SOME

COLLECTED REPRINTS

OF THE LATE

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28. A Case of Fibrinous Rhinitis (with Dr. Birkett)—*Can. Med. & Surgical Journal*, 1894.

*29. Death by Poisoning (7 cases).

30. The Use of the Autoclave for Sterilizing Nutrient Gelatin—*The Medical News*, 1895.

*31. A few Observations upon Sedimentation in Water—Trans. American Public Health Assoc., 1895.

*32. On Grouping Water Bacteria—Trans. American Public Health Assoc., 1895.

*33. Clinical Microscopy—Reference Handbook of Medical Sciences. Supplement, 1895.

*34. Biological Analysis of Water—*Ibid.*

35. Report of a Year's Work on Bacteriological Diagnosis of Diphtheria—MONTREAL MEDICAL JOURNAL, 1896.

*36. On the Application of the Serum Diagnosis in Typhoid Fever—*New York Medical Journal*, 1896.

37. A Note upon Serum Diagnosis by means of Dried Blood Samples in (Experimental) Cholera—*New York Medical Journal*, 1896.

38. Ueber den Gebrauch von im Wasser aufgelosten trockenen Blute fur die Serumdiagnose des Typhus—*Centralblatt fur Bakter. Parasit. u. Infekt.* XXI. Band, No. 13/14, 1897.

39. Three cases Illustrating the Value of Bacteriological Diagnosis of Leprosy for Public Health Purposes—MONTREAL MEDICAL JOURNAL, 1897.

40. On the Difference between Blood Serum and Blood Solutions, the Condition of the Test Culture and Significance of Bacterium Coli Infection in Relation to Typhoid Diagnosis—MONTREAL MEDICAL JOURNAL, 1897.

41. On the Iodine Test for Semen—*Boston Medical & Surgical Journal*, 1897.

42. On the Medico-Legal Application of Entomology—MONTREAL MEDICAL JOURNAL, 1897.

43. Portable Vacuum Chamber for Formaldehyde Disinfection—*New York Medical Journal*, 1897.

44. Notes on Household Disinfection by Formaldehyde—*British Medical Journal*, 1897.

*45. An Experiment with the Serum Reaction as a Test for Typhoid Infection in Water, etc.—*New York Medical Journal*, 1897.

46. On the Application of the Serum Diagnosis in Typhoid Fever to the Requirements of Public Health Laboratories—Trans. American Public Health Assoc., 1897.

47. Compilation of Pathological Reports of Montreal General Hospital from 1883-1895—August, 1897.

48. The Medico-Legal Significance of the Presence of Sugar and Glycogen in the Liver, Post-mortem—*Boston Medical & Surgical Journal*, 1898.

*49. Cardiac Embolism—MONTREAL MEDICAL JOURNAL, 1898.

50. The Condition of Test Cultures, especially as regards filtration, favourable to clear Serum Reactions by the Dried Blood Method—*British Medical Journal*, 1898.

51. On Serum Reaction with Bacteria other than the usual Pathogenic Forms—*British Medical Journal*, 1898.

52. A Quantitative Method of Serum Diagnosis by means of Dried Blood—*British Medical Journal*, 1898.

53. Notes on Progress of Legal Medicine—the Medico-Legal Study of Injuries—*Philadelphia Medical Journal*, 1898.

*54. Death by Electricity—MONTREAL MEDICAL JOURNAL, 1898.

55. Legal Medicine—*American Yearbook of Medicine & Surgery*, 1898.

56. Recent Work Bearing on the Pathology and Morbid Anatomy of Shock—*The Railway Surgeon*, 1899.

*57. Some Personal Experiences in Disinfection—Trans. American Public Health Assoc., 1900.

58. On the Practical Clinical Teaching of State Medicine—*Philadelphia Medical Journal*, 1900.

*59. Legal Medicine—*American Yearbook of Medicine & Surgery*, 1900.

