions of activated cells in the various titrations which are exactly alike as to the distribution of amboceptor. The importance of complement titration for the Wassermann or other complement absorption tests is rightly emphasized. If, however, the same method of sensitization is not used in this titration as is used in the final stage of the Wassermann reaction, the titration is of little value as an index of the activity of the serum. It is a common practice to introduce amboceptor and corpuscles separately in a complement titration (Exp. I, A), while corpuscles previously sensitized in bulk are used in the Wassermann reaction. By the latter method it is much easier to approximate an even sensitization (Exp. I, D). Thus the reading obtained in a complement titration in which amboceptor and corpuscles are added separately would lead one to use an excess of complement in performing the reaction.

In an amboceptor titration, the immune rabbit serum and corpuscles are usually introduced separately—to employ separate lots of corpuscles previously activated with each dilution would be an exceedingly tedious task—yet if one accepts the highest dilution with which complete hemolysis is obtained as the titer of the serum, and with this dilution titrates the same complement as was used in the amboceptor titration, hemolysis will be obtained, provided more evenly sensitized corpuscles are here used, with considerably less complement than was used in the amboceptor titration. This may be illustrated by reference to Experiment III. It is stated in this experiment that one unit of amboceptor was used. Both the anti-sheep and the anti-calf amboceptor were titrated before this experiment, using 0.25 c.c. of the same dilution of guinea-pig serum as was used in the experiment. The anti-sheep serum was found to give complete hemolysis of the 5 per cent corpuscles in a dilution of 1 to 800, incomplete in 1 to 1,000; the anti-calf gave complete hemolysis of the 5 per cent corpuscles in a dilution of 1 to 400, incomplete in 1 to 600. In the experiment, however, in which they were used in dilutions of 1 to 800 and 1 to 400, respectively, we find that they both give complete hemolysis when previously sensitized corpuscles are used (Exp. III, A) with one-half the amount of complement (0.125 c.c.) with which they

were titrated. A retitration of the anti-sheep serum with 0.25 c.c. of the complement dilution, using 0.5 c.c. of corpuscle suspension previously activated for each dilution by rapidly mixing 5 c.c. of a 5 per cent suspension of corpuscles with an equal amount of the proper amboceptor dilution, gave complete hemolysis in a dilution of 1 to 1,200. We must conclude, then, that an amboceptor as well as a complement titration is influenced by the evenness of the sensitization of the corpuscles.

CONCLUSIONS.

The absorption of amboceptor by corpuscles is rapid, a considerable amount being absorbed in as short a period as 15 seconds. In the sensitization of corpuscles, the amount of amboceptor absorbed by the different corpuscles is not uniform, the inequality depending on the time taken in obtaining an even distribution of the corpuscles through the diluted immune serum. Amboceptor absorption is apparently influenced by the concentration of the corpuscles, being more rapid with a 5 per cent than with a 2 per cent suspension, and thus the stronger concentration is more susceptible to inequality of sensitization from variations in the method of activation.

In experimental work it is of importance to obtain as equal a sensitization as possible. It is essential that in comparative titrations the same method of activation be employed in the several titrations, and where possible, it is advisable that all the cells to be used be sensitized together in bulk.

The results obtained in titrating hemolytic sera and complement depend to a considerable extent on the evenness of sensitization of the corpuscles.

THE DETERMINATION OF COPPER—A MODIFICATION OF THE IODIDE METHOD.*

E. C. KENDALL, Ph.D.

For the determination of copper the most important methods are the electrolytic, the iodide, and the cyanide. As the determination by means of the electrolytic method requires a considerable amount of time and apparatus, the only methods for the rapid estimation of copper are the iodide and the cyanide.

Upon an examination of the two volumetric methods mentioned it is apparent that in respect to the amount of time and attention required for a determination the cyanide has a great advantage over the iodide method. However, in respect to the accuracy of the results obtained the iodide method is conceded to be by far the more accurate of the two. As every consideration would be in favor of the iodide method if it could be modified in such a way as to make it as rapid and easy of manipulation as the cyanide method, an attempt was made to make such a modification.

In the determination of copper by the iodide method the copper may be originally present as copper, copper oxide, or sulfide. The first step is to obtain the copper in solution. Practically the only way to do this is to dissolve it in nitric acid. The solution of the copper with nitric acid produces nitrous acid in the solution, and it is the removal of this which causes the delay in the estimation of the copper. As the method is described in the literature, the nitrous acid is destroyed with bromine, the excess of bromine being removed by boiling; or the nitrous acid is removed by evaporating to dryness.

The modification of the iodide method as described in this paper consists in the destruction of the nitrous acid without boiling. This is accomplished by the addition of a small amount of sodium hypochlorite. The addition of sodium hypochlorite to a nitric acid solution produces hypochlorous acid. The interaction of hypochlorous acid

*From the Journal of the American Chemical Society, vol. xxxiii, No. 12, December, 1911.

and nitrous acid results in the oxidation of the nitrous acid and the formation of hydrochloric acid, and the reaction between hypochlorous and hydrochloric acid results in the destruction of the hypochlorous acid and the formation of free chlorine and water. As the solution of sodium hypochlorite contains small amounts of chlorides, hydrochloric acid will always be present when the solution is acidified, thus insuring the destruction of the hypochlorous acid and the formation of free chlorine. We thus see that the effect of adding sodium hypochlorite to the solution is the complete destruction of the nitrous acid and the formation of free chlorine.

To remove the free chlorine in solution some compound must be added which will take up the chlorine, but will not affect subsequent operations. Such a compound is found in phenol. Under the conditions of the determination, phenol will add chlorine directly to the benzene ring, but is not affected by iodine or any of the other compounds in the solution. Chlorophenol not being ionized removes all traces of free chlorine.

This modification of the method greatly reduces the time and attention required for a determination, and, in addition, the copper solution is prepared in such a way that iodine can be liberated by copper alone.

In the determination, the copper, copper oxide, or sulfide is dissolved in nitric acid. After the addition of the sodium hypochlorite and phenol, which requires but a moment, the solution is made slightly alkaline with sodium hydroxide, and is then made acid with acetic acid, when the solution is ready for titration. Potassium iodide and starch are added, and the titration is made to the disappearance of the starch iodide color. There is never any fear of the blue color "flashing back," and the solutions will remain colorless indefinitely after the titration. As the ionization constant for acetic acid is too low to allow nitrates to liberate iodine, the amount of nitric acid in solution is immaterial. Even 20 c.c. of concentrated nitric acid will not affect the titration. However, too great an acidity is to be avoided, as nitrophenol will be formed. The presence of nitrophenol prevents the determination of copper, but there is no danger of its formation even in the presence of a large amount of acid if the solution is neutralized soon after the addition of the phenol. If a large amount of nitric acid is used to dissolve the copper, it should therefore be partly neutralized before addition of the phenol.

As chlorine easily oxidizes phenol to compounds which prevent the

determination of copper, it is essential that all of the phenol be added quickly to the solution. Under these conditions the chlorine adds directly to the benzene ring, but if the phenol is added drop by drop the chlorine will oxidize it, producing colored compounds in solution.

In order to add the phenol quickly enough to the solution it may be poured in from a beaker, or, a more convenient way, from a pipette from which the tip has been removed so that the delivery is from an opening which is of the same bore as the rest of the tube. By forcing the phenol out of such a pipette with the breath, the entire volume is added very quickly and at the same time the phenol is well mixed with the contents of the flask.

After addition of the phenol the chlorine gas which is in the flask above the liquid is removed by blowing it out with the breath, and the sides of the flask are washed with a jet of water from a wash bottle. There should be no odor of chlorine just before the solution is made alkaline.

It should be remembered that the end point of the titration is not pure white. Cuprous iodide has a cream color, and when a large amount of copper is present the cuprous iodide gives a decided tint to the solution. When the end point is nearly reached a drop of the thiosulfate is allowed to fall into the center of the flask. If a change of color occurs the solution is given a slight rotary motion and after the solution is again quiet another drop of the thiosulfate is added. This "spot test" is easily recognized and gives a very accurate end point.

The speed of reaction of the copper with potassium iodide varies with the volume. In a small volume the action is rapid and all of the iodine is liberated at once, but in a large volume an appreciable time may be required for all of the copper to react. This is especially noticeable when a small amount of copper is present. A high concentration of potassium iodide greatly assists the liberation of the iodine. Accurate results cannot be obtained unless at least 3 grams of potassium iodide are added, irrespective of the amount of copper present, up to 500 mg. of copper.

The solutions required are:[1]

A. The Sodium Hypochlorite solution is made by boiling together a mixture of 112 grams of calcium hypochlorite and 100 grams of anhydrous sodium carbonate in 1,200 c.c. of water. After the calcium

[1]The weights given here are for calcium hypochlorite having 35 per cent or more available chlorine.

is precipitated as carbonate, the solution is filtered and its strength found as follows: 5 c.c. of the hypochlorite solution are added to 100 c.c. of water containing 5 c.c. of 30 per cent potassium iodide solution, and a few c.c. of dilute hydrochloric acid are added. The liberated iodine is titrated with 0.1 *N* sodium thiosulfate. The volume of the solution is now adjusted so that 5 c.c. of the hypochlorite solution are equivalent to 30 c.c. of 0.1 *N* sodium thiosulfate.

B. Phenol—A 5 per cent colorless solution of phenol.

C. Sodium Hydroxide—A 20 per cent solution.

D. Acetic Acid, 50 per cent.

E. Potassium Iodide—A convenient way to use this is to prepare a solution which contains 30 grams per 100 c.c. of solution. Then 10 c.c. will contain 3 grams, which is the amount needed for a determination.

F. Sodium Thiosulfate—For the accurate titration of the liberated iodine two solutions are used. One strong solution, 1 c.c. of which equals 6 mg. of copper, and a weak solution, 1 c.c. of which equals 1 mg. of copper. The strong solution is run in until the iodine liberated by the copper gives a light straw color to the solution. Starch is then added and the titration is finished with the weak solution.

As a thiosulfate solution loses strength, it should be restandardized from time to time. A convenient way to do this is as follows: A solution of sodium thiosulfate, approximately 0.1 *N*, is made by dissolving 24 grams of the crystallized salt per liter of water. After the solution has stood at least 24 hours it is standardized against copper by the method described below. Pure electrolytic copper which has been cleaned with emery paper should be used. After dissolving 150 to 200 mg. of the copper in 6 to 8 c.c. of 50 per cent nitric acid the solution is treated as described below and the thiosulfate is then standardized with this known weight of copper. The most convenient means of restandardizing the thiosulfate is to use a solution of acid potassium iodate. Acid potassium iodate has the formula $KIO_3.HIO_3$, so that a normal solution has one-twelfth the molecular weight in grams per liter. A 0.1 *N* solution is prepared by dissolving 3.249 grams of the salt in 1 liter of water, and it is standardized against a known strength of thiosulfate as follows: Add 10 c.c. of the acid iodate solution to 150 c.c. of water containing 0.5 to 1 c.c. of hydrochloric acid. Upon the addition of potassium iodide, iodine will be liberated according to the equation

$$HIO_3 + 5HI = + 3H_2O + 6I.$$

Starch is added and the titration is made to a colorless solution. From this titration the weight of copper to which 20 c.c. of this solution are equivalent is accurately determined. A 20 c.c. pipette is passed through a one-hole stopper and is allowed to remain in the acid iodate bottle. The end of the pipette is closed with a small rubber stopper. The exact copper equivalent of a thiosulfate solution is now easily found by titrating 20 c.c. of the acid iodate solution whose copper equivalent is known with the thiosulfate as described above. The acid iodate remains constant indefinitely.

G. Starch for Indicator—The best preparation for this purpose is a 0.5 per cent solution of Kahlbaum's soluble starch. This is prepared as ordinary starch, but gives a perfectly clear solution which is very sensitive with iodine. If ordinary starch must be used it should be free from all cloudiness.

DETAILED DESCRIPTION OF THE METHOD.

If the copper to be determined is present as metallic copper, 200-300 mg. are placed in a 300 c.c. flask and dissolved in 5-10 c.c. of 50 per cent nitric acid.

If the copper is present as cuprous oxide, it is filtered on a Gooch crucible through asbestos. The cuprous oxide is then dissolved through the Gooch crucible with 10-15 c.c. of 30 per cent nitric acid into a 300 c.c. Erlenmeyer flask.

If the copper is in the form of sulfide, it is filtered on a Gooch crucible which has a layer of asbestos one-eighth inch in thickness. The crucible is then placed in a small beaker of 50 c.c. capacity, and 10 c.c. of 50 per cent nitric acid are added. The beaker is placed on a hot plate, and the nitric acid allowed to boil until all the black sulfide has gone into solution. The crucible is then washed off, and the solution transferred to a 300 c.c. Erlenmeyer flask. The presence of the asbestos in the solution does not interfere with the titration of the copper.

If the copper to be determined is already in solution as sulfate, chloride, or other salt, sufficient solution is taken to give 100 to 300 mg. of copper.

Having obtained the copper in solution, preferably in a 300 c.c. Erlenmeyer flask, the volume being between 50 and 60 c.c., the acidity is adjusted to equal 4 to 5 c.c. of concentrated nitric acid. A greater volume of acidity is to be avoided. The temperature should not be above 25°. Five c.c. of the hypochlorite solution are now added to the copper solution, which is well mixed with a rotary motion. As

soon as the color of the copper solution changes from a clear blue to a greenish tint, sufficient hypochlorite has been added. Another indication of a sufficient amount of hypochlorite is the liberation of chlorine. For weights of copper up to 200 mg., 2-3 c.c. of the hypochlorite are sufficient. For larger amounts of copper more hypochlorite may be needed, but 5 c.c. will be sufficient for any amount of copper which would be determined by this method. The reactions between the hypochlorous and nitrous acid require an appreciable time and the best results are obtained by allowing the solution to stand about 2 minutes before the addition of the phenol. This, however, is not essential. Ten c.c. of the phenol solution are now added as quickly as possible, by blowing the solution from a pipette from which the tip has been removed.

The chlorine gas which remains in the flask above the liquid is removed by blowing into the flask and the sides are washed down with a jet of water. If the solution is allowed to stand at this point, nitrophenol will slowly form. Sodium hydroxide is therefore added until a very slight precipitate is obtained. The solution is now made acid with acetic acid; only a few drops should be required to dissolve the precipitate. Ten c.c. of the potassium iodide are added and the titration made with the standardized thiosulfate. If great accuracy is required the titration is finished with a weak solution of thiosulfate.

The following are some results obtained by the method described above. The milligrams found and the error are calculated only to a point which is within the degree of accuracy of the apparatus used.

DETERMINATION OF COPPER

Copper taken Mg.	Copper found Mg.	Error Mg.	Error Per cent.
20.00	20.01	+0.01	+0.05
20.00	19.99	—0.01	—0.05
20.00	20.00	0.00	0.00
30.00	29.99	—0.01	—0.03
30.00	30.00	0.00	0.00
40.00	39.98	—0.02	—0.05
40.00	39.96	—0.04	—0.10
60.00	60.01	+0.01	+0.02
60.00	60.01	+0.01	+0.02
80.00	80.12	+0.12	+0.15
80.00	80.03	+0.03	+0.04
80.00	79.98	—0.02	—0.02
80.00	79.98	—0.02	—0.02

Copper taken Mg.	Copper found Mg.	Error Mg.	Error Per cent.
100.00	100.00	0.00	0.00
100.00	99.99	—0.01	—0.01
120.00	119.95	—0.05	—0.04
140.00	140.00	0.00	0.00
160.00	160.00	0.00	0.00
160.00	160.00	0.00	0.00
180.00	180.00	0.00	0.00
180.00	180.00	0.00	0.00
200.00	200.00	0.00	0.00
200.00	199.9	—0.1	—0.05
203.2	203.2	0.00	0.00
220.2	220.1	—0.1	—0.05
240.0	240.0	0.00	0.00
240.0	240.2	+0.2	+0.08
261.6	261.6	0.00	0.00
280.0	280.0	0.00	0.00
280.0	280.3	+0.3	+0.10
300.0	300.1	+0.1	+0.03
320.0	319.9	—0.1	—0.03
320.0	319.9	—0.1	—0.03
340.0	340.0	0.00	0.00

Note.—The sum of the + and — errors very nearly equals zero.

For the opportunity of carrying out this work I wish to thank Dr. N. B. Foster and for assistance with the analytical work Mr. A. W. Thomas.

THE DETERMINATION OF IODINE IN THE PRESENCE OF OTHER HALOGENS AND ORGANIC MATTER.

E. C. KENDALL, Ph.D.

During an investigation of the iodine bearing compound of the thyroid gland a method for the determination of small amounts of iodine in organic combination was worked out in this laboratory. As the reactions involved in this method are quantitative when larger amounts of iodine are present, conditions have been established which furnish a rapid and accurate method for the determination of iodine in the presence of bromides, chlorides, and organic matter.

For the determination of iodine when present as an iodide or in the uncombined condition, Andrews[1] has proposed a volumetric method in which the iodine is oxidized to iodine chloride by means of iodic acid. The titration by Andrews' method is done in the presence of a large excess of hydrochloric acid, the end point being the disappearance of iodine. As the oxidation of the iodine is limited by the acid to the formation of ICl, one molecular weight of iodine reacts with but two molecular weights of chlorine.

$$KI + Cl_2 = KCl + ICl.$$

If the oxidation of the iodine is carried out under conditions which permit the quantitative formation of iodic acid, one molecular weight of iodine requires six molecular weights of chlorine, as shown by the equation

$$KI + 3Cl_2 + 3H_2O = KCl + HIO_3 + 5HCl.$$

Dupre's method for the determination of iodine is based upon this reaction, weak chlorine water being used for the oxidation. In a recent paper by Hunter[2] a method is proposed in which the iodine is oxidized to iodic acid with sodium hypochlorite, and after the removal of the excess of hypochlorite the weight of iodic acid is determined by the further addition of potassium iodide. Iodic acid and potassium iodide react as follows:

$$HIO_3 + 5HI = 3I_2 + 3H_2O.$$

[1]Jour. Amer. Chem. Soc., 25, 756.

[2]Jour. Biological Chem. (1910), vol. vii, p. 321.

The liberated iodine is titrated with sodium thiosulfate, the weight of iodine titrated being six times the weight originally present.

The method described in this paper is based upon the oxidation of the iodine to iodic acid and the subsequent determination of the amount of iodic acid formed.

DETERMINATION OF IODINE WHEN PRESENT AS AN IODIDE OR FREE IODINE.

For the determination of iodine when present as an iodide or in the uncombined condition, it is necessary to have a solution of the iodine which is free from organic matter or oxidizing agents, such as arsenic, antimony, copper, nitrites, and all compounds which liberate iodine from potassium iodide. If bromine or any compounds which interfere are present, the method is modified as described below.

The solution containing the iodine is placed in a 500 c.c. flask, the total volume of the solution being between 200 and 250 c.c. The solution[3] should have a neutral or very slightly alkaline reaction. Five c.c. of phosphoric acid (85 per cent diluted with an equal volume of water) are added to the solution. A solution of sodium hypochlorite[4] is now added, while the solution is shaken with a rotary motion. If an iodide is present iodine will be liberated, but the further addition of hypochlorite will oxidize this to iodic acid. The hypochlorite should be added slowly, and care should be taken to avoid adding more than is necessary to give a colorless solution. The solution is allowed to stand 2 to 3 minutes after becoming colorless and then 10 c.c. of a colorless 5 per cent solution of phenol are added. The phenol combines with the free chlorine in solution, forming chlorophenol. This compound being unionized, removes all traces of chlorine from the sphere of reaction. When a solution of phenol is slowly added to a solution containing free chlorine the phenol is partially oxidized, producing colored compounds, but if the phenol is

[3]To prevent loss of iodine at this point the solution must be cold, and when more than 100 mg. of iodine are present the solution in the flask should be covered with a few c.c. of benzol.

[4]A convenient means of preparing this reagent in a proper concentration is to add 112 gm. of calcium hypochloride whose available chlorine is approximately 35% to 1,200 c.c. of water. Stir the mixture, to break up any lumps, and heat to boiling. One hundred grams of anhydrous sodium carbonate are now added, and the solution boiled 10 to 12 minutes. After cooling, the precipitate of calcium carbonate is filtered off and the solution of sodium hypochlorite is kept in a black-colored bottle.

all added at once the chlorine adds to the benzol ring without oxidation of the phenol. For this reason the phenol is added to the flask as rapidly as possible. This is accomplished by forcing the phenol with the breath from a 10 c.c. pipet from which the tip has been removed, so that the delivery is from an opening which is the same bore as the rest of the tube. A few drops of phenolphthalein are added and the solution is made slightly alkaline with 30 per cent sodium hydroxide which is free from nitrites. The solution is now made acid with 10 c.c. of 50 per cent phosphoric acid. Upon the addition of potassium iodide the iodic acid in solution will liberate iodine which is titrated with sodium thiosulfate. The amount of potassium iodide added should be sufficient to leave an excess after reacting with the iodic acid. The weight of potassium iodide required is, roughly, eight times the weight of the iodine originally present.

The effect of the presence of small amounts of oxidizing compounds is considerably lessened if the solution is made alkaline and is then acidified again. This step is necessary to secure satisfactory results.

The most satisfactory method for standardizing the sodium thiosulfate which is used to titrate the iodine liberated by the iodic acid is as follows: Ten grams of freshly resublimed iodine are weighed out in a weighing bottle. This is placed in a large Erlenmeyer flask containing 5 grams of sodium hydroxide dissolved in 400-500 c.c. of water. The cover is removed from the bottle and the iodine is dissolved in the alkali. The solution is now diluted to two liters; 1 c.c. will contain 5 mgms. of iodine. For standardizing, the iodine solution is measured into a flask, the volume is made between 200-250 c.c. and then the acid and hypochlorite are added as described above. The number of c.c. of thiosulfate divided into the weight of iodine measured into the flasks is the standard of the sodium thiosulfate for the iodine originally present.[5] If more than 100 mgms. of iodine are present there is danger of loss of iodine by volatilization during the titration. A satisfactory means of preventing this is to add a few c.c. of benzol to the flask. This will float on the surface and prevent loss of iodine. Care should be taken to finish the titration with starch and to shake the solution vigorously when near the end point.

By the method described above the following results were obtained.

[5]The "original iodine" equivalent of sodium thiosulfate in this titration is one-sixth the amount found by the titration. Hence, if $N/_{10}$ thiosulfate is used, the standard will be approximately 2.115 mg. of original iodine per c.c.

The iodine was present in the form of potassium iodide, which was prepared by dissolving a known weight of pure iodine in potassium hydroxide and reducing with metallic aluminium.

Iodine taken	Iodine found	Error Mg.	Error Per cent
5.079	5.077	—.002	.04
5.079	5.040	—.039	.76
7.618	7.619	.001	.01
7.618	7.630	.012	.15
10.158	10.136	—.022	.22
10.158	10.150	—.008	.08
12.698	12.717	.019	.15
12.698	12.707	.009	.07
15.237	15.166	—.071	.46
15.237	15.213	—.024	.16
17.776	17.742	—.034	.19
17.776	17.756	—.020	.11
20.316	20.337	.021	.10
20.316	20.394	.078	.38
22.855	22.820	—.035	.15
22.855	22.871	.016	.07
25.395	25.434	.039	.15
25.395	25.340	—.055	.22
30.06	30.13	.07	.23
30.06	30.13	.07	.23
40.08	40.10	.02	.05
40.08	40.10	.02	.05
50.10	50.17	.07	.13
50.10	50.30	.20	.39
60.12	60.07	—.05	.09
60.12	60.26	.14	.23
70.14	70.18	.04	.05
70.14	70.27	.14	.20
80.16	80.19	.03	.04
80.16	80.11	—.05	.06
90.18	90.20	.02	.02
90.18	90.39	.21	.23
100.20	100.12	—.08	.08
100.20	99.74[6]	—.46[6]	.46
125.45	125.65	.20	.16
125.45	125.45	.00	.00
150.57	150.62	.05	.03
150.57	150.54	—.03	.02
150.57	150.54	—.03	.02
175.66	175.80	.14	.08

[6]Iodine was lost, as no benzol covering was used in this determination.