60. On the Estimation of Disability and Disease Due to Injury—MONTREAL MEDICAL JOURNAL, 1900.

61. On the Establishment of Medico-Legal Diplomas—*Boston Medical & Surgical Journal*, 1901.

62. A Simple Method of Bacteriological Examination of Milk Supplies—MONTREAL MEDICAL JOURNAL, February, 1902.

63. A New Morgue for the City of Montreal—MONTREAL MEDICAL JOURNAL.

64. Report of Committee on Teaching of Hygiene and Granting of Diploma of D.P.H.

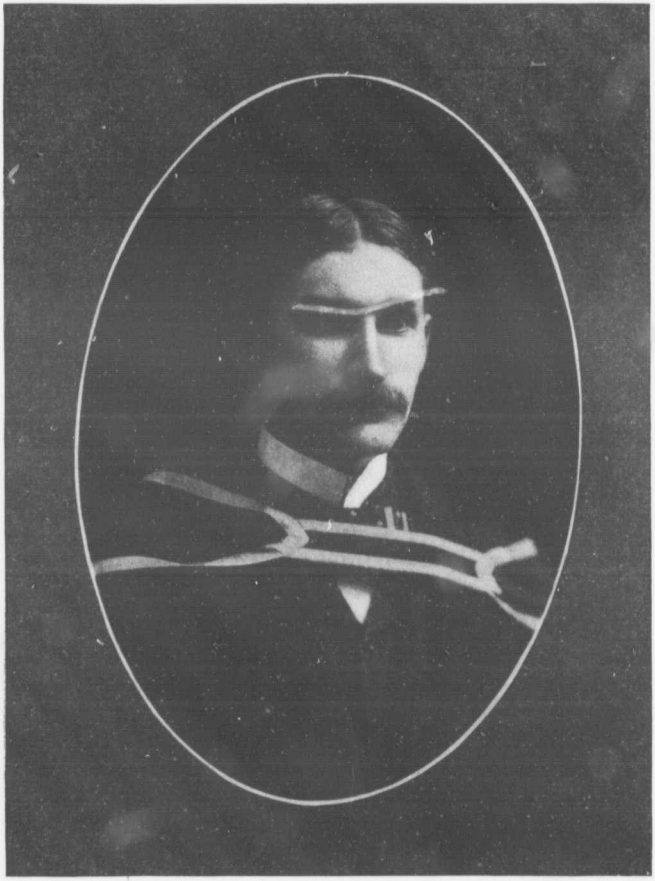
65. On Isolation Hospitals—Buck's Reference Handbook of the Medical Sciences, 1902.

Reprints of papers against which an asterisk (*) is placed could not be obtained in sufficient numbers.

OBITUARY.

PROFESSOR WYATT GALT JOHNSTON.

Reprinted from the Montreal Medical Journal, July, 1902.



THE LATE WYATT GALT JOHNSTON.

Obituary

PROFESSOR WYATT GALT JOHNSTON.

The death of Professor Wyatt Johnston, which occurred on the 19th of June, from pulmonary embolism, deprives the profession of Canada of one of its most brilliant and original members. His death was due to septic poisoning acquired in the autopsy room of the Montreal General Hospital. A streptococcus inoculation of his hand in February last incapacitated him for work for a few weeks, but he returned again to his duties, and probably received a second infection in April. A thrombus appeared in the internal saphenous vein of his left leg about the end of April, followed by a more extensive coagulation later. He then removed to a private ward in the General Hospital, and shortly after this the veins of the other leg became similarly affected, and there was evidence of the presence of thrombi in the iliac veins of both sides. Death resulted on the 19th of June from a pulmonary embolism. The autopsy showed extensive thrombi on both sides and a general streptococcus and diplococcus infection.

Dr. Wyatt Galt Johnston was the son of the late Dr. J. B. Johnston, of Sherbrooke, Que. He received his early education at Bishop's College, Lennoxville, and entered upon the study of medicine in McGill University in 1880, graduating in 1884. As a student he showed special aptitude for pathology, and was a constant associate of Dr. Osler, assisting him at autopsies and preparing material for demonstration. Immediately after graduation he became resident medical officer in the General Hospital, and during the period of his service had more than the usual responsibility on his shoulders, as the staff that year was small and continually changing. In the spring of 1885 he paid his first visit to Germany, working during the summer in Virchow's laboratory in Berlin. Next year he returned again to Germany and carried on researches in connection with pernicious anæmia in the laboratory of Professor Grawitz at Greifswald. He was appointed demonstrator in pathology at McGill, and alone he gave all the lectures and demonstrations in this department for a number of years. He subsequently returned to Germany and worked at comparative pathology in Munich and also for some months in the Zoological Gardens in London. Shortly after this he resigned his position in pathology in McGill, but still continued to work in the General Hospital, devoting himself almost exclusively to bacteriology and beginning his medico-legal work.

In 1890 he made a bacteriological study of the water supply of Montreal, including the bacteriology of surface water generally. His very thorough report on the methods of classification of water bacteria and their sanitary importance was widely published. He was appointed lecturer in bacteriology at McGill in 1895, and was connected with the departments of pathology and hygiene. About this time also he became bacteriologist for the Provincial Board of Health and medico-legal expert for the district of Montreal. In 1897 he was made Assistant Professor in Public Health and Lecturer in Medico-Legal Pathology, and only a few months before his death received the chair of Hygiene and director of that department in the Faculty of Medicine, McGill University. He had practically occupied this chair for the Sessions 1900-01, and 1901-02, but until just before his death he was not formally appointed.

In December, 1895, he married Julia, daughter of the late Michael Turnor, of Rugely, England.

Professor Johnston's career has been a varied one ; from pathology proper he proceeded to the study of comparative pathology and spent a year in the investigation of the Pictou Cattle Disease. During this period he was associated with the Faculty of Comparative Medicine in McGill University as well as the Faculty of Medicine. Becoming more interested in bacteriology, he made a specialty of the sanitary application of this science, and was recognized as one of the best authorities on the subject of the bacteriology of water supplies. Originality, inventiveness and the power of recognizing the simplest and most direct method of reaching results, characterized his work in every department. While engaged in the investigation of the water supply of Montreal, he devised a very rapid and convenient method for collecting samples of water at various depths in such a way as to exclude the possibility of contamination. He also devised a method of distinguishing and counting the various animalculæ found in surface waters. When engaged in bacteriology in the General Hospital, his simple method for the diagnosis of diphtheria by culture on hard-boiled eggs, which is even now very widely used, was announced. His modification of the Widal reaction for the diagnosis of typhoid by means of dried serum, is also widely in use in different countries. An indefatigable worker, he tried all the methods announced that gave promise of practical value in connection with the application of bacteriology to hygiene and medico-legal work or to the diagnosis of disease. Having studied these methods, they were either immediately discarded or utilized in his work, and almost invariably improved upon or simplified. Instance after instance occurs to the writer where his inventive genius has made practical and useful many laboratory methods in bacteriology and pathology, and so to a great

degree simplified the method of instruction or economized the time of both student and demonstrator.

Dr. Johnston's habit of mind, his rapidity of thought and quickness in seizing upon what was of immediate importance, makes his published writings a very poor index of the amount of work he accomplished, and only to those who knew him do they give anything but an imperfect idea of the soundness and extent of his knowledge. His mental habit of concentration and going to the root of the matter, neglecting all side issues, which made him so valuable and trustworthy as a medico-legal expert and a coroner's physician, showed itself in all his research. A given problem presented itself to him, and he worked at it until he had satisfier himself with regard to that problem only, and being satisfied with results, was extremely careless in placing them upon record. His papers are characterized by directness, they are unaccompanied by any full or orderly history of the development of his subject up to the point at which he took it in hand, and he was content, as a rule, to incidentally refer to the work of others which he was able either to confirm or refute. These references, however, were always adequate for those familiar with the matter, but not always so for the ordinary professional reader. The difficulties which he encountered in attacking the problem, the side issues which sprang up in the course of his investigations, were rarely more than hinted at; the part of the subject which interested him and which impressed him with its importance was recorded red hot. Thus his published papers, an incomplete list of the more important of which follow this notice, are apt to strike the reader as being short and hurried, and certainly do not do him justice. But one has only to glance over the list to appreciate his remarkable versatility.