Iodine taken	Iodine found	Error Mg.	Error Per cent
200.71	175.59	—.07	.04
200.71	200.80	.09	.04
200.71	200.76	.05	.02
225.85	225.53	—.32	.15
225.85	226.00	.15	.07
250.95	250.91	—.04	.02
250.95	250.91	—.04	.02
276.04	275.88	—.16	.06
276.04	276.12	.08	.03
301.15	301.45	.31	.10
301.14	301.05	—.09	.03
326.23	326.02	—.21	.07
326.23	326.02	—.21	.07
361.33	350.78	—.55	.15

DETERMINATION OF IODINE IN THE PRESENCE OF BROMIDES AND CHLORIDES.

When bromine or a bromide is present in a solution to which hypochlorite is added there is no oxidation of the bromine similar to the oxidation of iodine. Furthermore, the presence of the bromine does not interfere with the oxidation of the iodine. Hydrobromic acid, when present in large amount, will reduce iodic acid, but all hydrobromic acid may be removed by the addition of sufficient sodium hypochlorite.

The method for the determination of iodine in the presence of bromine is as follows: The iodine (in the form of iodide or uncombined) is dissolved in 200-250 c.c. of water having a neutral or slightly alkaline solution. Five c.c. of phosphoric acid (85 per cent diluted with an equal volume of water) and 10 c.c. of benzol are added. Sufficient sodium hypochlorite is now added to liberate all of the bromine and oxidize the iodine. Iodine will be liberated at first, but this will be further oxidized to iodic acid. It is imperative that all the bromine be liberated. A small amount of powdered pumice is added and the solution boiled. The benzol reacts with the hypobromite and hypochlorite, forming brom- and chlor-benzol. The free bromine boils out of solution. A precipitate of brom-benzol may form, but this does not affect subsequent operations. After a few minutes' boiling, all traces of bromine are removed. The solution is now removed from the flame and cooled. Under these conditions, it is not necessary to neutralize and acidify, but the potassium iodide is added

directly to the cold solution. A cover of benzol should be used for weights of iodine over 100 mg.

The following results show that there is no appreciable interference of the bromine in the determination of iodine by this method:

50	mgms.	iodine	and	no	potassium	bromide	required,	26.38	c.c.	sod.	thiosulfate
50	"	"	"	0.1 g.	"	"	"	26.38	"	"	"
50	"	"	"	0.5 g.	"	"	"	26.30	"	"	"
50	"	"	"	1.0 g.	"	"	"	26.30	"	"	"
100	"	"	"	no	"	"	"	20.50	"	"	"
100	"	"	"	0.1 g.	"	"	"	20.48	"	"	"
100	"	"	"	0.5 g.	"	"	"	20.50	"	"	"
100	"	"	"	1.0 g.	"	"	"	20.48	"	"	"
250	"	"	"	no	"	"	"	46.10	"	"	"
250	"	"	"	0.1 g.	"	"	"	46.00	"	"	"
250	"	"	"	0.5 g.	"	"	"	46.15	"	"	"
250	"	"	"	1.0 g.	"	"	"	45.88	"	"	"

In standardizing the sodium thiosulfate to be used when bromine is present, more satisfactory results are obtained by establishing the standard with a known weight of the iodine solution as prepared above, under the conditions which are described for the determination of iodine in the presence of bromine.

The presence of chlorides has no effect upon the determination of iodine by this method and there is no need of boiling the solution. The following results bear on this point:

50	mgms.	iodine	and	no	sodium	chloride	required,	26.50	c.c.	thiosulfate
50	"	"	"	1.0 g.	"	"	"	26.50	"	"
50	"	"	"	5.0 g.	"	"	"	26.51	"	"
50	"	"	"	10.0 g.	"	"	"	26.50	"	"

These results were obtained by the method outlined under the heading, Determination of Iodine When Present as Iodide or Free Iodine.

When iodine is to be determined in the presence of organic matter, or nitrites, copper, iron, lead, mercury, and silver, it is necessary to remove these interfering substances and prepare the iodine as an iodide for the determination.

DETERMINATION OF IODINE IN THE PRESENCE OF ORGANIC MATTER AND INTERFERING ELEMENTS.

The most satisfactory means of removing the above-mentioned substances is by a fusion which will destroy organic matter, retain the iodine as an iodide, and by forming insoluble compounds, remove interfering elements.

Many fusion mixtures have been proposed for the destruction of

organic matter, but when tried did not give entirely satisfactory results for the peculiar needs of this method. The determination of iodine in the presence of organic material, as worked out in this laboratory, is as follows:

DETAILED DESCRIPTION OF METHOD.

The Fusion.*—The fusion takes place in two stages: first, the oxidation of the organic matter; second, the reduction of all oxidizing compounds. The destruction of the organic matter is accomplished by fusion with a mixture of sodium potassium carbonate and potassium chlorate. The mixture is made by grinding together and passing through a 20-mesh sieve:

138 grams of potassium carbonate (anhydrous)
106 " " sodium carbonate (anhydrous)
100 " " potassium chlorate (anhydrous)

One gram or less of the organic material is placed in the bottom of a 2⅜-inch nickel crucible. This is dissolved in a few c.c. of 30 per cent sodium hydroxide. The water is evaporated by placing the crucible in a hot air oven at a temperature of 150-200°. Fifteen grams of the fusion mixture are now added, the cover is placed on the crucible and the crucible is heated strongly by a large Bunsen or, preferably, a Méker burner. The fusion begins before the crucible is red hot and proceeds quietly and quickly. The crucible should be supported on a triangle and surrounded by a collar of sheet asbestos. The one used in this laboratory was 3 inches in diameter, 3 inches deep, and one-quarter inch thick. The top of the collar was notched by cutting away small rectangles about 1 inch long by one-half inch deep. When a cover of sheet asbestos was placed over the collar these notches permitted the hot gases from the burner to escape after passing around the crucible. By heating in this manner the sides as well as the bottom of the crucible were heated to a red heat. After three minutes' heating with an 8-inch Méker burner, in a collar, as above described, all but a trace of the chlorate is destroyed, and the melt has ceased to liberate bubbles of oxygen. If the carbon content of the

*Since sending this article for publication this method of fusion has been found unreliable under certain conditions. In the Journal of the American Chemical Society another method of fusion will be described which can be relied upon under all conditions. With the exception of the fusion, the method is not changed.

added material is low, or if insufficient heat is applied, the fusion may require a longer time.

During the destruction of the organic matter, the nitrogen contained in the protein material is in part oxidized to a nitrate. The action of heat on the nitrate formed results in the production of nitrites in the fusion mass. If the fusion mass containing nitrites should be dissolved in water and acidified, the nitrous acid would oxidize the iodide, liberating iodine, which would be carried out of solution by the escaping carbon dioxide. It is therefore necessary to destroy the nitrites and the trace of chlorate before solution of the fusion mass. The most satisfactory reagent for this purpose was found to be metallic zinc, in granular form. The zinc must be free from arsenic. The size of the granules is unimportant, satisfactory results being obtained with both zinc dust and 20-mesh granules, but in order to facilitate subsequent operations, 20-mesh zinc granules were found most convenient.

After the first heating for 3 minutes, during which time the organic matter is destroyed and the chlorate decomposed, 2 grams of the zinc are added to the crucible without removing from the flame, the cover is replaced, and the crucible heated in the flame for an additional 2 minutes. The zinc reduces all traces of chlorate and nitrites. The crucible is removed from the flame and the melt is allowed to cool on the sides of the crucible. When cold, the crucible is nearly filled with water and placed on a hot plate. After the fusion mass has dissolved (this requires about 10 to 15 minutes), the solution is transferred to a beaker, and is then filtered to remove the excess of zinc and zinc oxide, and any interfering element, as lead, mercury, silver, copper, etc.

In order to avoid thorough washing of the filter paper, the solution may be placed in a 250 c.c. flask, diluted to the mark, well mixed and then filtered into a 200 c.c. flask, washing out the 200 c.c. flask with the first 15-20 c.c. of the filtrate. The most rapid filtration we have found is by using a fluted filter, Carl Schleicher & Schüll, No. 597. If no interfering element is present, filtering the solution may be omitted, if care is taken to decant the solution from the beaker into the 250 c.c. flask, leaving the granules of zinc in the beaker. The beaker and zinc are washed repeatedly with small amounts of water. The presence of zinc oxide or carbonate is not objectionable. The solution in the 250 c.c. flask is now adjusted to the mark of graduation and well mixed with a rotary motion. The zinc oxide settles

rapidly, and after a few minutes' standing, a solution comparatively free from the precipitate may be decanted from the flask. The 200 c.c. flask is washed out twice with a few cubic centimeters of this solution and is then filled to the mark by decanting the solution from the 250 c.c. flask. The iodine is now present as an iodide, free from interfering compounds. A few drops of methyl orange are added, and the solution is made very slightly acid[7] with 50 per cent sulfuric acid. The acidity should not be less than 2-3 c.c. (or more than 5 c.c.) of the 50 per cent sulfuric acid. Sodium hypochlorite is added until the iodine is oxidized to iodic acid. Phenol is added and the solution made slightly alkaline to phenolphthalein. Five c.c. of 50 per cent phosphoric acid are now added and the iodic acid is determined by addition of potassium iodide and titration with sodium thiosulfate. If bromine is present the method is varied by acidifying with 20 c.c. of syrupy 85 per cent phosphoric acid and adding sufficient hypochlorite to liberate all the bromine and oxidize the iodine. Ten c.c. of benzol are added, and the solution is boiled (with powdered pumice) until all bromine is expelled. Under these conditions it is not necessary to neutralize, but the potassium iodide is added to the cold solution. A cover of benzol should be used for more than 100 mg. of iodine.

Sulfuric acid is used to acidify the solution so that the neutralization will not form a salt with an ion in common with the acid used to acidify in the final titration. The presence of sodium phosphate greatly reduces the acidity from the phosphoric acid, but sodium sulfate has no such action. The methyl orange being destroyed by the hypochlorite does not interfere with subsequent operations. If the color from the methyl orange is destroyed upon acidification, or if there is any trace of iodine liberated, the fusion was not carried out properly. In this case, either heating to a higher temperature or for a longer time will be necessary to secure accurate results.

When the organic matter and fusion mixture are wet with water and evaporated to dryness before fusion, the action of water makes too intimate contact between the organic matter and the chlorate, and a violent explosion may result. By evaporating to dryness with sodium hydroxide and adding the fusion mixture to this, there is no danger of explosion. If some particles of carbon remain unoxidized,

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[7]The presence of a few c.c. of chloroform materially decreases the foaming from the escaping carbon dioxide.

it does not affect the result, as all the iodine will be retained as iodide.

THE DETERMINATION OF SMALL QUANTITIES OF IODINE IN ORGANIC COMBINATION.

In 1910, Hunter[8] published a method for the determination of small quantities of iodine in organic combination, which may be briefly stated as follows: The compound is fused with a mixture of sodium potassium carbonate and potassium nitrate. This fusion destroys the organic matter and fixes the iodine as an iodide. The fusion mass is dissolved in water and sodium hypochlorite is added. Upon acidification with phosphoric acid, the sodium hypochlorite oxidizes the nitrous acid formed during the fusion to nitric acid and the iodide is oxidized to iodic acid. The excess of free chlorine formed from the sodium hypochlorite is removed by boiling. After all free chlorine has been removed and the solution is cold, potassium iodide is added. The iodic acid in solution, which was obtained from the iodine originally in organic combination, reacts with the added potassium iodide, each weight of iodine present as iodic acid liberating six times its weight of iodine. The weight of iodine finally titrated, therefore, is equivalent to six times the amount of iodine originally present.

During the past 18 months I have had occasion to make determinations of iodine in thyroid preparations, and the method outlined above was used for this work. As the results obtained were not entirely satisfactory, a careful study of the chemical reactions involved was undertaken, with the hope of finding a reliable method for the determination of iodine. The one serious and unavoidable objection to Hunter's method is the fact that simply boiling a solution containing a large amount of sodium hypochlorite, to which phosphoric acid has been added, will not always completely remove compounds which liberate iodine from potassium iodide.[9]

Foerster and Jorre[10] have pointed out that when a solution of

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[8]Hunter: Jour. Bio. Chem., 1910, vii, 321.

[9]In this connection, Hunter says: "The reagent that gives most frequent trouble is the hypochloric solution. It must be reasonably fresh. If this condition be fulfilled, the commercial product often gives excellent results. Sometimes, however, it is impossible to get a commercial solution that does not give values too high."

[10]J. Pr. Chem., 1899 [2], *59*, 53.

sodium hypochlorite is acidified, oxy-chlorine compounds, among which may be chloric acid, are produced. The amount of chloric acid formed appears to depend upon the rate of acidification, the temperature, and concentration of the acid used.

In Hunter's method it is necessary to add the phosphoric acid to the solution containing a comparatively large amount of sodium hypochlorite. This results in the formation of oxychlorine acids, which, for a series of determinations, may vary between wide limits, depending upon the conditions of fusion. When the solution is boiled for 15 to 20 minutes, these acids are broken down and expelled, but rarely are they entirely removed from solution. As the amount of oxidizing compounds formed in the solution is uncertain, and as a variable amount may be removed by boiling, no constant correction can be applied to the results. For large amounts of iodine this correction is inappreciable, but when only a few one-hundredths of a milligram are present it is a serious objection. The chemical properties of chloric acid are closely analogous to those of iodic acid, and while some compounds will reduce one more easily and completely than the other, no single compound was found which could be satisfactorily used in a quantitative method to destroy chloric and leave the iodic acid unchanged. Another objection, though less serious, is the presence of a large amount of nitrate in the solution of the fusion mass. Nitrates in acid solution liberate iodine from potassium iodide. This reaction is slow in a solution slightly acid with phosphoric acid, but the titration of the iodic acid has to be made immediately after the addition of the potassium iodide as iodine is slowly liberated by the nitrates. Although many attempts were made to modify Hunter's method by removing all oxidizing compounds except iodic acid, no satisfactory modification was found.

Further investigation, however, has resulted in the following method for the determination of iodine, which has proved reliable* in this laboratory.

The method for small amounts of iodine is carried out as for larger amounts described above, with the following modifications: If the thyroid substance is in solution the equivalent of 1 gram of organic substance is placed in the 2⅜-inch nickel crucible and the water is

*Since sending this article for publication this method of fusion has been found unreliable under certain conditions. In the Journal of the American Chemical Society another method of fusion will be described which can be relied upon under all conditions. With the exception of the fusion, the method is not changed.

evaporated by placing the crucible in a hot-air oven at 150-200°. The 15 grams of fusion mixture are now added and the fusion carried out as described.

If the substance is in powder form, 1.2 grams are intimately mixed with 15 grams of the fusion mixture, and no water is added, but the fusion is carried out under the same conditions of heating and reduction with 20-mesh zinc granules. The zinc granules are separated from the fusion mass by filtering or decanting as described above. To the solution of the fusion mass 20 c.c. of syrupy 85 per cent phosphoric acid are added by allowing the acid to run from the pipet directly into the flask. There should be no liberation of iodine at this point. A few drops of bromine are added to the flask and the solution is shaken with a rotary motion until the bromine imparts a distinct yellow color. The solution is now boiled for 7-8 minutes. This will expel all but a trace of bromine. To the boiling solution, which should be colorless, 15-20 drops of 5 per cent sodium salicylate[11] are added. The salicylic acid produced in solution will remove all traces of bromine, but will not affect the iodic acid. After boiling 1 to 2 minutes after the addition of the salicylate, the solution is removed from the flame, cooled, potassium iodide is added, and the liberated iodine titrated[12] with thiosulfate.[13] For small amounts of

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[11]This is best prepared by dissolving pure salicylic acid in sodium hydroxide.

[12]Hunter recommends a clear 0.5 per cent solution of arrowroot starch for an indicator in finishing the titration. A 0.5 per cent. solution of Kahlbaum's soluble starch also furnishes a sensitive indicator for this work.

[13]The most convenient strength of sodium thiosulfate for amounts of iodine ranging from 0.5 to 5.0 gm. is $N/_{200}$. This is not a stable solution, and must be frequently restandardized. A convenient method is to prepare a solution of potassium acid iodate which is equivalent to a known weight of iodine. The strength of any sample of thiosulfate is readily found by titrating the iodine liberated by the acid iodate solution, which retains its strength indefinitely. The iodine equivalent of the potassium iodate is found as follows: Prepare an $N/_{10}$ solution of potassium acid iodate $KIO_3.HIO_3$ by dissolving 3.249 gm. of the salt in 1 liter of water. This solution, diluted 20 times, will be approximately $N/_{200}$. Dissolve a known weight of pure iodine (approximately 1 gm.) in 1 liter of water containing 1 to 2 gm. of sodium hydroxide. Dilute this ten times. 1 c.c. of this solution will contain 1 mg. of iodine. Measure 25 c.c. of this solution into a 500 c.c. flask, and dilute to 200 c.c.; add 5 c.c. of 50 per cent phosphoric acid and a few drops of bromine; boil out the bromine; add 15 to 20 drops of 5 per cent sodium salicylate, cool, add potassium iodide, and titrate the liberated iodine with approximately $N/_{200}$ thiosulfate. This will establish the relation between "original iodine" and the $N/_{200}$ thiosulfate, and from this

iodine, from 0 to 2-3 mg., this method will furnish very satisfactory results. It is imperative to have reagents of known purity. Potassium chlorate, as purchased in the open market, is often contaminated with a small amount of iodide. For our work Merck's reagent potassium chlorate gave no traces of iodine, but all of the ordinary grades of this salt contained a small amount. The zinc must be free from arsenic and antimony. A blank should be made, using some organic substance, free from iodine. If there is no iodine in any of the reagents used, there should result a perfect blank. As little as .005 mg. of original iodine can be detected by this method.

Some results showing that there is no loss of iodine by this method of fusion are as follows: 1.5 mg. of iodine, in the form of potassium iodide, was added to 15 gm. of the fusion mixture containing 1 gram of organic matter (Witte peptone). The fusions were carried out with a flame from an 8-inch Méker burner maintained as hot as possible, the crucible being surrounded with an asbestos collar, as described above. The length of time of heating, after addition of the zinc, was 2 minutes.

After the fusion the iodine was determined as above.

Time of heating	c.c. sodium thiosulfate used	Iodine found
3 minutes	12.71	1.50 mgm.
6 "	12.75	1.51 mgm.
9 "	12.70	1.50 mgm.
12 "	12.75	1.51 mgm.
15 "	12.72	1.50 mgm.

Some other results obtained by heating in the first stage of the fusion for 3 minutes and after addition of the zinc for the indicated times, are:

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the iodine equivalent of the potassium acid iodate can be found by adding a known volume of the acid iodate to 150 c.c. of water containing potassium iodide and 5 c.c. of 50 per cent phosphoric acid. When a small amount of iodic acid is in a solution which contains but a small amount of salts, the reaction with potassium iodide is retarded, and the end point of the titration with thiosulfate is uncertain. The addition of 5 to 10 gm. of sodium chloride to such a solution accelerates the liberation of iodine and makes the end point sharp and accurate.

Lgth. of heating after addition of zinc	c.c. sodium thiosulfate used	Iodine found
2 minutes	12.71	1.50 mgm.
4 "	12.80	1.51 mgm.
6 "	12.68	1.50 mgm.
8 "	12.80	1.51 mgm.
10 "	12.58	1.49 mgm.

These results show that there is no appreciable loss of iodine even when the length of time of the fusion is prolonged to 15 minutes.

In the determination of iodine by this method, where more than 3-4 mg. are present, the best results are obtained by acidifying with 20 c.c. of 85 per cent phosphoric and then oxidizing with sodium hypochlorite. After boiling for 10-12 minutes, the sodium salicylate is added, as described above for bromine.

When the hypochlorite is added to the acid solution the conditions are as unfavorable as possible for the formation of chloric acid, and with a comparatively large amount of iodine present, no appreciable error results from this source. However, the addition of an excess of hypochlorite should be avoided. Bromine and sodium hypochlorite should not be added together, as traces of iodine are liberated by such a solution even after prolonged boiling.

Hunter showed that iodine in organic combination, as well as in inorganic combination, is retained and converted into an iodide without loss by an alkaline fusion mixture. The original form of combination does not appear to affect the accuracy of the determination.

The following results were obtained by measuring a solution containing the indicated weights of iodine into nickel crucibles. The water was evaporated off, 15 gm. of the fusion mixture containing 1 gm. of Witte peptone were added, and the determination made as described above. The iodine solutions were made by dissolving pure iodine in sodium hydroxide, and diluting the solution to convenient strength for measuring the iodine.

Iodine taken Mg.	c.c. thiosulfate used	Iodine found Mg.	Error Mg.	Error Per cent
4.079	39.85	4.072	—.007	—0.17
4.079	39.65	4.052	—.027	—0.66
3.059	29.88	3.054	—.005	—0.16
3.059	29.85	3.05	—.009	—0.29
2.039	35.1	2.029	—.01	—0.49
2.039	35.1	2.029	—.01	—0.49

Iodine taken Mg.	c.c. thiosulfate used	Iodine found Mg.	Error Mg.	Error Per cent
1.02	17.5	1.011	—.009	—0.89
1.02	17.35	1.003	—.017	—1.66
0.714	24.40	0.717	+.003	+0.42
0.714	24.80	0.729	+.015	+2.10
0.510	17.27	0.510	0	0
0.510	17.85	0.525	+.015	+2.9
0.306	8.45	0.301	—.005	—1.6
0.306	8.51	0.303	—.003	—0.98
0.102	2.60	0.093	—.009	—0.88
0.102	2.60	0.093	—.009	—0.88
0.051	4.20	0.044	—.007	—13.00
0.051	3.90	0.041	—.010	—20.00
0.031	2.90	0.030	—.001	—3.2
0.031	2.80	0.029	—.002	—6.6
0	0	0	0	0

These results show that the method is accurate to within .02 mg. up to 3 or 4 mg. When used for a qualitative test for the presence of iodine, the method is especially valuable, as the use of bromine and salicylic acid assures a perfect blank in the absence of iodine.

Three samples of desiccated thyroid gland, kindly furnished by Parke, Davis & Company, when analyzed by this method, gave the following results:

	c.c. thiosulfate used	Iodine found per gram of substance Mg.
Sample 1	19.25	1.813
	19.28	1.816
Sample 2	19.77	1.862
	19.77	1.862
Sample 3	18.00	1.696
	18.25	1.719
	18.35	1.728

I wish to express my appreciation to Mr. A. W. Thomas for assistance during the course of this investigation.

SUMMARY.

This paper on the determination of iodine establishes the conditions for the determination of iodine—

First.—When present as a soluble iodide or in the uncombined form.

Second.—When present with bromine, bromides and chlorides.

Third.—When present with interfering compounds, as copper, silver, mercury, nitrites, etc.

Fourth.—When in organic combination.

Fifth.—When present in small amounts, special reference being given to the determination of the iodine content of the thyroid gland.

A NEW METHOD FOR THE DETERMINATION OF THE REDUCING SUGARS.*

E. C. KENDALL, Ph.D.

In the study of velocity of amylolytic action it became desirable to determine with the greatest possible accuracy the reducing sugars resulting from the digestion of starch. This led to the following study of some modifications of Fehling's reagent with a view to establishing the optimum medium and conditions for a gravimetric method of determining reducing power.

Benedict[1] and others have pointed out the fact that glucose is more readily destroyed with sodium hydroxide than with sodium carbonate, and that larger amounts of copper are reduced by the same weight of sugar if sodium carbonate is used in place of sodium hydroxide. A comparison of the results obtained with three of the more common methods and a copper solution where the sodium hydroxide is replaced with sodium carbonate follows.

50 mg. of glucose reduces according to:

Defren's method	Allihn's method	Munson and Walker's method	Na_2CO_3 solution replacing NaOH
89.8 mg. Cu.	98.2 mg. Cu.	102.0 mg. Cu.	147 mg.

We thus see that the use of sodium hydroxide gives only about two-thirds of the amount of copper reduced which may be obtained with the carbonate.

After a series of experiments with the various alkalies, it was found that potassium carbonate was the one best suited to furnish the alkalinity. As the reducing power of glucose decreases with increase in the volume of the solution, it is necessary to have the volume of the solutions containing the copper and alkali which are added to the

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[1]J. Biol. Chem., 3, 101 (1907); 5, 485 (1908).

sugar solution as small as possible. Potassium carbonate is better than sodium carbonate, being much more soluble and having a slight advantage in giving more copper reduced for the same weight of glucose. Some experiments showing the relation between these two carbonates are as follows:

50 mg. of glucose gave with

5 g. sodium carbonate, 129.8 mg. copper.
10 g. sodium carbonate, 133.3 mg. copper.
15 g. sodium carbonate, 133.3 mg. copper.

All conditions being the same with

12 g. potassium carbonate, 140.4 mg. copper.
14 g. potassium carbonate, 142.8 mg. copper.
16 g. potassium carbonate, 140.4 mg. copper.

Having found that potassium carbonate was best suited to furnish the alkalinity to the copper solution, it still remained to determine whether a better medium than Rochelle salts could be found to hold the copper in solution.

Some results according to Munson and Walker's method showed that unless the spontaneous reducing power of the alkaline tartrate solution is determined and allowed for, the results obtained will be considerably too high. Some results we obtained, using the method of Munson and Walker, but not allowing for the reducing power of the tartrate solution, are:

Sugar taken	Copper found	Sugar equivalent given in table
60	126.5	62.4
60	124.6	61.4
100	203.0	102.5
100	202.7	102.3

In their original article[2] Munson and Walker give a series of figures showing the reducing power of their alkaline tartrate solution from day to day during the course of the investigation. These results varied from 0 to 2.0 mg. of cuprous oxide. Apparently the reducing power of the alkaline tartrate solution varies with different samples of Rochelle salts and it is imperative to make some correction for all samples which we have examined.

The amount of copper reduced by the alkaline tartrate seems to be

[2]This Journal, 28, 663; 29, 541.

much greater when the reduction takes place in a bath of boiling water than it does when the solution is heated for a short time over the flame as in Munson and Walker's method. By heating for 20 minutes in a bath of boiling water 50 c.c. of the mixed Defren solution in a total volume of 150 c.c. may reduce as much as 7 mg. of copper. Provided the reduction was caused by an impurity in the tartrate it would be possible to free the solutions from such impurities by treating the alkaline tartrate with a copper solution, reduce by heating in a bath of boiling water, filter, and use the resulting solution which would have no reducing power of its own. This was tried and it was then found that when such a solution was heated again in the boiling water a second reduction, as large as the first, took place. If the cuprous oxide was then filtered off and the solution again heated, a third reduction took place. Since this showed that the reduction is due to the tartrate itself, and that a previous reduction is not capable of removing the source of error, it seemed imperative to find some medium other than Rochelle salts for keeping the copper in solution.

Many compounds have been proposed for this purpose, among which may be mentioned the bicarbonate solution of Soldaini[3] and the citrate solution proposed by Benedict.[4] In Benedict's volumetric method for the determination of sugar the disappearance of the blue color is taken as the end point of the titration and this method allows of an accurate determination of the sugar. Experiments were therefore made to see if Benedict's citrate solution could be adapted to a gravimetric method. It was then found that marked changes in the reducing powers of the sugars followed changes in concentration of the sodium carbonate and citrate.

Thus, using 50 mg. of glucose and 2 g. of copper sulfate in a volume of 150 c.c., the following weights of copper, expressed in milligrams, were obtained under the conditions as given below, when heated for 20 minutes in a bath of boiling water:

Sodium citrate Grams	Sodium carbonate 5 grams Mg.	Sodium carbonate 10 grams Mg.	Sodium carbonate 15 grams Mg.
5	116.5	134.1	144.2
10	100.0	117.3	127.1
15	89.3	107.9	122.0
20	65.3	101.8	117.8

[3]Gaz. chim. ital., 6, 322.

[4]J. Biol. Chem., 5, 485 (1908).

The change in the reducing power of sugar is explained only in part by assuming that the citrate solution dissolved the reduced cuprous oxide. This was shown by placing 150 mg. of Kahlbaum's cuprous oxide in each of four flasks and heating it under identical conditions of volume, time, and concentration of solution as in the above experiments. Oxidation of the cuprous oxide during the heating was prevented by displacing the air in the flask with illuminating gas and closing the flask with a two-hole stopper.

Determination of the copper content of 150 mg. of the cuprous oxide used showed on duplicate determination 125.6 and 126.1 mg. of copper, average 125.9 mg. All conditions being the same as above, the following weights of cuprous oxide were recovered after heating for 20 minutes:

Sodium citrate Grams	Sodium carbonate 5 grams Mg.	Sodium carbonate 15 grams Mg.
5	125.2	.....
10	123.3	122.4
15	120.2	122.4
20	118.3	122.1

As 20 g. of sodium citrate in the presence of 5 g. of sodium carbonate could dissolve but 7.6 mg. of cuprous oxide the low results obtained with the sugar must be due to a depression of the reducing power of the sugar by the citrate. The results of other experiments in which the weights of sugar varied showed that a citrate solution does not furnish a satisfactory solution in a gravimetric method.

As the spontaneous reduction of Rochelle salts and the depression and variations caused in the reducing power of sugar by sodium citrate are serious objections to these two salts, further work was done to find some other agent for holding the copper in solution.

Theoretically, any organic compound having a carboxyl and alcohol group is capable of holding the copper in solution in an alkaline mixture. Glycerol and mannite have also been suggested as possible agents, but they do not furnish a convenient solution with which to work. Lactic acid will hold the copper in solution, but the reducing power of sugar is but slight in such a solution.

Among a number of organic compounds which were tried, salicylic acid was found to be one which will furnish a medium for the reduction of sugar, but which has no reducing power of its own, and will not dissolve the cuprous oxide.

An alkaline salicylate solution replacing the alkaline tartrate showed no reduction of copper when heated in a bath of boiling water for 7 hours, and the following results show that there is no appreciable change in reducing power with small changes in concentration of the salicylic acid.

50 mg. glucose. 5 grams sodium carbonate in 150 c.c. volume.

3 grams salicylic acid, 125.7 mg. copper.
4 grams salicylic acid, 126.0 mg. copper.
5 grams salicylic acid, 125.7 mg. copper.
6 grams salicylic acid, 124.8 mg. copper.

Further experiments showed that with the other reducing sugars, maltose, lactose, and invert sugar, the alkaline salicylate solution furnishes a satisfactory medium for the reduction of the copper.

It now remained to determine what weights of copper, potassium carbonate and salicylic acid give the optimum conditions for the reduction of the copper.

In Munson and Walker's conditions, 1.858 grams of copper sulfate (crystalline) are used per determination, the largest weight of copper reduced being 435.3 mg. While larger amounts of copper give greater reducing powers to the sugars, it was decided to use two grams of copper sulfate (crystalline) per determination and limit the reduction to 450 mg. of copper.

The weights of potassium carbonate and salicylic acid which give the optimum conditions for maltose were determined and these weights were used for the determination of the reducing power of the other sugars.

The effect of varying amounts of potassium carbonate and salicylic acid is shown in the following table. The volume was 140 c.c. and 2 g. of copper sulfate were present:

Sugar maltose	Salicylic acid	Potassium carbonate		
Mg.	Grams	12 g.	14 g.	15 g.
100	4	151.7	153.8	154.9
100	5	151.0	157.1	155.0
100	6	149.1	154.6	154.8
100	7	135.7	150.4	154.9

These and other determinations showed that 15 grams of potassium carbonate, 5 grams of salicylic acid, and 2 grams of copper sulfate in a total volume of 140 c.c. give satisfactory conditions for the deter-

mination of maltose. The least volume of water which will conveniently dissolve the copper sulfate is 15 c.c. and the least volume for the 15 grams of potassium carbonate is 25 c.c. While it would be possible to make one solution of the three compounds, it was found that both copper and potassium salicylate are so slightly soluble that the volume of such a solution would be too great to give satisfactory results. The potassium carbonate and copper sulfate are therefore dissolved in water and added separately to the sugar solution while the salicylic acid is added in the dry condition.

In regard to the method of heating, the following experiments were carried out to determine whether or not any cuprous oxide was lost during the heating by surface oxidation:

150 mg. of Kahlbaum's cuprous oxide were placed in each of four flasks, 15 grams of potassium carbonate, 2 grams of copper sulfate and 5 grams of salicylic acid were added in a volume of 140 c.c.

The weight of copper in 150 mg. of the cuprous oxide used was found to be 125.9 mg. The weights of copper recovered from the four flasks after the treatment indicated below were as follows:

1. Solution boiled over free flame under conditions of Munson and Walker, 124.5 mg. copper recovered.

2. Solution heated 20 minutes in bath of boiling water, 126.3 mg. copper recovered.

3. Solution heated 20 minutes in bath of boiling water with surface covered with toluene, 125.7 mg. copper recovered.

4. Solution heated 20 minutes in bath of boiling water, air above solution being displaced with illuminating gas, 126.3 mg. copper recovered.

These results showed that there is no appreciable loss of cuprous oxide due to surface oxidation when the heating is continued for 20 minutes in the boiling water.

The two methods of heating which have been used for the reduction of copper with sugar are by heating over a free flame or in a bath of boiling water. In choosing between these two methods, ease of operation, time required, and accuracy of the results obtained were the factors considered.

The following results bear on this point:

The conditions of the solutions in each of the following sets were those found to be the optimum conditions for determining the reducing power of sugars. Volume 140 c.c., potassium carbonate 15 grams, salicylic acid 5 grams, copper sulfate 2 grams.

HEATING IN BATH OF BOILING WATER

Time, min.	Glucose, 50 mg. Mg. Cu.	Invert sugar, 50 mg. Mg. Cu.	Lactose, 100 mg. Mg. Cu.	Maltose, 100 mg. Mg. Cu.
10	132.7	142.4	123.6	124.8
15	145.1	152.5	144.5	144.8
20	149.8	154.7	154.4	153.1
25	150.1	157.4	161.1	158.5
30	155.0	162.9	165.4	163.3
40	157.1	164.9	171.6	167.9
50	159.9	166.3	176.2	172.2

HEATING OVER FLAME

Total time of heating, min.	Time of boiling, min.	Glucose, 50 mg. Mg. Cu.	Lactose, 100 mg. Mg. Cu.
6	2	112.2	103.0
8	4	134.1	132.8
9	5	137.1	139.0
12	8	143.8	154.1
14	10	143.0	161.1
16	12	148.3	163.6
18	14	150.4	167.3
20	16	153.4	169.4

The figures in the first line of the last table above give the weights of copper reduced under the conditions of Munson and Walker. It is apparent that the reduction under these conditions is far from complete and that the speed of reaction at this point is too great to allow of an accurate determination of reducing power. The reason for the incomplete reduction after two minutes' boiling is undoubtedly due to the slower reaction of the carbonate-salicylate solution than of the hydroxide-tartrate solution.

When the determinations of reducing power are done in sets of four or more time is saved per determination by making the time of heating as short as possible. However, it is evident that at least 12 minutes of boiling over a flame are required. When the time of heating is limited to 12 minutes it is impossible to filter one set while the following set is being heated, but if the time of heating be extended it is possible to give one's entire attention to filtering the reduced copper and hence there is no actual loss of time per determination.

To boil a solution over a flame for 12 to 16 minutes requires more

or less attention to maintain uniform conditions, but it is an easy matter to duplicate conditions of heating in a bath of boiling water and no attention is required during the heating. Furthermore, it was found that the results obtained by heating in boiling water are more accurate than those obtained by boiling the solution. After 20 minutes' heating in boiling water the reaction is nearly complete for glucose and invert sugar and there is only a slow rate of reduction for lactose and maltose. As heating beyond 20 minutes would mean a needless expenditure of time, it was decided to limit the reduction for all of the sugars to that obtained during 20 minutes' heating in boiling water.

Although the salicylic acid is employed in the alkaline solution and must, therefore, exist as potassium salicylate, it was found impossible to replace the acid with sodium salicylate and obtain the same reducing power for maltose.

The following results show the difference between the free acid and the sodium salt:

Grams	Sodium salicylate	Salicylic acid
3	117.4	141.3
4	126.0	143.1
5	131.9	148.4
6	136.2	148.7

The volume was 125 c.c., 2 g. copper sulfate, 11.5 g. potassium carbonate, and 100 mg. of maltose being present. The figures are milligrams of copper reduced.

When the salicylic acid and sodium salicylate were kept constant and the potassium carbonate varied, the following results were obtained:

Potassium carbonate Grams	Sodium salicylate 3 grams Mg. Cu.	Salicylic acid 3 grams Mg. Cu.
15	123.1	145.6
18	125.7	146.8
21	130.4	148.1
24	135.8	151.9

The volume was 125 c.c., 2 g. of copper sulfate and 100 mg. maltose were used.

Another series where more salicylic acid and sodium salicylate were used gave the following results:

Potassium carbonate Grams	Sodium salicylate Grams	Copper
15	6	138.3
18	6	142.1
21	6	144.3
24	6	147.8

Potassium carbonate Grams	Salicylic acid Grams	Copper
12	6	148.1
15	6	152.6
18	6	157.0
21	6	158.3

Although it would seem to make no difference when the sugar was added to the solution of potassium-copper salicylate, experiment showed that it is necessary to add the copper to the solution and not *vice versa.* A series where 15 g. of potassium carbonate and 5 g. of salicylic acid were used, and 100 mg. of maltose were added to this solution gave 152.6, 146.2, 149.1, and 156.2 mg. of copper reduced. Under identical conditions, but where the copper solution was added to the sugar, the following weights of copper were obtained: 154.9, 155.0, 154.8, 154.9.

The effect of mixing the sugar and alkaline copper solutions and allowing to stand in the cold is shown by the following results: To four flasks, each containing 100 c.c. of water, 100 mg. of lactose and 25 c.c. of copper sulfate (2 grams) were added. At intervals of 5 minutes, 12 grams of potassium carbonate and 3 grams of salicylic acid were added to the 4 flasks in succession. The flask to which the potassium carbonate and salicylic acid was first added would have stood 15 minutes before the mixture had been added to the fourth flask. As soon as the fourth flask was ready they were all placed in the boiling water, and allowed to remain 20 minutes; the following weights of copper were obtained:

Solution stood in the cold 15 min., 149.8
Solution stood in the cold 10 min., 147.4
Solution stood in the cold 5 min., 146.3
Solution stood in the cold 0 min., 146.5

These results show that the sugar can stand in the alkaline copper solution for 5-6 minutes in the cold without any appreciable change, but that a slight reduction will occur if they are allowed to stand

10-15 minutes. In practice 2-3 minutes is all that is needed to dissolve the salicylic acid and prepare the solutions for the boiling water.

The temperature at which the solutions are added to the boiling water is without appreciable influence between 18° and 50°. Four solutions, each containing 12 g. potassium carbonate, 3 g. salicylic acid, 2 g. copper sulfate, and 100 mg. lactose, when placed in the boiling water at the indicated temperature gave the following weights of copper reduced:

18°, 150.4 mg. copper; 30°, 150.7 mg. copper; 40°, 150.7 mg. copper; 50°, 151.5 mg. copper.

It is essential to have the boiling water heated with a flame large enough to cause the water to begin boiling within 1.5-2 minutes after the addition of the flasks containing the sugar-copper solutions.

Two flasks, containing 12 g. of potassium carbonate, 3 g. of salicylic acid, 2 g. of copper sulfate, and 120 mg. of lactose in 125 c.c., were placed in the boiling water with a flame under the bath, which caused the water to boil within 1.5-2 minutes after the flasks were placed in the bath. The copper reduced at the end of 20 minutes was 175.5 and 176.3 mg. Two other flasks containing identical solutions were placed in the boiling water with a flame under the bath which caused the water to boil in 5-6 minutes after the addition of the flasks. After 20 minutes from the time the flasks were placed in the water the copper reduced was 167.9 and 171.8 mg., showing lower and irregular results.

In order to show the variations caused by slight differences in the weights of potassium carbonate and salicylic acid added, the following series of determinations were made under identical conditions of time and volume. The volume was 140 c.c. and time 20 minutes:

Potassium carbonate Grams	Salicylic acid Grams	Glucose 50 mg.	Invert sugar 50 mg.	Lactose 100 mg.	Maltose 100 mg.
15	4	148.9	155.2	160.0	154.9
15	5	149.2	155.7	157.2	155.0
15	6	.....	156.7	154.9	154.9
15	7	150.0	158.0	151.4	154.9
13	5	151.4	.....	152.9	153.0
14	5	151.4	158.3	154.5	154.6
15	5	149.4	155.7	157.2	155.0
16	5	149.4	156.1	156.9	156.3
17	5	147.3	155.5	160.1	158.4

These results show that glucose, maltose, and invert sugar vary but slightly for differences in amounts of potassium carbonate and salicylic acid present and that lactose is more sensitive in this respect.

In practical determinations of sugar, the variations in the weights of potassium carbonate and salicylic acid can be controlled within ± 0.2 g. without taking any special precautions, and it is apparent that such a variation causes no appreciable change in the reducing power of any of the sugars.

DETERMINATION OF THE COPPER REDUCED.

The writer recently described a method for the determination of copper by means of the iodide method. The method described was devised primarily for the determination of copper obtained by the reduction with sugar. It differs from the original iodide method in that the solutions are prepared for titration in the cold, thus overcoming the delay caused by boiling the solution or evaporating to dryness.

During the course of this investigation several hundred determinations of copper have been made by this modification of the iodide method, and these results show that, if the conditions prescribed are followed, the determination of copper can be made by this method with great accuracy. Irrespective of the way the reduced copper is determined it has to be removed from its filter, and the most convenient way to do this is to dissolve it in nitric acid. The iodide method allows of the accurate determination of the copper thus dissolved, hence doing away with drying and weighing, which is time-consuming and laborious.

The cuprous oxide reduced by the sugar is filtered on a glass funnel such as is usually employed to hold a Gooch crucible. The filter is made by placing a perforated porcelain disk in the bottom of the funnel and making an asbestos felt 6 to 8 mm. in thickness. A porcelain disk should be used to hold the asbestos, as glass wool retains traces of alkaline copper solution. After the solution has been filtered with suction and washed with hot water, the funnel and rubber stopper are removed from the suction flask, washed free from any copper solution which may adhere to the outside, and placed on a 350 c.c. suction flask. If the stopper does not fit, the top of the flask is ground smooth on a carborundum hone so that when suction is applied it will hold the stopper down tightly over the mouth. Before the suction is applied to the flask the cuprous oxide is dissolved in not less than 10

c.c. of hot nitric acid (1 part of acid to 3 of water). It is imperative to have the nitric acid hot and it should be contained in a wash bottle which delivers a small stream. The flask in which the reduction takes place and the sides of the funnel are washed with the hot acid and then the asbestos is stirred up by the jet from the wash bottle. It is best not to have a porcelain disk on the surface of the asbestos. The hot nitric acid will dissolve only a trace of nitrous oxide, but if cold nitric acid is used large amounts of nitrous acid will be dissolved, which will prevent the accurate determination of the copper. When all of the cuprous oxide has been dissolved the suction is applied and the funnel is washed with several additions of small amounts of water —not more than 10-12 c.c. at a time. The wash water is sucked through each time before more is added. All of the copper, when washed in this way, can be removed with 40-50 c.c. of wash water. The copper in solution may now be determined as described in the method.[5]

PREPARATION AND ANALYSIS OF THE SUGARS USED.

The four sugars used to determine the relation between sugar and copper given in the table below were prepared and analyzed as follows:

The glucose of highest purity, furnished by Merck & Co., when analyzed for moisture and rotating power, showed 0.16 per cent of moisture and a specific rotating power of 52.68°. As the specific rotating power was in accordance with that given by Tollens for pure glucose no further purification was considered necessary.

The sucrose was prepared from Kahlbaum's C. P. saccharose by the method outlined by the International Commission for the Unification of Sugar Analysis. The sample thus prepared contained 0.13 per cent of moisture and showed a specific rotating power of 66.5°.[6]

The lactose was prepared from Kahlbaum's crystallized lactose by dissolving in boiling water, filtering, and allowing the lactose to crystallize for 7 days from this solution. These crystals were dried in a vacuum over sulfuric acid for three days, ground into a powder, and again dried in vacuum over sulfuric acid. The determination of

[5]This Journal, 33, 1947.

[6]The sucrose was inverted essentially by the method used by Munson and Walker, by heating for 30 minutes in boiling water with $N/_{10}$ HCl, using 20 c.c. for every 100 c.c. final volume. The solution was barely neutralized with $N/_{10}$ sodium hydroxide, cooled, and filled at 20° to the mark of graduation.

moisture showed 5.62 per cent of water. One molecule of water, corresponding to the formula $C_{12}H_{22}O_{11}H_2O$, requires 5.0 per cent of water, therefore the sample thus prepared contained 0.62 per cent excess water calculated as lactose hydrate. Its specific rotating power calculated as $C_{12}H_{22}O_{11}H_2O$ was 52.51°.

The maltose was prepared by letting barley diastase act on soluble starch as described by Baker and Day[7] and Baker.[8] The maltose thus prepared was dried in an electric oven at 70-80° for 15-18 hours, and was then ground and passed through a 60-mesh sieve. The moisture determination showed 5.33 per cent of water. As maltose hydrate, $C_{12}H_{22}O_{11}.H_2O$, requires 5 per cent of water there was present 0.33 per cent excess water. The specific rotating power was 137.3°.

The rotating power of all preparations was determined with sodium light, using a 4 decimeter tube in a Schmidt and Haensch polariscope at 20°. The solutions were 10 per cent of sugar, except for maltose, which was 5 per cent. The solutions, except for sucrose, were allowed to stand 24 hours at room temperature to destroy the multirotation.

The water content of all preparations were determined as follows: Two grams of the sample in a small weighing bottle was placed in the bottom of a 4-inch desiccator which had a suction outlet in the lid. Around the weighing bottle was placed a wire gauze collar about 1.75 inches in diameter. This wire gauze supported a Petri dish of 3 inches diameter which contained phosphorus pentoxide. A second Petri dish of almost the same diameter as the desiccator was supported above the first Petri dish by three wire supports at a distance of 0.5 inch above the lower Petri dish. This top Petri dish acted as a cover and prevented the phosphorus pentoxide from dusting when the suction was released. After placing the cover on the desiccator it was placed inside an electric oven and connected with stout suction hose to a Gaede pump. The temperature of the oven was kept for lactose at 130°, maltose 110°, sucrose and glucose 100°, for 4-5 hours. The suction was maintained during the entire time of heating. At intervals of one hour the weighing bottle was taken out of the desiccator and weighed. When heating for one hour produced a change of less than 0.5 mg. the heating was discontinued. Duplicate determinations were made in all cases.

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[7]Analyst, 33, 393 (1908).
[8]J. Chem. Soc., 1902, 1177.

DETERMINATION OF RELATION BETWEEN SUGAR AND COPPER.

For maltose and lactose the sugar was weighed out in such quantities as to make 5 mg. per c.c. of solution, allowance being made for the presence of the water; the weights of sugar were calculated as maltose and lactose hydrate, $C_{12}H_{22}O_{11}.H_2O$.