His mastery of many allied branches of medicine, gross and miscroscopic pathology, both human and comparative, bacteriology in a more abstract form as well as in its applications to hygiene and public health, sanitation, medical jurisprudence in many aspects, as well as medical education, will be found among his contributions. To each of these subjects he made valued and pre-eminently practical contributions, endeavouring to popularize each subject and to bring its methods within the reach of those to whom it would be useful.

Of recent years he devoted himself to hygiene and medical jurisprudence. One of his most thorough studies in the department of medical jurisprudence, in which he probably stands pre-eminent in Canada, if not on this continent, was a method of determining the pecuniary equivalent of injuries to one or other portions of the body, a subject which was very largely neglected by English-speaking medical jurists, although it has been very scientifically investigated in France and Germany. About the time of his death he was negotiating a scheme for the use of

companies with a large number of employees, and accident insurance companies, which would enable them to follow the after-effects of injuries and the conditions and treatment after leaving hospital, forming a basis from which valuable statistics could be compiled in this country and in the United States.

As a teacher, the same characteristics showed themselves, his great mental activity and his rapidity of thought often made it difficult for him to exhibit to his hearers the process by which conclusions were reached, hence, it was always necessary for him to carefully prepare his set lectures. But in practical teaching, at the autopsy table, at his weekly demonstrations in morbid anatomy, and, above all, to a few interested students, graduates or assistants in the laboratory, he was at his best. In devising methods of demonstration and of checking the work of classes in the laboratory, his originality was of much value. He was never contented unless he could develop some simple method of staining, some simple apparatus for class purposes, or for reproducing diagrams in a few minutes; method after method occurs to us all alike in their directness, simplicity and effectiveness for the purposes for which they were designed. He had a perfect genius for recognizing what was at the same time practical, scientific, sound and capable of performance by the simplest means.

In addition to his methods for the diagnosis of diphtheria and typhoid fever, already referred to, his simple method for the diagnosis of leprosy by scraping a suspected cutaneous nodule and staining the mixed blood and lymph, which exudes, and his introduction of the ordinary sterilized cotton wool swab at the end of a length of strong wire enclosed in a test tube, which now-a-days is used in all public health diphtheria outfits, may be cited as examples of how he always thought of the most direct and simplest methods of reaching his results.

But, after all, it is Wyatt Johnston, the man, the delightful companion, whose wit was ever full of such delightful surprises, that his friends and associates will regret the most. Intellectually honest, direct and simple to an unusual degree, he had the greatest contempt for all that savours of dishonesty and pretence in scientific work. His wonderful personal magnetism, his ready wit and sympathy, made him hosts of friends and admirers among those associated with him in the various organizations and societies with which he was connected—the Bar of Montreal, the Coroner's Court, the General Hospital, the Provincial Board of Health, the American Public Health Association, the American Medical Association, the American Medico-Legal Society, the Montreal Medico-Chirurgical Society, the Faculties of Law and Comparative Medicine.

The following resolution of regret, passed by the Faculty of Medicine, is but one of the many echoes of sorrow which has reached us from all parts of Canada and the United States:—

"The Members of the Faculty of Medicine of McGill University wish by this Resolution to put upon record their recognition of the great loss they have sustained by the untimely death of their brilliant colleague, Professor Wyatt Johnston.

"Throughout the twenty-one years during which he was associated with this Faculty, as student, demonstrator, lecturer and professor, his work was always characterized by a rare degree of conscientious exactness and originality. An earnest student, a thorough and successful investigator, and ever an advocate of advanced scientific medical education, his loss to the Faculty is indeed a great one.

"To his exertions this Faculty owes the practical character of the teaching in the various departments of State Medicine, with which he was connected, and also the introduction of advanced and post-graduate courses leading to the diplomas of Public Health and Legal Medicine.

"His high status among scientific men as a trustworthy investigator, especially in the fields of bacteriology and preventive medicine, has added not a little to the reputation of this University as a centre for research. His reputation as a reliable and scientific medical jurist and expert, was not confined to this city or this country, and his services to the Courts of Justice have done much to demonstrate to the professions of Law and Medicine the value of this branch of medical education.

"His colleagues in the Faculty of Medicine feel that in his untimely death each has lost a bright and cheering companion and a friend whose earnestness of purpose and enthusiasm in his work was a stimulus to all who came in contact with him,—one who was a high type of intellectual honesty combined with singular simplicity and modesty regarding his own capacity and the importance of his valuable original work.

"The Faculty further resolves to transmit a copy of this Resolution to Mrs. Wyatt Johnston and to his mother, Mrs. J. B. Johnston, and to convey to them their deep and heartfelt sympathy in the great loss which they have suffered."

The following is an uncomplete list of his more important contributions to scientific literature:—

Retrospect of Pathology—MONTREAL MEDICAL JOURNAL, 1889.
Thymus Gland—Reference Handbook of Medical Sciences, 1899.
Thyroid Gland—*Ibid.*
Retrospect of Pathology—MONTREAL MEDICAL JOURNAL, 1890.
An Unusual Case of Perityphlitis—MONTREAL MEDICAL JOURNAL, 1890.
A Rare Form of Kidney Tumer—MONTREAL MEDICAL JOURNAL, 1891.
Notes on the Bacteriological Study of Diptheria—MONTREAL MEDICAL JOURNAL, 1891.

On the Collection of samples of Water for Bacteriological Analysis—*Canadian Record of Science*, 1892.

A new Method for the Culture of Diphtheria Bacilli in Hard-boiled Eggs—*The Medical News*, 1892.

Anomalous Cases of Primary Nasal Diptheria—MONTREAL MEDICAL JOURNAL, 1892.

Fracture of the Skull from the Discharge of a Shot-gun into the left Orbit—*Montreal Medical Journal*, 1893.

Six months' Medical Evidence, Coroner's Court of Montreal—MONTREAL MEDICAL JOURNAL, 1893.

One Hunded Cases in Coroner's Court of Montreal in 1893—MONTREAL MEDICAL JOURNAL, 1893.

Return to an Order of Legislative Assembly of December 13th, 1893, upon Coroner's Inquests—1893.

"Coroner's Quest" Law in the Province of Quebec—Read before Medico-Legal Society, May, 1893.

Report of Special Committee appointed by Montreal Medico-Chirurgical Society to amend Coroner's Law for Province of Quebec—MONTREAL MEDICAL JOURNAL, 1894.

Coroners and Inquests—The *Gazette*, February 8th, 1894.

Statistics of Coroner's Court, Montreal, for 1893—MONTREAL MEDICAL JOURNAL, 1894.

A Biological Analysis of Montreal Water Supply from November, 1890-November, 1891—MONTREAL MEDICAL JOURNAL, 1894.

The Use of the Autoclave for Sterilizing Nutrient Gelatin—*The Medical News*, 1895.

A few Observations upon Sedimentation in Water—Trans. American Public Health Assoc., 1895.

On Grouping Water Bacteria—Trans. American Public Health Assoc., 1895.

Clinical Microscopy—Reference Handbook of Medical Sciences, Supplement, 1895.

Thymus Gland, Development of—*Ibid.*

Thyroid Gland, Pathology of—*Ibid.*

Biological Analysis of Water—*Ibid.*

Report on a Year's Work on Bacteriological Diagnosis of Diphtheria—MONTREAL MEDICAL JOURNAL, 1896.