For invert sugar and glucose, solutions were made which contained 2.5 mg. of sugar calculated as $C_6H_{12}O_6$.

The temperature of graduation of both flasks and burets was 20°.

THE REDUCING POWER OF THE SUGARS.

The reducing power of the sugars was determined as follows: The varying weights of sugar as indicated below were measured into 200 c.c. Erlenmeyer flasks and the volume in each case made up to 100 c.c. with distilled water. Five grams of salicylic acid were now added to each of 4 flasks containing the sugar to be determined. Fifteen c.c.[9] of copper sulfate solution and then 25 c.c. potassium carbonate[10] solution were added to each of the flasks without any agitation of the solution. It was found necessary to observe this order for the addition of the reagents. The flasks were then shaken with a rotary motion. The precipitate of copper carbonate dissolved, forming a dark green solution. As soon as the salicylic acid dissolved the four flasks were put in a holder and placed in a bath of boiling water.[11]

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[9]The copper sulfate solution is prepared by dissolving 133.33 grams of $CuSO_4.5H_2O$ per liter of water; 15 c.c. of such a solution contains 2 grams of copper sulfate.

[10]The potassium carbonate solution contains 600 g. of anhydrous potassium carbonate per liter; 25 c.c. of this solution contains 15 g. of potassium carbonate. As potassium carbonate is hygroscopic, it is necessary to drive off all water before making up the solution. This is done by heating the carbonate for 3 to 4 hours at a temperature from 190° to 200°. A sample of the salt thus treated, when heated in a small test tube, should not give any evidence of liberation of water. The carbonate solution should be made up in a liter or other size graduated flask, and should be diluted almost to the proper volume, well mixed, and then adjusted to the mark of graduation.

[11]The water bath used during this investigation was one 10 inches in diameter and 6½ inches deep. It contained 4 liters of water. The level of the water could be maintained constant by using the ordinary constant water-level bath, or by means of a syphon acting between the water bath and a reservoir of water of constant level. The syphon, for constant use, must have a T or Y tube inserted at its highest point. A 20 c.c. pipet closed at one end with rubber hose and pinchcock is connected with the T tube. To start the syphon,

The flame under the bath must be of such size that boiling begins within 1.5 to 2 minutes after addition of the flasks. At the end of 20 minutes from the time the flasks were placed in the bath the solutions were filtered and the copper determined as described above.

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both ends are placed under water and (the pinchcock being open) the air is withdrawn from the pipet. When the pipet is full of water the pinchcock is closed. Any bubbles of air liberated from the water in the tube of the syphon will rise to the highest point of the tube, and will there be caught in the pipet, displacing the water. Should the pipet become filled with air, the syphon may be re-established by again withdrawing the air through the rubber tube and pinchcock. The end of the syphon which is in the water bath must have a small opening (about ⅛ inch). If a large opening is used, the the water, when boiling, may syphon out of the bath. The diameter of the rest of the syphon may be of any sized tubing.

The flame under the water bath was supplied by one large-sized Fletcher and two Bunsen burners. The water should be boiling vigorously when the flasks are placed in the water, and all of the flames should be burning. As soon as the water has begun to boil, after the addition of the flasks (which should be in less than two minutes), the two Bunsen burners are turned out, as the Fletcher burner is sufficient to maintain the boiling.

In the practical application of this method for the determination of the reducing sugars, it is most convenient to make a set of 4 determinations at once. A rack is made to hold the 4 flasks, as follows: a brass rod ¼ inch in diameter and 14 inches long, is erected at the center point, and perpendicular to a brass disk 8 inches in diameter and $^{3}/_{32}$ inch thick. This disk forms the bottom upon which the flasks are placed. Another disk of the same size as the bottom one, but with a hole $^{5}/_{16}$ inch in diameter, slides up and down the rod. Four holes 1½ inches in diameter are drilled through this second disk, the centers of the holes being arranged symmetrically 2¼ inches from the center of the disk. This top disk being lifted up, the four flasks are placed on the bottom of the holder. When the top disk is lowered the flasks will pass through the four holes, and, as the opening is only sufficiently large to allow the neck of the flask to pass through, the flasks are held secure. A hook at the end of the rod is used to hang the entire holder from a support above the bath, at such a height that the rack sinks in the water up to the level of the top disk. In the bottom disk a number of ½-inch holes are drilled, so that the rack may be placed in and removed from the water with ease. When the flasks are removed from the rack the solutions are filtered immediately on four suction flasks.

TABLE I.

Weight of sugar	Glucose		Invert sugar		Weight of sugar	Lactose		Maltose	
Mg.					Mg.				
10	30.4	30.2	30.9	30.7	20	31.9	32.1	31.0	30.3
20	60.9	61.8	63.2	62.4	40	64.0	62.5	61.0	61.3
30	90.2	90.6	94.4	94.7	60	95.5	95.2	93.4	92.3
40	120.0	120.1	126.5	126.8	80	124.5	125.0	123.6	123.6
50	148.1	149.9	157.5	156.7	100	157.7	157.7	154.6	154.6
60	176.8	179.2	188.2	186.4	120	287.4	286.3	187.9	187.2
70	206.1	207.4	217.5 217.5	219.7 215.6	140	218.5	217.0	219.2	218.5
80	233.8	233.8	245.3 245.6	245.3 *248.0	160	246.3	246.7	250.6	250.4
90	261.3	263.3	276.1	276.1	180	279.0	278.4	278.6	280.1
100	288.0	289.2	302.6	302.6	200	306.8	305.9	309.6 310.9	*313.6 *312.4
110	316.2	314.7	330.8	330.7	220	338.7	337.1	342.2	342.2
120	343.1	341.5	359.4	358.6	240	364.6 364.9	*363.4	373.3	372.9
130	367.7	367.7	385.5	386.4	260	397.3	397.1	403.2	401.6
140	391.2	393.0	410.9	410.9	280	424.9	427.0	431.4	431.4 *429.4
150	418.5	418.5	438.1	438.1	300	455.6	454.3	459.4 *456.2	458.1 *454.3
160	443.3	442.1	462.1	460.9	...	.....	.....	.....	.....

TABLE II.

Glucose			Invert sugar			Lactose			Maltose		
F'nd	Calculated	Error	F'nd	Calculated	Error	F'nd	Calculated	Error	F'nd	Calculated	Error
Mg.	Mg.	Mg.	Mg.	Mg.	Mg.	Mg.	Mg.	Mg.	Mg.	Mg.	Mg.
30.3	30.6	0.3	30.8	31.43	0.63	32.0	32.07	0.07	30.6	30.2	—0.40
61.4	60.9	—0.5	62.8	63.55	0.75	63.3	63.38	0.08	61.2	61.96	0.76
90.4	90.8	0.4	94.6	95.24	0.64	95.4	94.52	—0.88	92.9	93.57	0.67
120.1	120.3	0.2	126.7	126.44	—0.26	124.8	125.48	0.68	123.6	125.02	1.42
149.0	149.4	0.4	157.1	157.15	0.05	157.7	156.28	—1.42	154.6	156.32	1.72
178.0	178.1	0.1	187.3	187.37	0.07	186.9	186.90	0	187.6	187.47	—0.13
206.8	206.4	—0.4	217.7	217.10	—0.60	217.8	217.34	—0.46	218.9	218.47	—0.43
233.8	234.3	0.5	245.5	246.33	0.83	246.5	247.60	1.10	250.5	249.32	—1.18
262.3	261.7	—0.6	276.1	275.07	—1.03	278.7	277.70	—1.00	279.4	280.04	0.64
288.6	288.8	0.2	302.6	303.33	0.73	306.4	307.63	1.23	310.3	310.59	0.29
315.5	315.5	0	330.8	331.09	0.29	337.9	337.38	—0.52	342.2	341.00	—1.20
342.3	341.7	—0.6	359.0	358.35	—0.65	364.8	366.95	2.15	373.1	371.25	—1.85
367.7	367.6	—0.1	386.0	385.14	—0.86	397.2	396.35	—0.85	402.4	401.35	—1.05
392.1	393.0	0.9	410.9	411.43	0.53	426.0	425.58	—0.42	431.4	431.31	—0.09
418.5	418.1	—0.4	438.1	437.23	—0.87	455.0	454.64	—0.36	458.7	461.12	2.42
442.7	442.7	0	461.5	462.55	1.05	.....	.....	....	.....	.....	....

TABLE III.

	Dextrose		Invert sugar		Lactose		Maltose	
Method	50 mg.	100 mg.	50 mg.	100 mg.	100 mg.	200 mg.	100 mg.	200 mg.
Defren	89.8	178.1	.....	.....	125.7	248.4	109.5	216.5
Munson and Walker	102.2	198.4	98.5	192.3	132.7	261.6	108.7	215.4
Allihn	98.2	195.0	.....	.....	.....	.....	.....	.....
Lehmann	.....	.....	101.4	190.0	.....	.....	.....	.....
Meissl	.....	.....	96.0	189.2	.....	.....	.....	.....
Soxhlet	.....	.....	.....	.....	138.3	269.6	.....	.....
Wein	.....	.....	.....	.....	.....	.....	115.1	226.8
Ost	165.6	294.3	170.0	298.0	.....	.....	167.5	331.8
Kjeldahl	107.2	197.7	101.7	190.2	.....	.....	.....	.....
Brown, Morris & Millar	103.0	202.7	97.5	194.1	.....	.....	.....	.....
This method	149.3	288.7	157.0	303.3	156.3	307.6	156.3	310.6

Using this method of procedure, the results in Table I were obtained for the indicated weights of sugar. All of the results which were obtained are given, but the ones which were not used in the calculation of the curve are starred:

From these results the following equations showing the relation between sugar and copper were deduced by means of the method of least squares.[12]

In the equations X refers to weights of copper reduced, Y to weights of sugar reacting:

Glucose ..... $X = -0.17 + 3.0923\ Y - 0.002026\ Y^2$
Invert sugar ..... $X = -1.30 + 3.2918\ Y - 0.002455\ Y^2$
Lactose ..... $X = 0.59 + 1.5786\ Y - 0.000217\ Y^2$
Maltose ..... $X = -1.69 + 1.5988\ Y - 0.000187\ Y^2$

The weights of sugar given in Table I were substituted in the proper equation and the corresponding values of X were found. The differences between the values thus calculated and actually found is given in Table II.

By means of the equation the copper equivalent to weights of maltose and lactose from 20 to 300 mg. were calculated for every 4 mg. of sugar. With glucose and invert sugar the copper equivalent for every 2 mg. of sugar from 10 to 160 was found. This gave a series of points which differed by about 6 mg. of copper. These figures were changed

[12] A good example of the use of this method is given in Allihn's original article, J. prakt. Chem., 22, 46 (1880).

to integral weights of copper and hence decimal weights of sugar. The figures lying between each 6 mg. of copper were interpolated, the interpolations being carried to the second decimal place. The complete table giving the relation between the 4 reducing sugars and copper for every mg. of copper from 30 to 450 mg. is given in Table IV.

A comparison of the reducing power of the sugars obtained by this method with that obtained with other methods in use is given in Table III.

I wish to express my appreciation to Mr. A. W. Thomas for assistance during the course of this investigation.

TABLE IV

Copper Mg.	Cuprous oxide Mg.	Cupric oxide Mg.	Glucose Mg. $C_6H_{12}O_6$	Invert sugar Mg. $C_6H_{12}O_6$	Lactose Mg. $C_{12}H_{22}O_{11}.H_2O$	Maltose Mg. $C_{12}H_{22}O_{11}.H_2O$
30	33.8	37.6	9.8	9.6	19.3	19.9
31	34.9	38.8	10.2	9.9	20.0	20.5
32	36.0	40.1	10.5	10.2	20.6	21.1
33	37.2	41.3	10.8	10.5	21.2	21.7
34	38.3	42.6	11.1	10.8	21.8	22.4
35	39.4	43.8	11.5	11.1	22.5	23.0
36	40.5	45.1	11.8	11.4	23.1	23.6
37	41.7	46.3	12.1	11.8	23.7	24.3
38	42.8	47.6	12.4	12.1	24.4	24.9
39	43.9	48.8	12.8	12.4	25.0	25.5
40	45.0	50.1	13.1	12.7	25.6	26.1
41	46.2	51.3	13.4	13.0	26.3	26.8
42	47.3	52.6	13.8	13.3	26.9	27.4
43	48.4	53.8	14.1	13.6	27.5	28.0
44	49.5	55.1	14.4	13.9	28.1	28.7
45	50.7	56.3	14.8	14.2	28.8	29.3
46	51.8	57.6	15.1	14.5	29.4	29.9
47	52.9	58.8	15.4	14.8	30.0	30.6
48	54.0	60.1	15.7	15.2	30.6	31.2
49	55.2	61.3	16.1	15.5	31.3	31.8
50	56.3	62.6	16.4	15.8	31.9	32.4
51	57.4	63.8	16.7	16.1	32.5	33.1
52	58.5	65.1	17.1	16.4	33.2	33.7
53	59.7	66.3	17.4	16.7	33.8	34.3
54	60.8	67.6	17.7	17.0	34.4	35.0
55	61.9	68.8	18.1	17.3	35.0	35.6
56	63.0	70.1	18.4	17.6	35.7	36.2
57	64.2	71.3	18.7	17.9	36.3	36.9

TABLE IV (*continued*)

Copper Mg.	Cuprous oxide Mg.	Cupric oxide Mg.	Glucose Mg. $C_6H_{12}O_6$	Invert sugar Mg. $C_6H_{12}O_6$	Lactose Mg. $C_{12}H_{22}O_{11}.H_2O$	Maltose Mg. $C_{12}H_{22}O_{11}.H_2O$
58	65.3	72.6	19.1	18.3	36.9	37.5
59	66.4	73.9	19.4	18.6	37.6	38.1
60	67.6	75.1	19.7	18.9	38.2	38.8
61	68.7	76.4	20.0	19.2	38.8	39.4
62	69.8	77.6	20.4	19.5	39.4	40.0
63	70.9	78.9	20.7	19.8	40.1	40.7
64	72.1	80.1	21.0	20.1	40.7	41.3
65	73.2	81.4	21.4	20.5	41.3	41.9
66	74.3	82.6	21.7	20.8	41.9	42.5
67	75.4	83.9	22.0	21.1	42.6	43.2
68	76.6	85.1	22.4	21.4	43.2	43.8
69	77.7	86.4	22.7	21.7	43.8	44.4
70	78.8	87.6	23.0	22.0	44.4	45.1
71	79.9	88.9	23.4	22.3	45.1	45.7
72	81.1	90.1	23.7	22.7	45.7	46.3
73	82.2	91.4	24.0	23.0	46.3	47.0
74	83.3	92.6	24.4	23.3	46.9	47.6
75	84.4	93.9	24.7	23.6	47.5	48.2
76	85.6	95.1	25.0	23.9	48.1	48.9
77	86.7	96.4	25.4	24.2	48.8	49.5
78	87.8	97.6	25.7	24.5	49.4	50.1
79	88.9	98.9	26.0	24.9	50.0	50.8
80	90.1	100.1	26.4	25.2	50.7	51.4
81	91.2	101.4	26.7	25.5	51.3	52.0
82	92.3	102.6	27.1	25.8	51.9	52.7
83	93.4	103.9	27.4	26.1	52.6	53.3
84	94.6	105.1	27.7	26.4	53.2	53.9
85	95.7	106.4	28.1	26.8	53.9	54.6
86	96.8	107.6	28.4	27.1	54.5	55.2
87	97.9	108.9	28.7	27.4	55.1	55.8
88	99.1	110.1	29.1	27.7	55.8	56.5
89	100.2	111.4	29.4	28.0	56.4	57.1
90	101.3	112.7	29.7	28.3	57.1	57.7
91	102.4	113.9	30.1	28.7	57.7	58.3
92	103.6	115.2	30.4	29.0	58.4	58.9
93	104.7	116.4	30.8	29.3	59.0	59.6
94	105.8	117.7	31.1	29.6	59.7	60.2
95	106.9	118.9	31.4	29.9	60.3	60.9
96	108.1	120.2	31.8	30.2	60.9	61.5
97	109.2	121.4	32.1	30.6	61.6	62.2
98	110.3	122.7	32.4	30.9	62.2	62.8
99	111.5	123.9	32.8	31.2	62.8	63.4

TABLE IV (*continued*)

Copper Mg.	Cuprous oxide Mg.	Cupric oxide Mg.	Glucose Mg. $C_6H_{12}O_6$	Invert sugar Mg. $C_6H_{12}O_6$	Lactose Mg. $C_{12}H_{22}O_{11}.H_2O$	Maltose Mg. $C_{12}H_{22}O_{11}.H_2O$
100	112.6	125.2	33.1	31.5	63.5	64.1
101	113.7	126.4	33.5	31.8	64.2	64.7
102	114.8	127.7	33.8	32.2	64.8	65.3
103	116.0	128.9	34.1	32.5	65.4	66.0
104	117.1	130.2	34.5	32.8	66.1	66.6
105	118.2	131.5	34.8	33.1	66.8	67.2
106	119.3	132.7	35.2	33.4	67.4	67.8
107	120.5	134.0	35.5	33.8	68.0	68.5
108	121.6	135.2	35.9	34.1	68.7	69.1
109	122.7	136.5	36.2	34.4	69.3	69.8
110	123.8	137.7	36.6	34.7	70.0	70.4
111	125.0	139.0	36.9	35.0	70.6	71.1
112	126.1	140.2	37.2	35.4	71.3	71.7
113	127.2	141.5	37.5	35.7	71.9	72.3
114	128.3	142.7	37.9	36.0	72.6	73.0
115	129.4	144.0	38.2	36.3	73.2	73.6
116	130.6	145.2	38.5	36.7	73.8	74.2
117	131.7	146.5	38.9	37.0	74.5	74.9
118	132.8	147.7	39.2	37.3	75.1	75.5
119	134.0	149.0	39.6	37.6	75.8	76.2
120	135.1	150.2	39.9	37.9	76.4	76.8
121	136.2	151.5	40.3	38.3	77.1	77.4
122	137.4	152.7	40.6	38.6	77.7	78.1
123	138.5	154.0	40.9	38.9	78.4	78.7
124	139.6	155.2	41.3	39.2	79.0	79.4
125	140.7	156.5	41.6	39.5	79.7	80.0
126	141.9	157.7	42.0	39.9	80.3	80.6
127	143.0	159.0	42.3	40.2	81.0	81.3
128	144.1	160.2	42.6	40.5	81.6	81.9
129	145.2	161.5	43.0	40.8	82.3	82.5
130	146.4	162.7	43.3	41.2	82.9	83.2
131	147.5	164.0	43.7	41.5	83.6	83.8
132	148.6	165.2	44.0	41.8	84.2	84.5
133	149.7	166.5	44.4	42.1	84.9	85.2
134	150.9	167.7	44.7	42.5	85.5	85.8
135	152.0	169.0	45.1	42.8	86.2	86.5
136	153.1	170.2	45.4	43.1	86.8	87.1
137	154.2	171.5	45.7	43.4	87.4	87.7
138	155.4	172.7	46.1	43.8	88.1	88.3
139	156.5	174.0	46.4	44.1	88.7	88.9
140	157.6	175.2	46.8	44.4	89.4	89.6
141	158.7	176.5	47.1	44.7	90.0	90.2

TABLE IV (*continued*)

Copper Mg.	Cuprous oxide Mg.	Cupric oxide Mg.	Glucose Mg. $C_6H_{12}O_6$	Invert sugar Mg. $C_6H_{12}O_6$	Lactose Mg. $C_{12}H_{22}O_{11}.H_2O$	Maltose Mg. $C_{12}H_{22}O_{11}.H_2O$
142	159.9	177.7	47.5	45.1	90.7	90.8
143	161.0	179.0	47.8	45.4	91.3	91.5
144	162.1	180.2	48.1	45.7	92.0	92.1
145	163.2	181.5	48.5	46.0	92.6	92.8
146	164.4	182.7	48.8	46.4	93.3	93.4
147	165.5	184.0	49.2	46.7	93.9	94.0
148	166.6	185.2	49.5	47.0	94.6	94.7
149	167.7	186.5	49.9	47.4	95.3	95.3
150	168.9	187.8	50.2	47.7	95.9	96.0
151	170.0	189.0	50.6	48.0	96.6	96.6
152	171.1	190.3	50.9	48.3	97.2	97.2
153	172.3	191.5	51.3	48.7	97.9	97.9
154	173.4	192.8	51.6	49.0	98.5	98.5
155	174.5	194.0	52.0	49.3	99.2	99.2
156	175.6	195.3	52.3	49.6	99.8	99.8
157	176.8	196.5	52.7	50.0	100.5	100.4
158	177.9	197.8	53.0	50.3	101.1	101.1
159	179.0	199.0	53.4	50.6	101.8	101.7
160	180.1	200.3	53.7	50.9	102.4	102.4
161	181.3	201.5	54.0	51.3	103.1	103.0
162	182.4	202.8	54.4	51.6	103.7	103.6
163	183.5	204.0	54.7	51.9	104.4	104.3
164	184.6	205.3	55.1	52.3	105.0	104.9
165	185.8	206.5	55.4	52.6	105.7	105.6
166	186.9	207.8	55.8	52.9	106.3	106.2
167	188.0	209.0	56.1	53.3	107.0	106.8
168	189.1	210.3	56.5	53.6	107.6	107.5
169	190.3	211.5	56.8	53.9	108.3	108.1
170	191.4	212.8	57.2	54.2	108.9	108.8
171	192.5	214.0	57.5	54.6	109.6	109.4
172	193.6	215.3	57.9	54.9	110.2	110.1
173	194.8	216.5	58.2	55.2	110.9	110.7
174	195.9	217.8	58.6	55.6	111.6	111.3
175	197.0	219.0	58.9	55.9	112.2	112.0
176	198.1	220.3	59.3	56.2	112.9	112.6
177	199.3	221.5	59.6	56.6	113.5	113.3
178	200.4	222.8	60.0	56.9	114.2	113.9
179	201.5	224.0	60.3	57.2	114.9	114.5
180	202.6	225.3	60.7	57.6	115.5	115.2
181	203.8	226.5	61.0	57.9	116.1	115.8
182	204.9	227.8	61.4	58.2	116.8	116.5
183	206.0	229.0	61.7	58.6	117.4	117.1

TABLE IV (*continued*)

Copper Mg.	Cuprous oxide Mg.	Cupric oxide Mg.	Glucose Mg. $C_6H_{12}O_6$	Invert sugar Mg. $C_6H_{12}O_6$	Lactose Mg. $C_{12}H_{22}O_{11}.H_2O$	Maltose Mg. $C_{12}H_{22}O_{11}.H_2O$
184	207.1	230.3	62.1	58.9	118.1	117.8
185	208.3	231.5	62.4	59.2	118.8	118.4
186	209.4	232.7	62.8	59.6	119.4	119.0
187	210.5	234.0	63.1	59.9	120.1	119.7
188	211.7	235.3	63.5	60.2	120.7	120.3
189	212.8	236.5	63.9	60.6	121.4	121.0
190	213.9	237.8	64.2	60.9	122.0	121.6
191	215.0	239.0	64.6	61.2	122.7	122.3
192	216.2	240.3	64.9	61.6	123.3	122.9
193	217.3	241.5	65.3	61.9	124.0	123.6
194	218.4	242.8	65.6	62.2	124.7	124.2
195	219.5	244.0	66.0	62.6	125.3	124.8
196	220.7	245.3	66.3	62.9	126.0	125.5
197	221.8	246.5	66.7	63.2	126.6	126.1
198	222.9	247.8	67.0	63.6	127.3	126.8
199	224.0	249.0	67.4	63.9	127.9	127.4
200	225.2	250.3	67.8	64.2	128.6	128.1
201	226.3	251.5	68.1	64.6	129.2	128.7
202	227.4	252.8	68.5	64.9	129.9	129.4
203	228.5	254.0	68.8	65.2	130.6	130.0
204	229.7	255.3	69.2	65.6	131.2	130.6
205	230.8	256.5	69.5	65.9	131.9	131.3
206	231.9	257.8	69.9	66.2	132.5	131.9
207	233.0	259.0	70.2	66.6	133.2	132.6
208	234.2	260.3	70.6	66.9	133.8	133.2
209	235.3	261.5	71.0	67.3	134.5	133.9
210	236.4	262.8	71.3	67.6	135.2	134.5
211	237.6	264.0	71.7	67.9	135.8	135.2
212	238.7	265.3	72.0	68.3	136.5	135.8
213	239.8	266.5	72.4	68.6	137.1	136.5
214	240.9	267.8	72.7	69.0	137.8	137.1
215	242.1	269.0	73.1	69.3	138.5	137.8
216	243.2	270.3	73.4	69.6	139.1	138.4
217	244.3	271.5	73.8	70.0	139.8	139.1
218	245.4	272.8	74.2	70.3	140.4	139.7
219	246.6	274.1	74.5	70.7	141.1	140.3
220	247.7	275.4	74.9	71.0	141.8	141.0
221	248.7	276.6	75.2	71.4	142.4	141.6
222	249.9	277.9	75.6	71.7	143.1	142.3
223	251.0	279.1	76.0	72.0	143.7	142.9
224	252.1	280.4	76.3	72.4	144.4	143.6
225	253.3	281.6	76.7	72.7	145.1	144.2