On the Application of the Serum Diagnosis in Typhoid Fever—*New York Medical Journal*, 1896.

A Note upon Serum Diagnosis by means of Dried Blood Samples in (Experimental) Cholera—*New York Medical Journal*, 1896.

Ueber den Gebrauch von im Wasser aufgelosten trockenen Blute fur die Serumdiagnose des Typhus—*Centralblatte fue Bakter. Parasit. u. Infekt.* XXI. Band, No. 13/14, 1897.

Three cases Illustrating the Value of Bacteriological Diagnosis of Leprosy for Public Health Purposes—MONTREAL MEDICAL JOURNAL, 1897.

On the Difference between Blood Serum and Blood Solutions, the Condition of the Test Culture and Significance of Bacterium Coli Infection in Relation to Typhoid Diagnosis—MONTREAL MEDICAL JOURNAL, 1897.

On the Iodide Test for Semen—*Boston Medical & Surgical Journal*, 1897.

On the Medico-Legal Application of Entomology—MONTREAL MEDICAL JOURNAL, 1897.

Notes on Household Disinfection by Formaldehyde—*British Medical Journal*, 1897.

An Experiment with the Serum Reaction as a Test for Typhoid infection in Water, etc.—*New York Medical Journal*, 1897.

On the Application of the Serum Diagnosis in Typhoid Fever to the Requirements of Public Health Laboratories—Trans. American Public Health Assoc., 1897.

Compilation of Pathological Reports of Montreal General Hospital from 1883-1895, August, 1897.

The Medico-Legal Significance of the Presence of Sugar and Glycogen in the Liver, Post-mortem—*Boston Medical & Surgical Journal*, 1898.

Cardiac Embolism—MONTREAL MEDICAL JOURNAL, 1898.

The Condition of Test Cultures, especially as regards filtration, favourable to clear Serum Reactions by the Dried Blood Method—*British Medical Journal*, 1898.

On Serum Reaction with Bacteria other than the usual Pathogenic Forms—*British Medical Journal*, 1898.

A Quantitative Method of Serum Diagnosis by means of Dried Blood—*British Medical Journal*, 1898.

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Death by Electricity—MONTREAL MEDICAL JOURNAL, 1898.

Legal Medicine—*American Yearbook of Medicine & Surgery*, 1898.

Recent Work Bearing on the Pathology and Morbid Anatomy of Shock—*The Railway Surgeon*, 1899.

Some Personal Experiences in Disinfection—Trans. American Public Health Assoc., 1900.

On the Practical Clinical Teaching of State Medicine—*Philadelphia Medical Journal*, 1900.

Legal Medicine—*American Yearbook of Medicine & Surgery*, 1900.

On the Estimation of Disability and Disease Due to Injury—Read before Montreal Medico-Chirurgical Society, January, 1900.

On the Establishment of Medico-Legal Diplomas—*Boston Medical & Surgical Journal*, 1901.

A simple Method for Bacteriological Examination of Milk Supplies—MONTREAL MEDICAL JOURNAL, February 1902.

(*Reprinted from the* MONTREAL MEDICAL JOURNAL, *Sept.*, 1891.)

NOTES ON THE BACTERIOLOGICAL STUDY OF DIPHTHERIA.*

BY WYATT JOHNSTON, M.D., MONTREAL.

My original object in studying cases of diphtheria bacteriologically was to settle the nature of a series of those doubtful cases in which a diagnosis is practically impossible by the ordinary means of observation, and where the occurrence of post-diphtherial paralysis or the outbreak of similar or more typical sore throats is the earliest positive proof of the true nature of the case. As I have found it impossible to continue the work on account of the difficulty in obtaining clinical histories of the cases, I wish to publish the results obtained from the examination of another series of cases, most of which were capable of being diagnosed as diphtheria without much difficulty.