TABLE IV (*continued*)

Copper Mg.	Cuprous oxide Mg.	Cupric oxide Mg.	Glucose Mg. $C_6H_{12}O_6$	Invert sugar Mg. $C_6H_{12}O_6$	Lactose Mg. $C_{12}H_{22}O_{11}.H_2O$	Maltose Mg. $C_{12}H_{22}O_{11}.H_2O$
226	254.4	282.9	77.0	73.1	145.7	144.9
227	255.6	284.1	77.4	73.4	146.4	145.5
228	256.7	285.4	77.8	73.7	147.0	146.2
229	257.8	286.6	78.1	74.1	147.7	146.8
230	258.9	287.9	78.5	74.4	148.4	147.5
231	260.1	289.1	78.8	74.8	149.0	148.1
232	261.2	290.4	79.2	75.1	149.7	148.8
233	262.3	291.6	79.6	75.4	150.3	149.4
234	263.4	292.9	79.9	75.8	151.0	150.1
235	264.6	294.1	80.3	76.1	151.7	150.7
236	265.7	295.4	80.6	76.5	152.3	151.4
237	266.8	296.6	81.0	76.8	153.0	152.0
238	268.0	297.9	81.4	77.2	153.6	152.6
239	269.1	299.1	81.7	77.5	154.3	153.3
240	270.2	300.4	82.1	77.8	155.0	153.9
241	271.3	301.6	82.5	78.2	155.6	154.6
242	272.5	302.9	82.8	78.5	156.3	155.2
243	273.6	304.1	83.2	78.9	157.0	155.9
244	274.7	305.4	83.5	79.2	157.6	156.5
245	275.8	306.6	83.9	79.6	158.3	157.2
246	277.0	307.9	84.3	79.9	159.0	157.8
247	278.1	309.1	84.6	80.2	159.6	158.5
248	279.2	310.4	85.0	80.6	160.3	159.1
249	280.3	311.6	85.4	80.9	160.9	159.8
250	281.5	312.9	85.7	81.3	161.6	160.4
251	282.6	314.1	86.1	81.6	162.2	161.1
252	283.7	315.4	86.5	82.0	162.9	161.7
253	284.8	316.6	86.8	82.3	163.6	162.4
254	286.0	317.9	87.2	82.7	164.2	163.0
255	287.1	319.1	87.6	83.0	164.9	163.7
256	288.2	320.4	87.9	83.4	165.6	164.3
257	289.3	321.6	88.3	83.7	166.2	165.0
258	290.5	322.9	88.7	84.1	166.9	165.6
259	291.6	324.1	89.0	84.4	167.6	166.3
260	292.7	325.4	89.4	84.8	168.2	166.9
261	293.8	326.6	89.8	85.1	168.9	167.6
262	295.0	327.9	90.1	85.5	169.5	168.3
263	296.1	329.1	90.5	85.8	170.2	168.9
264	297.2	330.4	90.8	86.1	170.9	169.6
265	298.3	331.6	91.2	86.5	171.6	170.2
266	299.5	332.9	91.6	86.8	172.2	170.9
267	300.6	334.1	91.9	87.2	172.9	171.5

TABLE IV (*continued*)

Copper Mg.	Cuprous oxide Mg.	Cupric oxide Mg.	Glucose Mg. $C_6H_{12}O_6$	Invert sugar Mg. $C_6H_{12}O_6$	Lactose Mg. $C_{12}H_{22}O_{11}.H_2O$	Maltose Mg. $C_{12}H_{22}O_{11}.H_2O$
268	301.7	335.4	92.3	87.5	173.5	172.2
269	302.8	336.7	92.7	87.9	174.2	172.8
270	304.0	338.0	93.1	88.2	174.9	173.5
271	305.1	339.2	93.4	88.6	175.5	174.1
272	306.2	340.5	93.8	88.9	176.2	174.8
273	307.3	341.7	94.2	89.3	176.9	175.4
274	308.5	343.0	94.5	89.6	177.5	176.1
275	309.6	344.2	94.9	90.0	178.2	176.7
276	310.7	345.5	95.3	90.3	178.9	177.4
277	311.9	346.7	95.6	90.7	179.5	178.0
278	313.0	348.0	96.0	91.1	180.2	178.7
279	314.1	349.2	96.4	91.4	180.9	179.3
280	315.2	350.5	96.7	91.8	181.5	180.0
281	316.4	351.7	97.1	92.1	182.2	180.6
282	317.5	353.0	97.5	92.5	182.9	181.3
283	318.6	354.2	97.9	92.8	183.5	181.9
284	319.7	355.5	98.2	93.1	184.2	182.6
285	320.9	356.7	98.6	93.5	184.9	183.2
286	322.0	358.0	99.0	93.9	185.5	183.9
287	323.1	359.2	99.4	94.2	186.2	184.6
288	324.2	360.5	99.7	94.6	186.9	185.2
289	325.3	361.7	100.1	94.9	187.6	185.9
290	326.4	363.0	100.5	95.3	188.2	186.5
291	327.5	364.2	100.8	95.6	188.8	187.2
292	328.7	365.5	101.2	96.0	189.5	187.8
293	329.9	366.7	101.6	96.3	190.1	188.5
294	331.0	368.0	101.9	96.7	190.8	189.1
295	332.1	369.2	102.3	97.1	191.4	189.8
296	333.3	370.5	102.7	97.4	192.1	190.4
297	334.4	371.7	103.1	97.8	192.8	191.1
298	335.5	373.0	103.4	98.1	193.5	191.8
299	336.6	374.2	103.8	98.5	194.2	192.4
300	337.8	375.5	104.2	98.9	194.9	193.1
301	338.9	376.7	104.6	99.2	195.6	193.7
302	340.0	378.0	105.0	99.6	196.2	194.4
303	341.1	379.2	105.3	99.9	196.9	195.0
304	342.3	380.5	105.7	100.3	197.6	195.7
305	343.4	381.7	106.1	100.6	198.3	196.3
306	344.5	383.0	106.4	101.0	198.9	197.0
307	345.6	384.2	106.8	101.3	199.6	197.6
308	346.8	385.5	107.2	101.7	200.3	198.3
309	347.9	386.7	107.6	102.1	200.9	199.0

TABLE IV (*continued*)

Copper Mg.	Cuprous oxide Mg.	Cupric oxide Mg.	Glucose Mg. $C_6H_{12}O_6$	Invert sugar Mg. $C_6H_{12}O_6$	Lactose Mg. $C_{12}H_{22}O_{11}.H_2O$	Maltose Mg. $C_{12}H_{22}O_{11}.H_2O$
310	349.0	388.0	107.9	102.4	201.6	199.6
311	350.1	389.2	108.3	102.8	202.3	200.3
312	351.3	390.5	108.7	103.1	202.9	200.9
313	352.4	391.7	109.1	103.5	203.6	201.6
314	353.5	393.0	109.5	103.8	204.3	202.2
315	354.6	394.2	109.8	104.2	204.9	202.9
316	355.8	395.5	110.2	104.6	205.5	203.6
317	356.9	396.7	110.6	104.9	206.2	204.2
318	358.0	398.0	111.0	105.3	206.8	204.9
319	359.1	399.2	111.4	105.6	207.5	205.5
320	360.3	400.5	111.7	106.0	208.2	206.2
321	361.4	401.7	112.1	106.4	208.9	206.8
322	362.5	403.0	112.5	106.7	209.6	207.5
323	363.7	404.2	112.9	107.1	210.3	208.2
324	364.8	405.5	113.2	107.5	211.0	208.8
325	365.9	406.7	113.6	107.8	211.7	209.5
326	367.0	408.0	114.0	108.2	212.3	210.1
327	368.2	409.2	114.4	108.5	213.0	210.8
328	369.3	410.5	114.8	108.9	213.6	211.5
329	370.4	411.8	115.1	109.3	214.3	212.1
330	371.5	413.1	115.5	109.6	214.9	212.8
331	372.7	414.3	115.9	110.0	215.6	213.4
332	373.8	415.6	116.3	110.3	216.3	214.1
333	374.9	416.8	116.7	110.7	217.0	214.7
334	376.0	418.1	117.0	111.1	217.7	215.4
335	377.2	419.3	117.4	111.5	218.4	216.1
336	378.3	420.6	117.8	111.8	219.1	216.7
337	379.4	421.9	118.2	112.2	219.8	217.4
338	380.5	423.1	118.6	112.5	220.4	218.0
339	381.7	424.4	119.0	112.9	221.1	218.7
340	382.8	425.6	119.4	113.3	221.7	219.3
341	383.9	426.9	119.8	113.6	222.4	220.0
342	385.0	428.1	120.1	114.0	223.0	220.7
343	386.2	429.4	120.5	114.4	223.7	221.3
344	387.3	430.6	120.9	114.7	224.4	222.0
345	388.4	431.9	121.3	115.1	225.1	222.6
346	389.6	433.1	121.7	115.5	225.8	223.3
347	390.7	434.4	122.1	115.8	226.5	224.0
348	391.8	435.6	122.4	116.2	227.2	224.6
349	392.9	436.9	122.8	116.6	227.9	225.3
350	394.0	438.1	123.2	117.0	228.5	225.9
351	395.2	439.4	123.6	117.3	229.2	226.6

TABLE IV (*continued*)

Copper Mg.	Cuprous oxide Mg.	Cupric oxide Mg.	Glucose Mg. $C_6H_{12}O_6$	Invert sugar Mg. $C_6H_{12}O_6$	Lactose Mg. $C_{12}H_{22}O_{11}.H_2O$	Maltose Mg. $C_{12}H_{22}O_{11}.H_2O$
352	396.3	440.6	124.0	117.7	229.9	227.3
353	397.4	441.9	124.3	118.1	230.6	227.9
354	398.6	443.1	124.7	118.4	231.2	228.6
355	399.7	444.4	125.1	118.8	231.9	229.2
356	400.8	445.7	125.5	119.2	232.6	229.9
357	401.9	446.9	125.9	119.5	233.2	230.6
358	403.1	448.1	126.3	119.9	233.9	231.2
359	404.2	449.4	126.7	120.3	234.6	231.9
360	405.3	450.6	127.1	120.7	235.3	232.6
361	406.4	451.9	127.5	121.0	236.0	233.2
362	407.6	453.1	127.9	121.4	236.6	233.9
363	408.7	454.4	128.2	121.8	237.3	234.5
364	409.8	455.6	128.6	122.1	238.0	235.2
365	410.9	456.9	129.0	122.5	238.7	235.9
366	412.1	458.1	129.4	122.9	239.4	236.5
367	413.2	459.4	129.8	123.2	240.1	237.2
368	414.3	460.6	130.2	123.6	240.7	237.8
369	415.4	461.9	130.6	124.0	241.4	238.5
370	416.6	463.1	131.0	124.3	242.1	239.2
371	417.7	464.4	131.4	124.7	242.8	239.8
372	418.8	465.6	131.8	125.1	243.5	240.5
373	420.0	466.9	132.1	125.5	244.1	241.2
374	421.1	468.1	132.5	125.8	244.8	241.8
375	422.2	469.4	132.9	126.2	245.5	242.5
376	423.3	470.6	133.3	126.6	246.2	243.1
377	424.5	471.9	133.7	127.0	246.8	243.8
378	425.6	473.0	134.1	127.4	247.5	244.5
379	426.7	474.3	134.5	127.7	248.2	245.1
380	427.8	475.6	134.9	128.1	248.9	245.8
381	429.0	476.8	135.3	128.5	249.6	246.5
382	430.1	478.1	135.7	128.8	250.2	247.1
383	431.2	479.3	136.1	129.2	250.9	247.8
384	432.3	480.6	136.5	129.6	251.6	248.5
385	433.5	481.8	136.9	130.0	252.3	249.1
386	434.6	483.1	137.3	130.3	253.0	249.8
387	435.7	484.3	137.7	130.7	253.6	250.4
388	436.8	485.6	138.0	131.1	254.3	251.1
389	438.0	486.9	138.4	131.5	255.0	251.8
390	439.1	488.2	138.8	131.9	255.7	252.4
391	440.2	489.4	139.2	132.2	256.4	253.1
392	441.3	490.7	139.6	132.6	257.0	253.8
393	442.4	491.9	140.0	133.0	257.7	254.4

TABLE IV (*continued*)

Copper Mg.	Cuprous oxide Mg.	Cupric oxide Mg.	Glucose Mg. $C_6H_{12}O_6$	Invert sugar Mg. $C_6H_{12}O_6$	Lactose Mg. $C_{12}H_{22}O_{11}.H_2O$	Maltose Mg. $C_{12}H_{22}O_{11}.H_2O$
394	443.6	493.2	140.4	133.4	258.4	255.1
395	444.7	494.4	140.8	133.8	259.1	255.8
396	445.9	495.7	141.2	134.2	259.8	256.4
397	447.0	496.9	141.6	134.5	260.5	257.1
398	448.1	498.2	142.0	134.9	261.1	257.8
399	449.2	499.5	142.4	135.3	261.8	258.4
400	450.3	500.7	142.8	135.7	262.5	259.1
401	451.5	502.0	143.2	136.1	263.2	259.8
402	452.6	503.2	143.6	136.4	263.9	260.4
403	453.7	504.5	144.0	136.8	264.5	261.1
404	454.8	505.7	144.4	137.2	265.2	261.8
405	456.0	507.0	144.8	137.6	265.9	262.4
406	457.1	508.2	145.2	137.9	266.6	263.1
407	458.2	509.5	145.6	138.3	267.3	263.8
408	459.4	510.7	146.0	138.7	267.9	264.4
409	460.5	512.0	146.4	139.1	268.6	265.1
410	461.6	513.2	146.8	139.5	269.3	265.8
411	462.7	514.5	147.2	139.9	270.0	266.4
412	463.8	515.7	147.6	140.2	270.7	267.1
413	465.0	517.0	148.0	140.6	271.4	267.8
414	466.1	518.2	148.4	141.0	272.1	268.4
415	467.2	519.5	148.8	141.4	272.8	269.1
416	468.4	520.7	149.2	141.8	273.5	269.7
417	469.5	522.0	149.6	142.2	274.1	270.4
418	470.6	523.2	150.0	142.6	274.8	271.1
419	471.8	524.5	150.4	143.0	275.5	271.8
420	472.9	525.7	150.8	143.3	276.2	272.4
421	474.0	527.0	151.2	143.7	276.9	273.1
422	475.1	528.2	151.6	144.1	277.6	273.8
423	476.2	529.5	152.0	144.5	278.3	274.4
424	477.4	530.7	152.4	144.9	278.9	275.1
425	478.5	532.0	152.8	145.3	279.6	275.8
426	479.6	533.2	153.2	145.7	280.3	276.5
427	480.7	534.5	153.6	146.1	280.9	277.1
428	481.9	535.7	154.0	146.4	281.6	277.8
429	483.0	537.0	154.4	146.8	282.3	278.5
430	484.1	538.2	154.8	147.2	282.9	279.1
431	485.3	539.5	155.3	147.6	283.6	279.8
432	486.4	540.7	155.7	148.0	284.3	280.5
433	487.5	542.0	156.1	148.4	285.0	281.1
434	488.6	543.2	156.5	148.8	285.7	281.8
435	489.7	544.5	156.9	149.1	286.4	282.5

TABLE IV (*continued*)

Copper Mg.	Cuprous oxide Mg.	Cupric oxide Mg.	Glucose Mg. $C_6H_{12}O_6$	Invert sugar Mg. $C_6H_{12}O_6$	Lactose Mg. $C_{12}H_{22}O_{11}.H_2O$	Maltose Mg. $C_{12}H_{22}O_{11}.H_2O$
436	490.9	545.7	157.3	149.5	287.1	283.1
437	492.0	547.0	157.7	149.9	287.8	283.8
438	493.1	548.2	158.1	150.3	288.5	284.5
439	494.3	549.5	158.5	150.7	289.2	285.2
440	495.4	550.7	158.9	151.1	289.9	285.8
441	496.5	552.0	159.3	151.5	290.6	286.5
442	497.6	553.2	159.8	151.9	291.3	287.2
443	498.8	554.5	160.2	152.3	292.0	287.8
444	499.9	555.7	160.6	152.7	292.7	288.5
445	501.0	557.0	161.0	153.1	293.4	289.2
446	502.1	558.2	161.4	153.5	294.1	289.8
447	503.2	559.5	161.8	153.9	294.8	290.5
448	504.4	560.7	162.2	154.3	295.5	291.2
449	505.5	562.0	162.6	154.7	296.2	291.9
450	506.6	563.3	163.0	155.1	296.9	292.5

ATROPIN THERAPY IN DIABETES MELLITUS.*†

HERMAN O. MOSENTHAL, M.D.

The atropin therapy of diabetes mellitus has recently been advocated by Rudisch[1] and by Forchheimer.[2] Carbohydrate tolerance is, according to Rudisch, greater with atropin than without. The more striking results were obtained with atropin sulphate, though in some instances atropin methyl bromid was substituted.

The following two cases of diabetes mellitus were tested out with atropin sulphate. A constant weighed diet, as detailed in the protocols, was given. When the daily variations in the amount of glucose excreted had been reduced to a minimum, atropin sulphate was administered in increasing doses for a sufficiently long period of time to give the drug a chance to exert any action of which it was capable. With both subjects the experiments were continued until toxic effects were observed. Under these conditions the glycosuria should be diminished if atropin sulphate is capable of increasing the carbohydrate tolerance.

Any drug which modifies the carbohydrate metabolism may influence the formation of the so-called acid bodies, acetone, diacetic acid and beta-oxybutyric acid. If atropin diminished the utilization of glucose within the organism, the acid bodies would increase in the urine. Therefore there is some interest attached to the determination of these substances in a study of this kind. In one of the cases observed, small but persistent amounts of acetone and diacetic acid were present in the urine, as shown by previous qualitative tests. Any possible increase in these substances was measured by the ammonia output and its relation to the total nitrogen of the urine. Normally the amount of ammonia nitrogen excreted is about 5 per

*From the service of Dr. F. C. Wood, St. Luke's Hospital, New York.

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[1]Rudisch, J.: The Journal A. M. A., Oct. 23, 1909, p. 1366.

[2]Forchheimer, F.: Am. Jour. Med. Sc., 1911, cxli, 157.

cent of the total nitrogen. A rise in this percentage indicates an increase in the excretion of acid substances.

In these analyses the glucose was determined by Benedict's method,[3] the ammonia according to Folin, and the nitrogen by the Kjeldahl process. The presence or absence of acetone was established by the Legal reaction, that of diacetic acid by the ferric chlorid test.

TABLE 1.—URINE ANALYSES AND MEDICATION IN A CASE OF DIABETES MELLITUS (CASE 1).

	Urine in Twenty-four Hours.								
Date, 1911.	Volume, c.c.	Glucose, Per Cent.	Glucose, gm.	Total Nitrogent, gm.	Ammonia, gm.	Ammonia N. in Per Cent. of Total N.	Atropin Sulphate, Grains per Dose.	Doses per Day.	Symptoms.
3/18	1,460	2.2	23.4	9.1	.34	3.1*	0	.	
3/19	1,290	2.4	30.3	9.6	.45	3.9*	0	.	
3/20	1,720	2.8	47.5	8.9	.55	5.1	0	.	
3/21	1,680	1.9	31.1	9.9	.58	4.9	0	.	
3/22	1,080	2.1	22.9	7.7	.48	5.1	0	.	
3/23	1,200	2.0	23.5	9.7	.58	4.9	1/100	3	
3/24	1,320	2.0	26.7	9.8	.55	4.6	1/100	3	Mouth slightly dry.
3/25	1,800	1.5	26.7	9.9	.63	5.2	1/100	3	Cheeks flushed.
3/26	1,910	1.5	29.0	12.2	.79	5.4	1/100	4	
3/27	1,560	1.2	18.4	9.7	.57	4.9	1/50	3	
3/28	1,300	1.6	20.2	9.2	.65	5.8	1/50	3	
3/29	1,480	0.6	8.7	5.5	.43	6.5	1/50	3	
3/30	1,300	0.8	13.4	7.0	.54	6.3	1/25	3	Vertigo; very dim vision.
3/31	1,940	1.6	30.3	10.1	.81	6.6	1/25	2	
4/1	1,560	1.3	19.5	10.1	.66	5.4	1/25	2	Head "feels full."
4/2	1,730	1.9	32.0	10.1	.75	6.1	0	.	
4/3	1,200	2.9	35.4	8.7	.59	5.6	0	.	
4/4	930	1.8	17.1	7.8	.59	6.2	0	.	
4/5	1,175	2.1	24.9	12.6	.76	5.0	0	.	

*The initial low figures for ammonia may be regarded as the after-effects of bicarbonate of soda taken before admission to the hospital.

[3]Benedict, S. R.: The Journal A. M. A., Oct. 7, 1911, p. 1193.

TABLE 2.——RECORD OF URINE ANALYSES AND MEDICATION IN A CASE OF DIABETES MELLITUS (CASE 2).

	Urine in Twenty-four Hours.					
Date, 1911.	Volume, c.c.	Glucose, Per Cent.	Glucose, gm.	Atropin Sulphate, Grains per Dose.	Doses per Day.	Symptoms.
8/9	1,000	1.0	9.6	0	.	
8/10	740	1.1	7.8	0	.	
8/11	1,150	1.1	12.2	0	.	
8/12	860	1.0	8.6	0	.	
8/13	810	1.1	8.8	0	.	
8/14	.....	..	...	1/100	3	
8/15	980	0.6	6.2	1/100	3	Mouth slightly dry.
8/16	.....	..	...	1/100	3	
8/17	1,085	0.7	7.2	1/100	3	
8/18	1,145	0.7	8.1	1/100	3	Vision dim temporarily.
8/19	1,095	0.8	9.1	2/100	3	
8/20	1,550	0.5	8.2	2/100	3	
8/21	1,485	0.3	4.6	2/100	3	Mouth very dry.
8/22	1,340	0.3	4.0	3/100	3	
8/23	1,150	0.4	4.1	3/100	3	
8/24	1,690	1.0	16.6	3/100	3	
8/25	1,155	1.0	11.6	4/100	3	
8/26	1,800	1.7	29.9	4/100	3	
8/27	1,030	1.3	12.9	5/100	3	Face flushed.
8/28	2,360	0.4	9.9	5/100	3	
8/29	1,920	0.7	13.6	6/100	3	
8/30	1,720	0.4	7.1	6/100	3	
8/31	1,600	0.5	7.2	7/100	3	Mouth intensely dry.
9/1	865	1.0	8.3	7/100	3	Pupils dilated.
9/2	1,430	0.4	6.0	7/100	3	
9/3	1,300	0.5	5.9	0	.	
9/4	1,400	2.3	31.8	0	.	
9/5	1,045	1.1	11.1	0	.	
9/6	1,400	1.8	25.2	0	.	

Case 1.—Patient, a tailor, of Russian birth, aged 38, about two years ago began to suffer with polyuria, thirst, increased appetite and loss of weight. Glucose was discovered in the urine, but dietary restrictions were never observed for very long periods of time. After being treated for one month at the Vanderbilt Clinic as an out-patient, he was sent to Dr. Wood's service at St. Luke's Hospital. The patient's urine gave constant positive reactions of moderate intensity for acetone and diacetic acid. He was placed on the following diet:

Breakfast.—Coffee or tea, with 1½ ounces of cream; two eggs, cooked with ½ ounce butter; 3 ounces ham; one slice bread, weight exactly 1 ounce, with ¼ ounce butter.

Lunch.—Bouillon, with one raw egg; 3 ounces any lean meat, 1 ounce

bacon; vegetables from list,[4] 3 ounces, with ½ ounce butter or oil; 1 ounce whisky or brandy; one slice bread, weight exactly 1 ounce, with ¼ ounce butter.

Afternoon tea, with ½ ounce cream.

Dinner.—Any clear soup; 4 ounces any lean meat; vegetables from list,[4] 3 ounces, with ½ ounce butter or oil; 1 ounce cheese, English, pineapple, Swiss, or full-cream cheese; one slice white bread, weight exactly 1 ounce, with ¼ ounce butter; 1 ounce whiskey or brandy; demitasse coffee.

Case 2.—Patient, a native of France, aged 59, foreman in a factory, was found to have sugar in his urine about four years ago. Only during the last year before examination had he been complaining of diabetic symptoms: occasional increased appetite and thirst. There had been some stiffness, pain and weakness in the legs. The neurologic department of the Vanderbilt Clinic diagnosed the case as one of multiple neuritis of diabetic origin. The urine occasionally showed a trace of acetone, but no diacetic acid. After being observed for one month at the Vanderbilt Clinic, he was sent to St. Luke's Hospital. He was given the same diet as the patient in Case 1, except that 2 ounces of bread were ordered with each meal instead of 1.

The above reports give no indication that atropin sulphate effects any change in the carbohydrate tolerance of sufficient importance to make the drug of clinical value in the treatment of diabetes mellitus.

[4]Vegetables allowed were: Asparagus, beet greens, Brussels sprouts, cabbage, cauliflower, celery, chicory, cresses, cucumbers, egg plant, endive, lettuce, mushrooms, radishes, rhubarb, salsify, spinach, string beans, tomatoes, vegetable marrow.

ANATOMICAL STUDY OF A THORACOPAGUS.

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From the Pathological Department.

F. C. Wood, M.D., Director.

This specimen was sent to the laboratory from the service of Dr. C. L. Gibson, in February, 1911. Some one had named the twins "John and Mary," evidently forgetting his embryology, for they are, of course, identical or homologous twins; *i.e.,* have developed in the same amniotic sac and have resulted probably from the division of a single ovum. In such cases, there may be produced two separate individuals, *i.e.,* normal twins of like sex; or, on the other hand, there may be formed, in some way, a double monster.

We may classify double monsters in two main classes: first, those showing decidedly unequal development; for example, the case of a more or less completely developed autosite having an acardiac parasite springing from its thorax; and, second, those showing practically equal development. This second class may still further be divided (according to Adami) into two classes: first, those that have resulted from cleavage in the very early embryo—at the superior pole, at the inferior pole, at both, or between these poles; *i.e.,* if cells of the head center become split, those cells developing from them at each side would form duplicate sets of tissue, a double head, for instance; whereas the cells of the rump center developing in the normal way would produce a single body. (Examples of superior duplication would thus include all varieties, from a monster having two heads, four arms, a double trunk, and two legs, down to a case simply of bifurcation of the hypophysis of the brain.) His second class includes those that he believes have resulted from fusion of what would otherwise have become identical twins; and these may be subdivided according to the location and extent of this fusion.

The specimen we have to consider belongs to this latter class, and it may be termed an equal monosymmetrical thoracopagus. The term equal is used because the twins show practically the same measure-

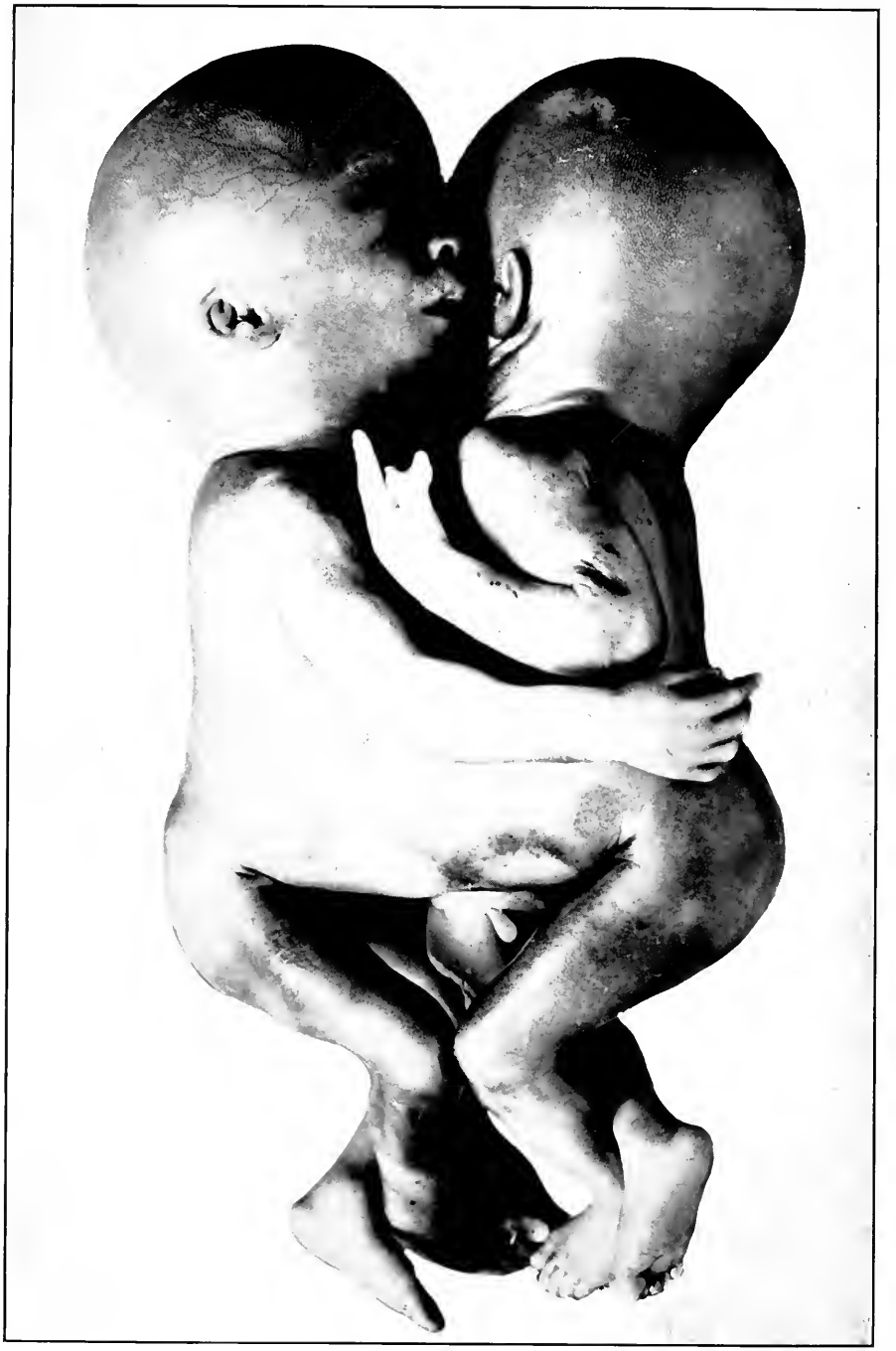

Fig. 1.—The specimen before dissection, showing the double thumb.

ments. They are monosymmetrical because, as is more often the case, the union is not exactly face to face; *i.e.*, the arms, for example, are somewhat farther apart on one side than on the other because the antero-posterior planes of the fetuses do not exactly coincide. The term thoracopagus is really not exact because the union includes not only the thorax but the upper part of the abdomen as well, for it will be noticed in the photograph taken before dissection that the cord springs from the under surface of the bond of union. (The same picture shows also the double thumb in one fetus which Nature added as a finishing touch to an already interesting specimen.)

The age of the twins may be estimated at about four months, to judge from their development; the mother's last menstruation occurred October 14, 1910, and she aborted February 2, 1911.

In order to show the relations of the viscera, one side of the specimen (which, for convenience, we may speak of as the "front") was removed completely. In dissecting up the skin, the recti muscles were exposed and found to extend from the lower ribs downward and outward to each pubis. By making translucent the portion of chest-wall removed, we found that instead of the sternum of each side coming in contact face to face (as, at first glance, we might expect), each sternum had split, the corresponding parts on each side, *i.e.*, the front and back of the specimen, forming a separate sternum.

The thorax, therefore, is common; it contains a single pericardium inclosing one heart having five chambers. The apex of the heart, as we look at the specimen, points directly forward. The auricles lie at about the same level, so that the long axis of the heart is in a horizontal plane. Two aortæ leave the two left ventricles, the arch in each fetus taking the normal direction. Two inferior venæ cavæ pierce the diaphragm and enter a common right auricle. This is continuous with a common left auricle. Into this common chamber enters also a pulmonary vein from the right fetus. (The other pulmonary veins have not been followed.) The course taken by the blood seems to have been as follows: entering the common right auricle by the inferior and superior venæ cavæ, it reached the common left auricle, and thence to the two left ventricles and the two aortæ to the arterial system, but also partly back to the lungs through the ductus arteriosus of each fetus. Some left the common right auricle to enter the single right ventricle, then through the pulmonary artery of the right fetus to the lungs on that side. The pulmonary artery of the left fetus appears to be represented only by a fibrous cord which extends

to the root of the aorta (there being no right ventricle for that fetus), so that its lungs received blood only from its aorta by way of the ductus arteriosus.

The lungs show the usual divisions into lobes. The left lung of the right fetus presents a cardiac incisure which receives the apex of the heart. The left lung of the left fetus is abnormal in that a lobe hooks over the aorta, and there is, in addition, a groove accommodating the left superior vena cava, which runs down between the outer surface of the left lung and the chest wall.

We come next to the diaphragm, which is single and arches over a single large liver. The upper part of the liver has in the median line a shallow groove, from which a thin fibrous partition extended to the abdominal wall. The umbilical vein is single and enters the liver through a deep notch situated at about the center of this "front" surface. Above and below this the liver is continuous across. At each side of this opening there is a deep horizontal cut, as if to represent what would have been an umbilical fissure in each liver. The umbilical vein, followed into the substance of the liver, divides into two branches: that at the left becomes lost by smaller subdivisions, but the right branch also connects by a distinct branch with the right inferior vena cava, this connection being a ductus venosus.

The intestinal tract is very interesting. As the abdominal wall was opened, a small, pointed pouch, about half a centimeter long, was seen adherent to the inner surface of the cord. It is the remnant of the vitelline duct, or a Meckel's diverticulum. This opens into a horizontal loop of gut and is located at a Y-shaped junction formed by what we may call a common jejunum meeting the two ilea. That is to say, below this point each fetus has its own intestinal tract. The cecum and appendix are located at the left side in the right fetus and slightly to the right of the median line in the left fetus. Following upward this common jejunum, we find that it continues single almost up to the stomachs, where it joins a horizontal loop made up of the two short duodena. This is shown in the diagram of the intestinal tract.

We see, then, that in regard to the two important systems, viz., the circulatory and the alimentary, the twins are very closely connected. The viability of such a monster, even if it could have been delivered at term, seems very unlikely. We have spoken of the specimen as the "Siamese twins," but the two cases are really not very similar, since the Siamese twins were joined only by a band which

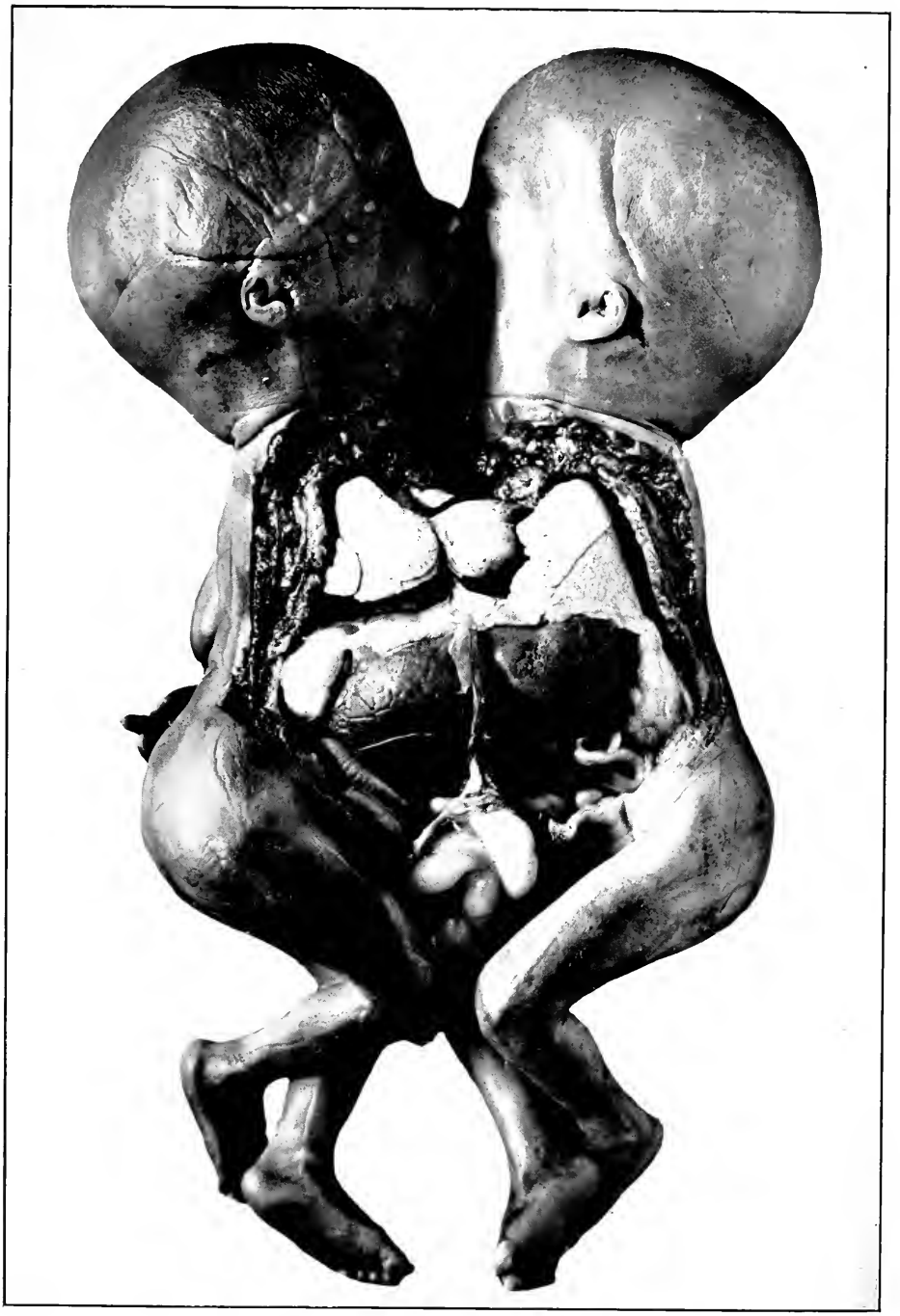

Fig. 2.—The dissection of thorax and abdomen.

Fig. 3.—The liver; the Meckel's diverticulum (just below and in the median line).

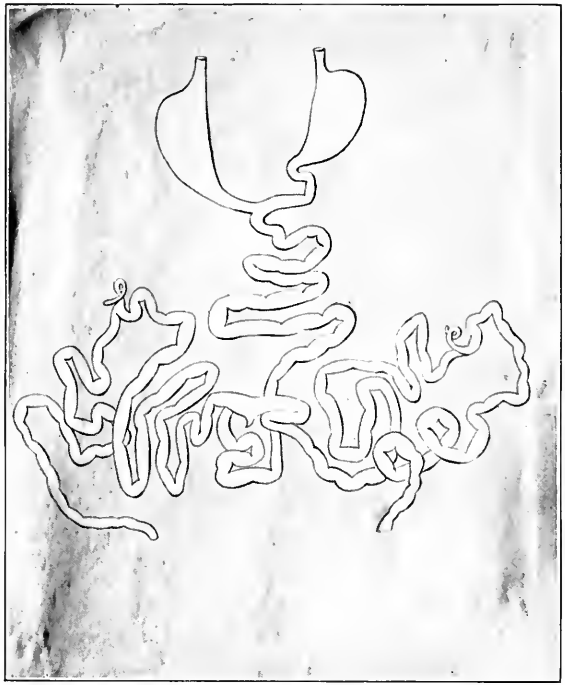

Fig. 4.– Diagram of the intestinal tract; the Meckel's diverticulum in the center, at the junction of three loops of gut.

measured a few inches in diameter when they had reached adult life. It contained, however, a narrow band of liver. They lived to be sixty-three years old.

The underlying causes of the production of such monsters are, of course, far from settled. Experimental teratology surely is an interesting study, but so far it has been confined chiefly to lower animals, *e.g.*, the production of double tadpoles by tying a fine thread about the egg, or of spina bifida and cyclopia by subjecting normal fish-eggs to the action of sodium salts. These and similar experiments tend to show that the production of monsters is the result of external causation. It is believed that the same holds good for human pathological embryos and monsters.

F. P. Mall calls attention to the fact that whereas only 0.7 per cent of all pregnancies end in the production of pathological ova, in tubal pregnancies 96 per cent become pathological or produce monsters; and the data of von Winkel of 87 live fetuses removed from ruptured tubal pregnancies show that only 8 were really normal.

Professor Mall concludes from this and from experimental teratology that the cause of the production of monsters is not germinal, *i.e.*, inherent in the ovum or sperm, but is the result of external influences. His 33 cases of pathological human ova, he finds, can be classified in three groups: in the first group of 11 cases, a severe hemorrhage for several days preceded the abortion; the second group of 12 specimens were abortions from newly married women or relatively sterile women who had been married for some time; and the third group of 10 specimens were from women who had given birth to a number of healthy children and then began to abort, often two or three times. This last group showed that the cause could not have been germinal, because these women had previously borne normal children. The explanation is rather that the uterus was at first normal, but later became pathological, so that the fertilized ovum could not implant itself properly, but was aborted.

In this connection it is interesting to note that the case we have been considering was that of a woman, aged 33, married 7 years, who had had two children, followed by three abortions, the last of which was preceded by a metrorrhagia lasting three months. And it may be added that this patient returned to the hospital in January, 1912, about a year after her last abortion, presenting again the symptoms of threatened abortion, but left after a week, the pregnancy not having been interrupted.

REPORT OF THE PATHOLOGICAL DEPARTMENT OF ST. LUKE'S HOSPITAL FOR THE YEAR 1911.

F. C. Wood, M.D., Director.

The following abbreviated report gives the statistical results of the work done in the various laboratories of the department during the year.

DIVISION OF SURGICAL PATHOLOGY.

In the course of the year 762 specimens of tissue were examined histologically. The diagnoses were as follows:

TUMORS.

Adenocarcinoma of abdomen	1
Adenocarcinoma of breast	1
Adenocarcinoma of colon	3
Adenocarcinoma of ovary	1
Adenocarcinoma of rectum	6
Adenocarcinoma of stomach	1
Adenocarcinoma of uterus	5
Adenomyoma of uterus	1
Carcinoma of antrum	1
Carcinoma of abdominal wall	1
Carcinoma of breast	34
Carcinoma of cervix uteri	2
Carcinoma of jaw	1
Carcinoma of lymph nodes	2
Carcinoma of neck	1
Carcinoma of omentum	2
Carcinoma of ovary	5
Carcinoma of peritoneum	1
Carcinoma of prostate	1
Carcinoma of rectum	2
Carcinoma of sternum	1
Carcinoma of stomach	1
Carcinoma of tonsil	1

TUMORS—Cont.

Carcinoma, gelatinous, of caput coli	1
Carcinoma, gelatinous, of omentum	1
Carcinoma, gelatinous, of ovary	1
Carcinoma, gelatinous, of rectum	3
Carcinoma, squamous cell, of urinary bladder	1
Cystadenoma of breast	6
Cystadenoma of ovary	3
Cystoma of ovary	1
Cystoma of peritoneum	1
Cystoma, multilocular mucinous, of ovary	1
Epithelioma of ala nasi	1
Epithelioma of cervix uteri	7
Epithelioma of cheek and face	6
Epithelioma of conjunctiva	1
Epithelioma of esophagus	1
Epithelioma of eyelid	2
Epithelioma of groin	1
Epithelioma of hand	2
Epithelioma of jaw	2
Epithelioma of larynx	1
Epithelioma of lip	9
Epithelioma of lymph nodes	5
Epithelioma of mouth	1
Epithelioma of neck	2
Epithelioma of pharynx	1
Epithelioma of scalp	1
Epithelioma of tongue	4
Epithelioma of vulva	1
Epithelioma, basal cell, of face	4
Epithelioma, basal cell, of eyelid	2
Fibroadenoma of breast	7
Fibroadenoma of prostate	1
Fibroadenoma, intracanalicular, of breast	2
Fibroma of thigh	1
Fibroma, soft, of hand	1
Fibromyoma of uterus	75
Fibrosarcoma of femur	1
Fibrosarcoma of small intestine	1
Hemangioma of face	1
Lipoma of arm	1
Lipoma of back	1
Lipoma of chest	1
Lipoma of neck	1
Lipoma of knee	1
Lipoma of thigh	1
Myxo-fibroma of ulnar nerve	1

TUMORS—Cont.

Neuro-fibro-lipoma	1
Osteoma of palate	1
Papilloma of bladder	1
Papilloma of face	1
Polyp of cervix uteri	3
Polyp of endometrium	1
Sarcoma of abdomen	1
Sarcoma of orbit	1
Sarcoma of pelvis	1
Sarcoma of spinal cord and vertebræ	1
Sarcoma, giant cell, of jaw	1
Sarcoma, lympho-, of cervical nodes	1
Sarcoma, lympho-, of neck	1
Sarcoma, lympho-, of tonsil	1
Sarcoma, lympho-, of thorax	1
Sarcoma, melano-, of ovary, breast and peritoneum	1
Sarcoma, myxo-, of foot	1
Sarcoma, osteo-, of tibia	1

CYSTS.

Dermoid cyst of ovary	7
Epidermoid cyst of neck	1
Follicular cyst of ovary	2
Parovarian cyst	4
Perinephritic cyst	1
Sebaceous cyst of scalp	1
Strangulated ovarian cyst	1

MISCELLANEOUS TISSUES.

REPRODUCTIVE SYSTEM—FEMALE.

Corpus luteum	1
Decidua and Chorionic Villi	18
Ectopic gestation	10
Endocervicitis, glandular	7
Endometrium, normal	9
Endometrium, atrophy of	3
Endometrium, hyperplasia of	21
Endometrium, edema of	6
Endometrium, menstrual	4
Endometrium, premenstrual	2
Endometritis, chronic	4
Endometritis, interstitial	6
Fallopian tubes, normal	11

REPRODUCTIVE SYSTEM—FEMALE—Cont.

Fallopian tubes, atrophy of	2
Hematosalpinx	1
Galactocele	1
Hydrosalpinx	2
Mastitis, acute	1
Mastitis, chronic	3
Mastitis, tuberculous	1
Oophoritis, subacute	1
Oophoritis, chronic	42
Ovary, normal	2
Pyosalpinx	13
Salpingitis, acute	7
Salpingitis, chronic	20
Salpingitis, subacute	10
Salpingitis, tuberculous	2
Salpingo-oophoritis, acute	6
Salpingo-oophoritis, chronic	26
Salpingo-oophoritis, subacute	7
Sinus of breast	1
Tubo-ovarian abscess	5
Uterus, tubes and ovaries, tuberculosis of	1

REPRODUCTIVE SYSTEM—MALE.

Epididymis, tuberculosis of	2
Prostate, adenomatous hyperplasia of	1
Prostate, hypertrophy of	2
Prostatitis, subdurative	1
Testicle, tuberculosis of	2

GASTROINTESTINAL SYSTEM.

Appendicitis, acute	15
Appendicitis, catarrhal	4
Appendicitis, chronic	26
Appendicitis, subacute	6
Appendicitis, peri-	1
Appendicitis, tuberculous	1
Appendix, normal	3
Cholecystitis, acute	2
Cholecystitis, chronic	6
Cholecystitis, subacute	2
Colitis, acute	1
Peritonitis, acute	4
Peritonitis, subacute	1
Peritonitis, tuberculous	2

GASTROINTESTINAL SYSTEM—Cont.

Thrombosed vein of intestine	1
Tonsil, chronic inflammation of	3
Tonsil, normal	1
Tonsil, tuberculosis of	1

URINARY SYSTEM.

Kidney, multiple abscesses of	1
Kidney, tuberculosis of	3
Hemorrhage into kidney, site not discovered	1
Hydronephrosis	2
Pyonephrosis	1
Nephritis, suppurative	3

BONES AND JOINTS.

Osteitis	1
Osteitis, productive	1
Osteitis, rarefying and productive	1
Osteomyelitis, chronic	4
Osteomyelitis, tuberculous	3
Synovitis, chronic	1
Tuberculosis of carpal bones	1
Tuberculosis of chest wall	1
Tuberculosis of femur	1
Tuberculosis of knee	1

LYMPH NODES

Adenitis, simple	4
Adenitis, tuberculous	29
Nodes, chronic hyperplasia of	3
Nodes, normal	1

MISCELLANEOUS.

Abscesses of liver, miliary	1
Actinomycosis of abdomen	1
Blood clot	6
Cartilage	1
Connective tissue	8
Connective tissue, inflamed	11
Corneal ulcer	1
Endarteritis, with gangrene of foot	1
Fibrin	3
Furuncle	1

MISCELLANEOUS—Cont.

Gangrene of thumb, diabetic	1
Goitre, colloid	8
Goitre, exophthalmic	1
Granulation tissue, simple	11
Granulation tissue, tuberculous	4
Hemorrhoids, inflamed granulation tissue	1
Iridocyclitis	1
Mucous membrane, normal	1
Myositis	3
Nasal polyp	2
Panophthalmitis, chronic	1
Pigmented mole of abdomen	1
Salivary glands, normal	1
Sebaceous cyst, chronic inflammation of	1
Tuberculosis of intercostal tissue	1
Ulcer of leg	1

POST-MORTEM EXAMINATIONS.

During the past year fifty-three autopsies have been performed. Several of the more interesting cases are reported at length elsewhere.

840. Anatomical Diagnosis: Acute aortitis. Chronic fibrous myocarditis, with cardiac hypertrophy and dilatation, and relative mitral and tricuspid insufficiency. Healed tuberculosis of lungs, with passive congestion. Chronic diffuse nephritis. Chronic passive congestion of liver and spleen.

841. Case of sudden death, a child, twelve years of age, in the hospital for tuberculosis of spine, hip and both knees. Besides the above tuberculous conditions, the autopsy showed a very large thymus extending from the thyroid gland to within one inch of lower border of heart. The left lateral lobe passed down over the left side of heart in a thin, flat layer. The mesenteric and transverse mesocolic nodes were enlarged, and there was hyperplasia of the lymphoid nodules throughout the intestine.

842. Anatomical Diagnosis: Subdural hemorrhage. Multiple cerebral and cerebellar hemorrhages. General arteriosclerosis. Coronary sclerosis. Cardiac hypertrophy. Healed tuberculosis of lungs. Chronic adhesive pleurisy. Chronic diffuse nephritis.

843. Anatomical Diagnosis: Epithelioma of cervix, with extension to pelvic and inguinal lymph nodes and left iliac vein. Metastases to spleen and lung. Thrombosis of cerebral veins and softening of right hemisphere. Double hydrothorax. Anemia of viscera.

844. Anatomical Diagnosis: Acute fibrino-purulent pericarditis and empyema. Acute bronchopneumonia. General lymphatic hyperplasia. Cloudy swelling of liver and kidneys, with congestion. Acute splenic tumor. Culture from pericardial exudate showed pneumococcus.

845. Case of corrosive sublimate poisoning. Partial autopsy. Anatomical diagnosis: Acute parenchymatous nephritis.

846. Anatomical Diagnosis: Chronic mitral endocarditis, with acute exacerbation. Auricular thrombosis. Hypertrophy of heart, dilatation of auricles. Infarction of lung. Hydrothorax. Atelectasis of lung. Healed tuberculosis of lungs. Acute ulcerative aortitis. Chronic diffuse nephritis (chiefly parenchymatous). Chronic passive congestion of liver, spleen and intestine. Chronic gastritis. Chronic interstitial pancreatitis. Edema of cerebral pia mater. Cystic degeneration of left lenticular nucleus (old softening).

847. Anatomical Diagnosis: Chronic diffuse nephritis. Cardiac hypertrophy. Edema of lungs. Ulcerative laryngitis and pharyngitis. Acute splenic tumor. Fatty degeneration of liver. Chronic cystitis.

848. Anatomical Diagnosis: Chronic interstitial nephritis of severe grade. Practically no other changes. Moderate hypertrophy of left ventricle.

849. Anatomical Diagnosis: Tuberculous meningitis. General miliary tuberculosis. Perforating appendicitis. General purulent peritonitis.

850. Anatomical Diagnosis: Lobar pneumonia of right upper, middle, and part of lower lobe. Congestion and chronic tuberculosis of both lungs. Chronic adhesive pleuritis and pericarditis. Cloudy swelling of liver and kidneys. Edema of pia.

851. Anatomical Diagnosis: Arteriosclerosis. Cylindrical aneurism of aorta. Chronic myocarditis. Double hydrothorax. Congestion, edema, and healed tuberculosis of lungs. Passive congestion of liver, spleen and intestine. Slight chronic diffuse nephritis. Meckel's diverticulum.

852. Anatomical Diagnosis: False aneurism of aorta, with rupture into left pleura. Fusiform and dissecting aneurisms of aorta. Extreme aortitis. Edema and congestion of lungs. Subacute serofibrinous pleurisy. Aortic insufficiency and cardiac hypertrophy. Passive congestion of liver, spleen and kidneys.

853. Anatomical Diagnosis: Acute vegetative endocarditis, involving mitral, aortic and tricuspid valves. Free thrombus in right auricle. Acute serofibrinous pericarditis and pleurisy. Mitral insufficiency and dilatation of right auricle. Edema of lungs. Passive congestion of heart, liver, spleen and kidney.

Bacterial Diagnosis: Smears from mitral valve and from pericardium show Gram-positive diplococci resembling pneumococci. Cultures show similar organisms in mixed culture.

854. Partial Autopsy. Anatomical Diagnosis: Acute ulcerative colitis. Etiology not determined.

855. Anatomical Diagnosis: Chronic pulmonary tuberculosis. Cavity in right lower lobe. Acute mitral endocarditis. Acute splenic tumor. Chronic hyperplasia of lymph nodes. (Death occurred after diabetic coma.)

856. Anatomical Diagnosis: Chronic endocarditis, with ball thrombus in right auricle. Thrombosis of right vertebral artery, with softening in medulla. Infarct of spleen. Chronic passive congestion of lungs and liver.

857. Anatomical Diagnosis: Microgyria, with secondary external and internal hydrocephalus ex vacuo. Bronchopneumonia.

858. Partial Autopsy. Anatomical Diagnosis: Cholelithiasis of common duct. Bacillus aerogenes capsulatus infection of sinus and liver, and septicemia following cholecystectomy.

859. Anatomical Diagnosis: Bronchopneumonia. Fibrinopurulent pleuritis.

860. Anatomical Diagnosis: Chronic mitral endocarditis. Fatty degeneration of heart. Tuberculosis of bronchial nodes. Fatty degeneration of liver. Hydrosalpinx. Cystic ovaries. Fibromyoma of uterus.

861. Anatomical Diagnosis: Chronic fibrous pleuritis. Tuberculosis of the lungs. Tuberculosis of bronchial lymph nodes. Bronchopneumonia. Endocarditis, acute mitral. Chronic diffuse nephritis. Ulceration (typhoid) of ileum, cecum and colon. Hyperplasia of lymph nodules and Peyer's patches of ileum. Hyperplasia of mesenteric nodes. Congestion and hyperplasia of spleen.

862. Anatomical Diagnosis: Chronic fibrous pleurisy. Healed tuberculosis of lungs. Carcinoma of lesser curvature of stomach, with perforation. Metastases in liver, pancreas, mesenteric lymph nodes and sigmoid, involving bladder wall. Acute peritonitis. Chronic diffuse nephritis.

863. Anatomical Diagnosis: Acute colitis.

864. Anatomical Diagnosis: Bronchopneumonia. Acute enteritis.

865. Partial Autopsy: Glioma, with softening, of floor of fourth ventricle.

866. Anatomical Diagnosis: Acute vegetative endocarditis. Bronchopneumonia of left upper lobe. Cyst of brain partially replacing lenticular nucleus and anterior limb of internal capsule on right side. Cloudy swelling of kidneys.

867. Anatomical Diagnosis: Acute hemorrhagic pancreatitis. Acute cholecystitis. Multiple areas of old necrosis in and about the pancreas. Fatty degeneration of the liver. Tuberculosis of the liver.

868. Anatomical Diagnosis: Sarcoma of retroperitoneal region, with metastases in kidneys, lymph nodes, and subcutaneous tissue. Left pyonephrosis. Atrophy and dilatation of heart. Passive congestion of spleen and liver. Edema of lungs. Anasarca of legs and hips, due to blocking of inferior vena cava and left common iliac veins. Chronic cystitis. Compensatory hyperplasia of bone marrow.

869. Anatomical Diagnosis: Chronic fibrous pleurisy. Lobar pneumonia. Healed pulmonary tuberculosis. Chronic diffuse nephritis.

870. Anatomical Diagnosis: Umbilical hernia. Umbilical fistula. Ascites. Acute peritonitis. Cirrhosis of liver. Chronic passive congestion of spleen. Chronic diffuse nephritis. Chronic endocarditis. Aortic stenosis.

871. Anatomical Diagnosis: Acute ulcerative endocarditis of the aortic and mitral valves. Cardiac hypertrophy and dilatation. Hydropericardium. Double hydrothorax and ascites. Edema and chronic tuberculosis of lungs. Infarct of spleen. Parenchymatous degeneration of left kidney. Chronic passive congestion and hemangioma of liver. Chronic seminovesiculitis.

872. Partial Autopsy. Anatomical Diagnosis: Hyperplasia and ulceration of Peyer's patches and lymph follicles of ileum, cecum and colon (typhoid). Perforation of ileum. Hyperplasia of mesenteric nodes. General peritonitis.

873. Case of man 45 years of age, who had been troubled for nine months

previous to entering hospital with difficulty in swallowing, and pain in chest, of indefinite localization. He had lost nine pounds in two months. Three weeks after entrance, the patient vomited three ounces of blood, grew gradually weaker, and died in five hours. At autopsy, a tumor was found projecting into the esophagus from its anterior wall, about 1 cm. above the level of the bifurcation of the trachea, the lumen thus being narrowed so as just to admit the passage of the index finger. From this point to about 1 cm. above the cardiac orifice of the stomach, the entire mucosa and a considerable portion of the walls of the esophagus were destroyed, a large cavity being formed in the posterior mediastinum, bounded by soft necrotic tissue. At the level of the fourth intercostal artery the wall of the aorta, over an area about 2 cm. in diameter, was destroyed nearly to the intima. The fourth right intercostal artery was torn from the aorta, and its point of exit marked by a small perforation about 2 mm. in diameter, leading directly into the esophagus. The stomach contained one liter of clotted blood. There were metastases in the pancreas and liver. Microscopical examination showed the tumor to be an epithelioma.

874. Anatomical Diagnosis: Double hydrothorax. Pericarditis. Cardiac hypertrophy. Chronic endocarditis. Mural thrombus in right auricle. Rupture of chordæ of anterior cusp of mitral valve. Infarction of both lungs. Chronic adhesive peritonitis. Chronic passive congestion of liver and spleen.

875. Anatomical Diagnosis: Double hydrothorax. Acute and chronic endocarditis. Aortic stenosis and insufficiency. Cardiac hypertrophy. Chronic diffuse nephritis. Sclerosis of coronaries and aorta.

876. Anatomical Diagnosis: Chronic ulcerative colitis. Chronic parenchymatous nephritis. Left bronchopneumonia. Miliary abscesses of both lungs. Subacute cholecystitis. Multiple ulcers of skin.

877. Partial Autopsy. Anatomical Diagnosis: Operative skull defect. Local meningitis. New growth of cerebellum and cyst communicating with aqueduct of Sylvius. Compression of fourth ventricle. Internal hydrocephalus.

878. Anatomical Diagnosis: Tuberculosis of lungs. Chronic adhesive pleurisy. Miliary tuberculosis of liver and spleen. Chronic diffuse nephritis. Thrombosis of right femoral vein. Arteriosclerosis.

879. Case of a woman 23 years of age, entering hospital in moribund condition. No history was obtained, except that she had had a headache and backache for eight days, with temperature varying from 101° to 103°. She is said to have coughed considerably for some time, the expectoration being at times bloody, but never to have had heart trouble until three weeks before, when she began to complain of shortness of breath. The autopsy findings were interesting on account of the extreme grade of congenital pulmonary stenosis, the orifice, 2.7 cm. in circumference, barely admitting the tip of the little finger. The right auricle and ventricle were greatly hypertrophied, the right ventricular wall measuring 2.3 cm. in thickness. The left auricle and ventricle were both small. The left ventricular wall measured 1.5 cm. Neither the foramen ovale nor the ductus arteriosus were patent. There were three small, apparently recent, vegetations on one cusp of the pulmonary valve. The lungs showed healed tuberculous lesions and two areas of infarction in

the right lower lobe and one in the left. The liver and spleen showed the effects of chronic passive congestion.

880. Anatomical Diagnosis: Chronic endocarditis. Mitral stenosis. Cardiac hypertrophy and dilatation. Edema, ascites and double hydrothorax. Chronic passive congestion of liver, spleen and kidneys.

881. Partial Autopsy. Anatomical Diagnosis: Tuberculous enteritis and localized peritonitis. Amyloid degeneration of spleen. Chronic parenchymatous degeneration of spleen. Passive congestion of liver.

882. Anatomical Diagnosis: Carcinoma of stomach. Metastases in liver, spleen, retroperitoneal and posterior mediastinal lymph nodes. Mural thrombus of left ventricle. Septic thrombus of right pulmonary artery, with septic infarct of lung and acute fibrinopurulent pleurisy. Left hydrothorax. Edema of lungs. Chronic diffuse nephritis.

883. Anatomical Diagnosis: Edema of legs. Right hydrothorax. Chronic adhesive pleurisy. Chronic adhesive pericarditis. Gumma of heart wall. Cardiac hypertrophy. Aneurism of aorta, ascending and transverse portion. Arteriosclerosis. Edema of lungs. Gummata of liver. Atrophy of left lobe of liver. Gall stones. Chronic passive congestion of liver, spleen and kidneys.

884. Partial Autopsy. Anatomical Diagnosis: Carcinoma of the bronchi, with metastases in pleura, liver, kidney and peritoneum.

885. Anatomical Diagnosis: Double hydrothorax. Acute pericarditis. Cardiac hypertrophy. Edema of lungs. Chronic passive congestion of liver and spleen. Chronic interstitial nephritis. Colitis.

886. Anatomical Diagnosis: Chronic diffuse nephritis. Hypertrophy of the heart. Arteriosclerosis. Right bronchopneumonia. Petechial hemorrhages in intestines.

887. Anatomical Diagnosis: Chronic adhesive pleurisy. Edema of lungs. Ulcerative colitis. Ethmoiditis.

888. Anatomical Diagnosis. Lobar pneumonia of right lower and middle lobes and left lower lobe. Double fibrinopurulent pleurisy. Cloudy swelling of kidneys.

889. Anatomical Diagnosis: Infected wound of wrist. Acute axillary adenitis. Bronchopneumonia. Infarction of spleen. General lymphatic hyperplasia. Cloudy swelling of kidneys. Smears from axillary nodes show Gram-positive cocci in chains. Similar organism in lung.

890. Anatomical Diagnosis: Chronic adhesive pleurisy. Peritonitis. Fatty degeneration of liver. Acute hemorrhagic pancreatitis.

891. Partial Autopsy. Anatomical Diagnosis: Carcinoma of breast. Metastatic carcinoma of ribs, left femur (with fracture of femur), and spleen.

892. Anatomical Diagnosis: Lobar pneumonia of right upper lobe. Acute fibrinous pleurisy. Healed pulmonary tuberculosis.

DIVISION OF BACTERIOLOGY.

The routine bacteriological examinations made during the year may be classified as follows:

Blood Cultures:			
Typhoid bacillus		28	
Streptococcus		11	
Staphylococcus		4	
Pneumococcus		4	
Negative		129	
Total		176	
Urine Cultures:			
Colon bacillus		27	
Typhoid bacillus		2	
Staphylococcus		3	
Streptococcus		2	
Mixed cultures		8	
Negative		38	
Total		80	
Throat Cultures for diphtheria bacillus:			
Positive		104	
Negative		233	
Total		337	
Miscellaneous Cultures		189	
Smears examined for tubercle bacillus:			
Sputum	Positive	158	
	Negative	479	
			637
Urine	Positive	2	
	Negative	40	
			42
Chest and abdominal fluids	Positive	1	
	Negative	5	
			6
Spinal fluid	Positive	10	
	Negative	24	
			34
Stools	Positive	2	
	Negative	2	
			4
			723
Guinea-pigs inoculated for tubercle bacillus	Positive	9	
	Negative	45	
			54

Guinea-pigs inoculated for diphtheria bacillus	23
Mice inoculated	28
Vaginal smears examined for gonococcus	481
Urethral smears examined for gonococcus	60
Miscellaneous smears examined	197

The only serological examinations which have been made in any number are the Wassermann and Widal reactions:

Wassermann reaction	597
Widal reaction	312

DIVISION OF CLINICAL PATHOLOGY.

The following routine specimens were examined during the year:

Abdominal fluids	26
Blood: Estimation of coagulation time	15
" " hemoglobin	898
" " red cells	582
" " white cells	3,780
Examination for malarial parasites	77
" " filaria	5
" " trichinellæ	2
Chest fluids	94
Duodenal contents	4
Gastric contents	254
Glyco-tryptophan tests	4
Spinal fluids: Total and differential counts	31
Butyric acid tests	6
Stools	381
Urines	23,780

Roentgen Ray Laboratory

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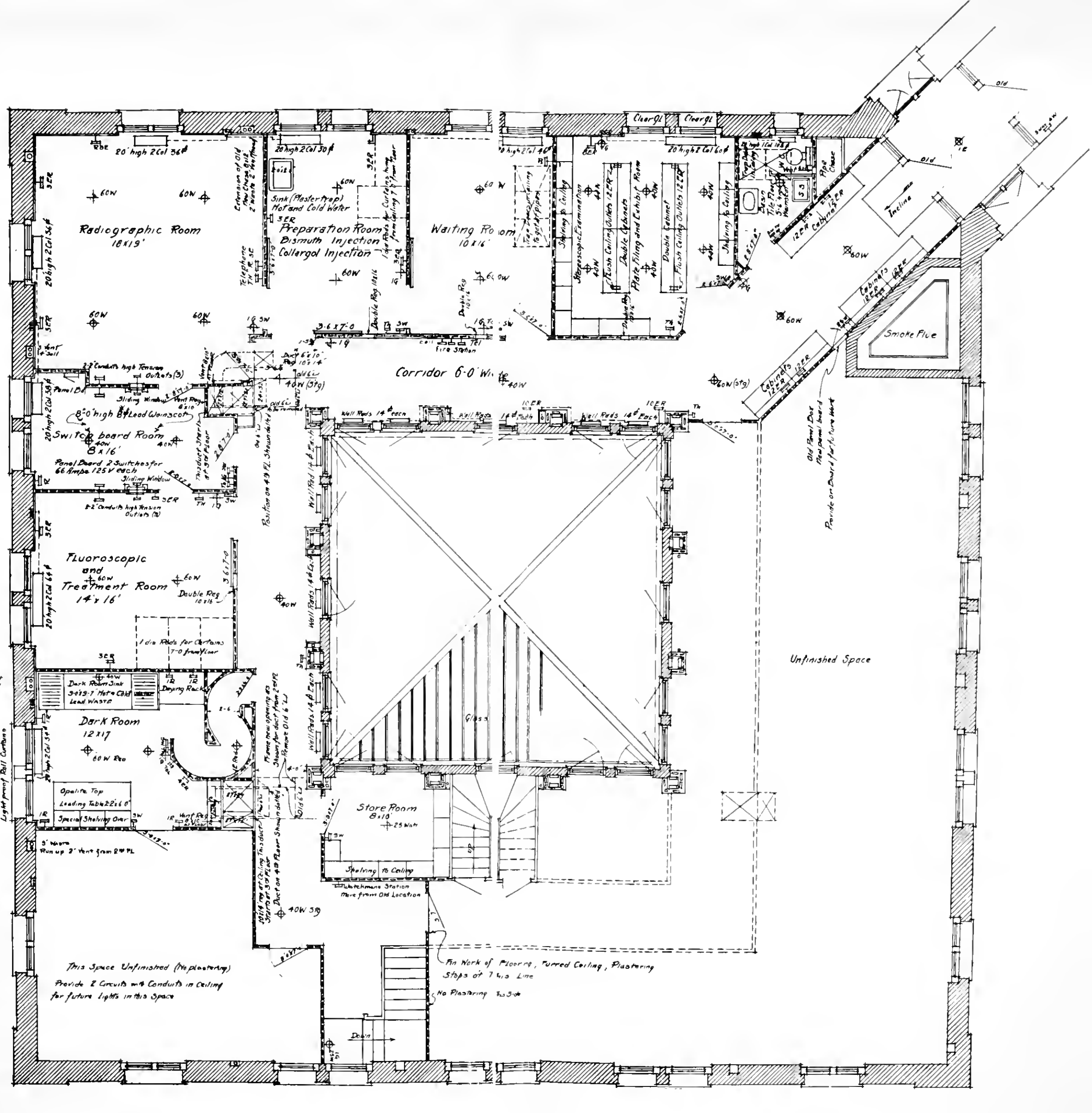

Radiographic Room
16 x 19'
Preparation Room
Bismuth Injection
Collargol Injection
Sink (Plaster trap)
Hot and Cold Water
Waiting Room
10 x 16'
Stereoscopic Examination
Plate Filing and Exhibit Room
Double Cabinets
Double Cabinet
Shelving to Ceiling
Flush Ceiling Outlets 12 ER
Cabinets
Incline
Pipe Chase
Smoke Flue
Corridor 6-0" Wide
Switch board Room
8 x 16'
8'-0" high 8# Lead Wainscot
Panel Board 2 Switches for
66 Amps 125 V each
Sliding Window
Fluoroscopic
and
Treatment Room
14' x 16'
Double Reg.
1 dia Rods for Curtains
7-0 from floor
Dark Room
12 x 17
Dark Room Sink
Drying Rack
Opalite Top
Loading Table
Special Shelving Over
Light proof Roll Curtains
Store Room
8 x 10'
Shelving to Ceiling
Watchmans Station
Move from Old Location
Wall Pads 14# each
Glass
Unfinished Space
Old Panel Box
Provide on Board for future Work
Fin Work of Flooring, Furred Ceiling, Plastering
Stops at This Line
No Plastering This Side
This Space Unfinished (No plastering)
Provide 2 Circuits with Conduits in Ceiling
for future lights in this Space
Down
Fire Station

PLANS OF THE ROENTGEN RAY LABORATORY, UNDER CONSTRUCTION ON THE THIRD FLOOR OF THE TRAVERS PAVILION, ST. LUKE'S HOSPITAL.

LEON THEODORE LE WALD, M.D.

Protection for the patients and the operators has been secured by the use of X-Ray-proof partitions and steel doors. Access to the dark room will be through a labyrinth, and a method of tank-development, which will accommodate the largest sized plates, will be installed. Room for expansion of the laboratory has been reserved on the same floor.

The plans appear on the two succeeding pages of this report.

REPORT OF A CASE OF DILATATION OF THE STOMACH. MEDICAL TREATMENT. RECOVERY RECORDED BY MEANS OF THE X-RAY.

LEON THEODORE LEWALD, M.D.

The following case appears to be worth reporting on account of the striking result of treatment, and the graphic record of this result as shown by the X-Ray examination.

Miss A. O., aged 21, a telephone operator by occupation, was admitted to the service of Dr. Austin W. Hollis on February 24, 1912, suffering from "chronic stomach trouble." Her family history was negative. She had had the usual diseases of childhood, and at the age of seven she first showed symptoms relevant to her trouble on admission. At that time she had been seized, while playing, with an attack of vomiting. There was no nausea, either before or after the attack, and the patient went on playing entirely undisturbed. For three months thereafter, each meal was followed immediately by an attack of vomiting, which was sometimes projectile in character, sometimes not. Occasionally the patient was nauseated. The stomach was not emptied at once, but the vomiting would continue at intervals for as much as five hours after each meal, being increased by any exertion and allayed by keeping quiet. A cramplike pain in the epigastrium with soreness and tenderness in this region accompanied the vomiting.

At first these attacks had occurred at intervals of four or five months, and lasted from two to three months, the patient's skin being dry and yellow and her bowels constipated throughout the period of disturbance. Recently the attacks had been more frequent, occurring every two or three months, with especial severity in spring and fall.

The attack which occasioned the patient's entrance to St. Luke's began two weeks previous to admission with severe and unremitting headache in the right occipital region. A week before admission vomiting recommenced, accompanied by nausea. Six months previous to this admission the patient had been operated on at St. Luke's for appendicitis. The physical examination made on her present entrance was negative, except for a slight general tenderness of the abdomen on deep pressure.

The X-Ray examination made on March 4, 1912, showed the stomach dilated and the greater curvature 4½ inches below the umbilicus. The stomach was not empty in 6 hours. The colon was sluggish.

The course of treatment consisted mainly of rest in bed, with daily lavage and a restricted diet, chiefly protein. On March 27th, one

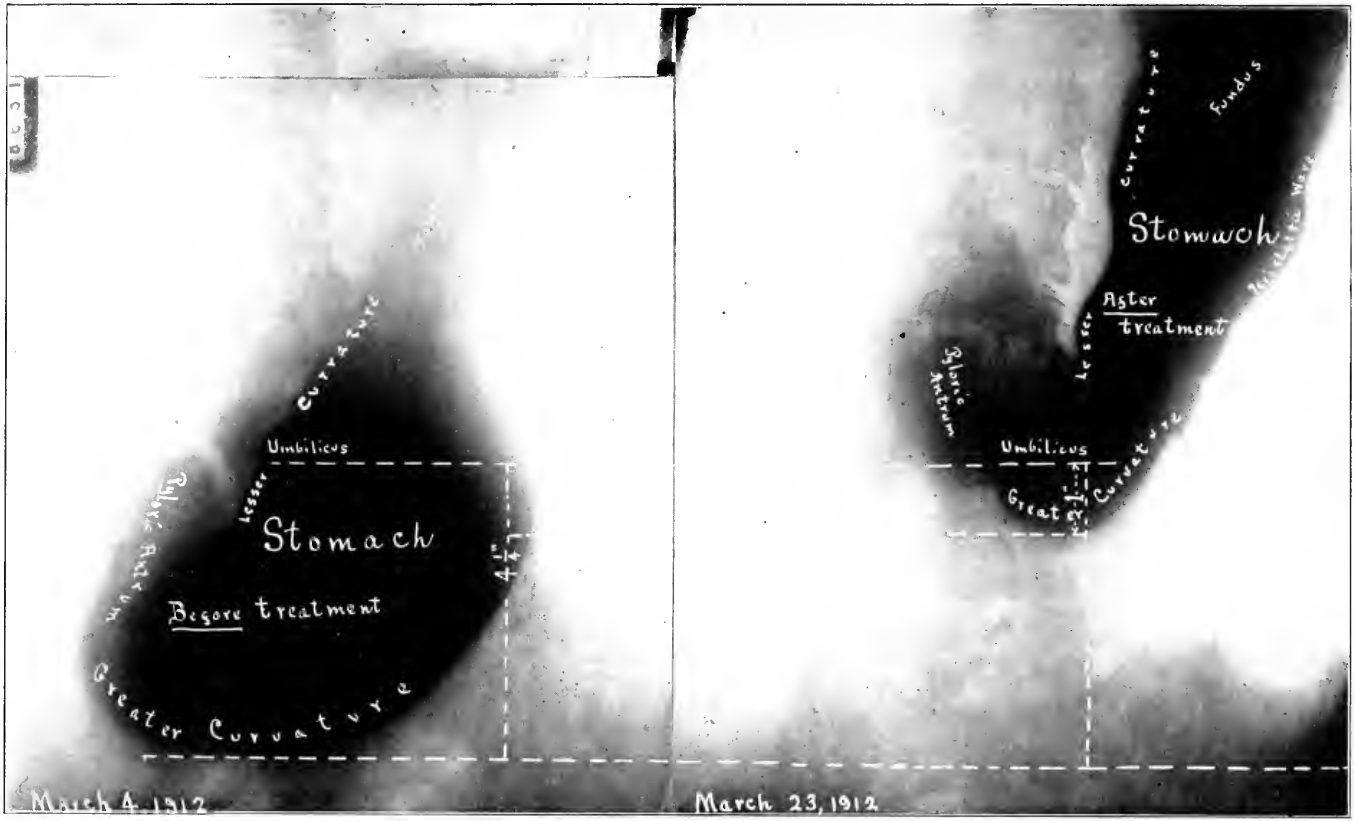

Fig. 1.—Radiographs showing the stomach before and after treatment, nineteen days elapsing between first and second X-Ray examinations.

month after admission, the patient had apparently regained her health, and could now eat without nausea or discomfort. The second X-Ray examination, made for the purpose of determining the condition of the stomach after treatment, shows in a very striking manner that the stomach *has* regained its tone. The dilatation has disappeared; the greater curvature has retracted so that it is only one inch below the umbilicus in contrast to the four and a half inches shown before treatment. The size and position are within normal limits, so that a very good prognosis can be offered as to continued good health if ordinary care is exercised. A further examination shows that the stomach empties itself in normal time. The tone of the colon has also improved, so that the tendency to constipation has been relieved.

Out-Patient Department

PRACTICAL NOTES FROM THE SURGICAL DIVISION OF THE OUT-PATIENT DEPARTMENT.

WILLIAM S. THOMAS, M.D.

The intention of this paper is to present a number of procedures in frequent use in the Surgical Division of the O. P. D., with comments upon the result of experience with them.

The subjects considered will be as follows:

1. Nitrous oxide anesthesia.
2. Use of picric acid in burns.
3. Removal of foreign bodies from the tissues.
4. Use of scarlet red on ulcers.
5. Open treatment of fractures.
6. Mode of demonstrating lesion of anal region.
7. Rigid supporters for varicosities.
8. Enucleation of tonsils.
9. Spring retractors.

1. NITROUS OXIDE ANESTHESIA.—Nitrous oxide gas, with oyxgen, in the past 3 years has been coming into very frequent use in our minor surgical work. It has so far supplanted ether and chloroform that, without statistics before me, I feel safe in saying that where one of the latter was administered 10 times 3 or 4 years ago, it is not used more than once now. Nitrous oxide gas has proved especially useful in the diagnosis of treatment of bone fractures and its use permits careful and painless manual examination in almost every case. It seems as though patients were entitled to its benefit. It is contraindicated or ineffectual in the cases of very young patients, patients with severe organic cardio-vascular diseases, and alcoholics.

In the case of alcoholism, rather than struggle to anestheticize a patient with gas or ether alone, it is frequently found quite feasible to attain relaxation and insensibility if the gas is preceded by a hypodermic of morphine. The particular advantage of this short anesthesia and rapid awakening without nausea or sickness in the case of ambulatory patients is obvious.

2. Use of Picric Acid in Burns.—The use of this acid in superficial burns was begun in the surgical clinic in 1907, and has proven to be a distinct advance over old procedures. At first, used as an ointment, later in hypersaturated solutions, it was found to have a poisonous effect if used on large surfaces. Its best manner of use seems to be as a wet dressing in watery solutions of one-half of 1 per cent. In burns of the first and second degree, pain is relieved, the serous effusion ceases and the growth of epithelium is enhanced. The principal disadvantage of the remedy is its ability to stain everything yellow with which it comes in contact.

3. Foreign Bodies.—Bits of steel, fragments of glass, wooden splinters, but especially fragments of sewing-needles, lost under the skin, are very common in any minor surgical clinic, and frequently prove to be difficult of removal. After free incision, and guided by a skiagram made immediately before, there is no doubt that the most important aid in discovering these lost fragments is the sense of touch. In the case of metallic foreign bodies material assistance has been afforded in our clinic by the telephonic searcher described in the bulletin of last year, which is in steady use, and is made by Wappler. When a metallic foreign body is located in a finger or toe or in the webs between them, its shadow may often be seen in the dark room by transillumination with a small electric light shielded on all sides but one, as is used for the illumination of the accessory sinuses of the nose. Elsewhere, these lights are of no use. When used to demonstrate a foreign body in a finger or toe the light must be applied to that side farthermost from the foreign body. In other words, the foreign body must lie nearest to the skin next the observer's eye or it cannot be seen.

4. Scarlet Red.—The extended use of this dye as a dressing for granulating surfaces has demonstrated that it has a field of usefulness. In our experience, corroborated by control experiments, it has been shown to hasten the growth of epithelium upon healthy surfaces. In the case of varicose, or infected ulcers, where there is no previous tendency to heal, scarlet red alone is worse than useless. It seems to have no antiseptic power.

5. Open Treatment of Fracture.—Continued use of the metallic plate in selected cases of fracture confirms the good opinion of this surgical procedure. It is seldom necessary, however, and the indications for the method seem to be clearly the following: Impossibility of fairly good reduction of deformity, rotation deformity of radius,

mal-union and persistent non-union. Careful asepsis and avoidance of traumatism, when operating, are necessary. Lane's steel plates require a considerable outlay for a full set and cannot always be obtained. Sheet aluminium is cheap, and may be easily obtained at wholesale hardware stores and fashioned into the proper form for internal splints in a few minutes, to suit the exigencies of each case. The writer described the use of such plates in the Bulletin of 2 years ago. In an experience of 3 years with the use of aluminium plates, no serious cases of infection have been encountered. In 2 patients there was enough infection to make it necessary to remove the plates in order to cure a sinus, but in both of these cases the ultimate result of the operation was perfectly good.

6. Mode of Demonstrating Lesion of Anal Region.—Ever since suction cups have been used as recommended by Bier in his hyperæmia treatment, we have turned this method of producing a partial vacuum to use in certain rectal conditions. With the patient in a lithotomy position, a suction cup of a diameter of 1½ to 2½ inches, and properly curved, is applied over the anus and the air exhausted. Any external hemorrhoidal conditions will be exaggerated and plainly visible where they might otherwise be obscured by horizontal posture of the patient. In the case of internal hemorrhoid or a fissure or of a lesion within the first inch of the rectum, suction with a cup will evert the rectal mucous membrane in such a manner that hemorrhoids will stand out more distinctly and the mucous membrane of the whole circumference of the bowel will be brought into view.

7. Rigid Supporters for Varicosities.—The day of the elastic supporter for slack abdominal walls and misplaced viscera is past, or ought to be. In like manner the elastic stocking for the support of the leg has at last found a rival in rigid appliances. A writer in the *New York State Medical Journal* has recommended the use of adhesive strapping of the whole leg in cases of varicose ulcer; and Dr. John B. Murphy, of Chicago, in the *Journal of the American Medical Association* of March 27, 1909, recommended the use of inelastic leggings in varicose ulcers. A mode of procedure in use in the Medical Division of the O. P. D. was to treat varicose leg ulcers by adhesive strapping from the toes to the knee, omitting the sole, and after the ulcer is cured to have made a muslin corset for the leg to prevent recurrence. The strapping is of strips about one-half inch wide, applied obliquely in two directions and crossing each other so as to leave openings of regular intervals, like a checker-board. This strap-

ping is left on about a week, with a pad of gauze over the ulcer and the usual bandage over the whole, changed as frequently as necessary. The method is not applied when there is a phlebitis, or where the skin is much macerated or eczematous, or where the ulcer is behind or below the ankle. The method seems to have given most encouraging results in these cases, which have long been considered the bane of every minor surgical clinic.

8. Enucleation of Tonsils.—For the past 4 years it has been our practice to enucleate the tonsils in practically every case operated upon. Only in cases where the tonsil projects far into the pharynx and is not covered by the interior pillar of the fauces has the guillotine been used without a preliminary dissection of the tonsil from its bed. The procedure ordinarily employed requires complete anesthesia, but is very simple. In children, where the tonsil has not been the seat of a fibroid degeneration, to make it abnormally adherent to its surroundings, a short incision is made along the most prominent portion of the edge of the interior pillar. The finger is worked into this incision with its palmar surface toward the tonsil, and the latter is shelled out of its bed by blunt dissection from all its attachments except along its posterior aspect. At this point the tonsil is grasped with the sponge forceps or other convenient instrument, and the pedicle is snipped with a pair of curved scissors or possibly with a tonsillotime. This method is chosen rather than sharp dissection on account of the fact that less hemorrhage follows and because there is less danger of cutting what is not desired to be cut. Routine questioning of patients in respect to possible hemophilia is insisted upon. In the case of weak, anæmic children, calcium salts are administered some days prior to the operation, in the hope of lessening hemorrhage.

9. Spring Retractors.—The German silver wire self-holding retractors described in the *Journal of the American Medical Association,* in April, 1903, have been found to be of use in our operating room, where the desired number of assistants are not always available.

POSSIBLE CAUSES OF FAILURE FOLLOWING THE USE OF BACTERIAL VACCINES AND ANTISERA.*

H. E. Plummer, M.D.

"The most mischievous ignorance is that of the critic." So many unjust criticisms are heard relative to the value of bacterial vaccines and antiserum in the treatment of infections, that the above quotation of Voltaire seems scarcely out of place. Some reports indicate such successful results and others such absolute failures in similar cases that they stimulate us to inquire into the reasons for such diversity of conclusions. May not these failures be in part due to faulty dosage, to incorrect intervals elapsing between the doses, to a wrong appreciation of the benefits to be expected from the use of a bacterial vaccine or antiserum, and to a faulty selection of the remedy to be used? Let us first study briefly the qualities of a bacterial vaccine and of an antiserum and the theories upon which their use is based.

Antisera are obtained from some bacteria that do not produce extracellular toxins in sufficient quantities: as, for example, the streptococcus and the gonococcus. The germs themselves are injected into the animal, first in minute doses of greatly attenuated cultures, then in gradually increasing doses until such a resistance exists in the animal that large amounts are tolerated. These antisera may be said to possess antibacterial power. The fact that antisera are elaborated in the horse distinguishes them from bacterial vaccines which are simply suspensions of killed bacteria in physiological salt solution. From this it may be seen that when antisera are used the patient is inoculated with the protective substances produced by an animal, whereas when bacterial vaccines are injected the patient must produce his own protective bodies.

Wright and others have demonstrated the fact that there are sub-

*Read before the West Side Clinical Society.

Note.—The vaccines used in the preparation of this article were kindly furnished by the Department of Experimental Medicine of Parke, Davis & Co.

stances in the blood stream that assist, or are necessary to aid the phagocytes in their successful warfare against invading bacteria. If, however, Nature's laboratory is unable to completely overwhelm them at once, the destruction of a portion of the invading host will produce a strengthening of the defending force. This is known as autoinoculation and is best illustrated in the pneumonic crisis. In some infections the protection afforded is lasting, as in smallpox and in yellow fever, while in others, as in tuberculosis, the protection is transient. When Nature is able to cope with the infecting bacteria, self-immunization is likely to take place. This is due to the death of a certain number of the organisms and their immediate effects as immunizing agents. The artificial introduction of a suitable number of dead microorganisms, *i.e.*, the injection of a bacterial vaccine, may turn the scale and produce the reinforcement necessary to Nature, which at the moment is so urgent. As a result of the injection of these bacterial products such indefinite substances as bacteriolysins, precipitins, agglutinins, and opsonins are produced. The latter, which are measurable, act on the bacteria in such a way as to make them more vulnerable to the attacks of the phagocytes. The phenomena accompanying spontaneous recovery from an infectious disease, and which for a time at least prevents a new attack, we term natural acquired active immunity. That produced by the injection into the tissues of small quantities of living or killed microorganisms or of toxins produced by these organisms we call acquired active immunity. "In passive acquired immunity, on the other hand, the patient does little or nothing toward obtaining this immunity. The toxins, which characterize the disease, are simply neutralized or rendered inert by the injection into the individual of protective substances, which have developed in the serum of another animal, as the result of active immunization. This form of immunity is a temporary expedient, which simply serves to hold the disease processes in check sufficiently long to permit Nature to manufacture and bring into play such protective and bactericidal substances as will rid the individual of the offending bacteria and their toxins."

The opsonic index is the comparative phagocytosis of the patient's serum to a normal pool serum. The technique is complicated and the slightest inaccuracy produces decidedly varied results. The index has proven of great value in indicating the proper dosage, frequency of injection, and results obtained. It has been found that the careful observation of the clinical symptoms acts as a sufficient guide to

the treatment by bacterial vaccines in the more common infections. By determination of the opsonic index, Wright has demonstrated that following the injection of vaccines, there is first a drop in the opsonic index and later a rise. This drop he calls the negative phase and the rise the positive phase. The use of bacterial vaccines in infections is clearly defined. It is useless to suppose that every case of infection is a suitable one for this treatment. The question is of necessity whether the patient is capable of producing antibodies to the infecting agent or whether they should be introduced from without, *i.e.*, from an animal already immunized to the infecting agent.

A case occurs to me of a patient infected by an attenuated strain of streptococcus which ran a chronic course. A culture was obtained and an autogenous vaccine prepared and administered at intervals. The patient continued to grow worse and ultimately died. This patient was already so surfeited with streptococci that the introduction of a few million more dead germs seemed scarcely the rational treatment for such a condition. Had such a patient the power to produce his own antibodies, it would appear rational that he would have produced them with the many streptococci swarming in his system, without the introduction of more. Thus antistreptococcic serum should have been first used to modify the infection. Therefore, in general septicemias the serums are indicated; whereas in localized or semi-localized conditions the bacterial vaccines are to be preferred.

In reviewing the important subject of dosage we find a great variety of opinions. There are, on the one hand, the advocates of small doses and, on the other, those of large doses, but no set rule can be laid down for the administration of bacterial vaccines. The guiding factor in these cases must be more or less the resistance of the individual to the infecting organism and therefore the ability of the tissues of the patient to produce antibodies. Overdosage has been a not infrequent cause of absolute failure. It has often been noted that the administration of 400,000,000 staphylococci in cases of furunculosis has produced an increased number of pustules or furuncles instead of benefiting the condition. In other words, there has been a production of a prolonged or more severe so-called negative phase, thereby allowing the invading bacteria to obtain the mastery over the protecting forces of the blood stream. During the aggravated or very violent stage of the infection one should not administer a vaccine, as the activity of the infecting agent itself may be producing a negative phase.

No definite time can be positively made as to the proper interval for reinoculation, but in every case sufficient time should elapse between injections to allow the formation of the high wave of the positive phase. In scarcely any instance should the interval between the administrations be less than 3 days or in very rare instances more than 7 days, the average time being about 4 to 5 days. An instance has occurred to me in which bacterial vaccines were administered in maximum doses daily and it was noted that the patient was gradually becoming worse. The treatment was therefore discontinued, and at the end of the second day most marked improvement was observed in the condition of the infection. The lack of improvement was undoubtedly due to the fact that the patient was kept in a constant state of negative phase.

Freeman, working in Wright's laboratory, noted the occurrence of autoinoculation following the manipulation and massage of affected joints in gonorrheal arthritis. Therefore, in the handling of localized infections one must always bear this fact in mind; that following any form of treatment, be it massage, X-ray, electric, radiant heat application or Bier's hyperemia, observations have shown the regular sequence of positive and negative phase and phase of increased resistance, identical with that produced by an ordinary vaccine prepared from the invading organism. Therefore, when treating a patient with bacterial vaccines care must be taken to avoid overmanipulation of the infected area, as this may result in self-inoculation which would be equivalent to an overdose of bacterial vaccine.

On the other hand, knowing as we do that the opsonins render the bacteria vulnerable to the phagocytes, it is very important to the success of the treatment that the lymph be made to flow through the infected tissues. In cases of infection, English authorities advise the use of a wet dressing compound of 4 per cent sodium chloride and .5 per cent sodium citrate. This solution is an ideal lymphagogue, and prevents, by inhibiting coagulation, the formation of a scab. Let us remember also that leucocytes are essential to the success of vaccine therapy and that the best results may be expected when large numbers of healthy leucocytes are present. MacWatter claims that the leucocytes may be increased in number 6 or 7 fold by the administration of yeast. This method is rather crude, as no definite amount of nucleinic acid is administered. As the success following the use of yeast in these cases is undoubtedly due to the nucleinic acid which it

contains, it would seem more scientific to use nucleinic acid which, under the name of nuclein solution, is readily obtainable.

Some failures are due to the selection of unsuitable vaccines, as the following case will show: A young man suffering from chronic prostatitis came under my personal observation. Improvement was obtained by the use of gonococcus vaccine, but it seemed impossible by this means to effect a cure. At this stage, I resorted to the use of a combined vaccine made from a mixture of common pyogenic organisms. The results of these injections were very gratifying. In this case, though the infection was primarily due to the gonococcus it is evident that other organisms replaced, at least in part, the one named. It should be borne in mind that though a specific organism may be the original cause of the trouble, the advent of other germs producing a mixed infection may delay a cure and, in the absence of indications to the contrary, it may be well in such cases to employ such a mixture of vaccines.

CONCLUSIONS.

1. There should be a proper determination of whether an antiserum or a bacterial vaccine is indicated. 2. Care should be exercised in deciding on the proper dosage for each individual infection. 3. The administration of vaccines during the aggravated stage of infection may produce harmful effects. 4. A proper interval should elapse before repeating the injections. 5. Too much local treatment of the infection may produce harmful results. 6. Such aids as tend toward the increase of leucocytosis or the freer movements of lymph should be given to assist the effects of the bacterial vaccine. 7. Any possible change in the character of the infection should be observed.