Some time ago I showed before this Society cultures obtained from cases of diphtheria in which the growth had presented the typical characters of the Klebs-Lœffler bacilli. The main characteristics of this organism are :

(1) Rapid growth in serum at blood temperature leading to the formation of well characterized colonies in 16 to 24 hours.

(2) Peculiarities of structure, especially the presence of involution, bacillus forms having clubbed or swollen ends, with granular, unevenly stained protoplasm.

(3) Toxic effects, producing pseudo-membranous inflamma-

* Read before the Medico-Chirurgical Society of Montreal.

tions, followed by characteristic paresis, in cats and rabbits, and uniformly killing guinea-pigs in two to five days when injected subcutaneously, with production of necrosis, surrounded by local inflammations and œdema at the site of inoculation, and usually associated with more or less marked parenchymatous degenerations and areas of cell-necrosis of the viscera. This condition is distinguished from other forms of experimental septicæmia by the fact that bacteria are absent from the blood and viscera.

These toxic effects are most striking, and serve to distinguish this organism absolutely from all other species of bacteria. They depend on the fact that the diphtheria bacilli generate an albuminous poison or toxin which, when absorbed into the system, produces fever, cell necrosis and paralysis, which symptoms and lesions can be also brought about by the injection of sterilized cultures containing the *toxin* alone without any living bacilli. The primary local lesions in diphtheria are probably due both to the toxic action of the poisonous substances evolved and the presence of the bacilli.

The disease diphtheria is therefore complex in nature; the presence of the local exudation of false membrane and the toxic constitutional effects being each a necessary part of all cases of true diphtheria. As the diagnosis is naturally based upon the local inflammatory appearances, and as diphtheria is by far the commonest cause of pseudo-membranous inflammations, the term diphtheritic has come to be applied to all severe inflammations attended with the formation of false membrane and accompanied by necrosis, while the term croupous is applied to milder inflammations where there is no necrosis. It should be borne in mind that the terms diphtheritic and croupous are of anatomical and not of ætiological significance, as the neglect of this distinction has led to a great deal of confusion. Diphtheritic inflammation is most often caused by the disease diphtheria, but is not by any means invariably due to this cause, since every sloughing inflammation of a mucous surface presents diphtheritic characters and must be called, on anatomical grounds, diphtheritic. On the other hand, croupous inflammations have been shown by Paltauf-

Kolisko to be sometimes the effect of the action of the Klebs-Lœffler bacilli, so that in this case we have a *croupous non-diphtheritic diphtheria.*

To prevent error, the terms diphtheria and diphtheritic should not be considered synonymous; and since the word diphtheritic has become too firmly established to be dropped from the vocabulary, it might be well if a suggestion of Dr. MacAllister (*Practitioner*, June, 1890) were adopted and the word *diphtherial* used always to express ætiologically in relation to the specific cause diphtheria, applying the term diphtheritic simply in its more general anatomical sense.

There is evidence to show that cases occur in which an anatomically genuine diphtheritic sore throat may be caused by other organisms than the Lœffler bacilli. Roux and Yersin (*Pasteur Annales*, July 1890) record with great care several such cases where the infection was due apparently to streptococci. Out of 80 cases of angina admitted to the diphtheria wards of the Childrens' Hospital, the bacilli were found in 61. Roux and Yersin had no scruples in classing the other 19 cases, in which no bacteria were found, as non-diphtherial angina. A much more remarkable series was that published by Dr. T. M. Prudden, where, in 24 cases of diphtheritic sore throat occurring in children, streptococci were found in 22 and staphylococcus aureus in 2, while the Lœffler bacillus was not met with in a single instance. It is important to note that these cases were obtained from an epidemic among children who were inmates of an institution in which scarletina and erysipelas were epidemic at the time.—(*Amer. Jour. Med. Sci.*, May 1889.)

A subsequent series of 12 cases, all fatal, by the same author (*N.Y. Medical Record*, April 18th, 1891), showed the presence of the virulent Lœffler bacilli in every instance, and a carefully prepared table appended, giving the total number of cases investigated in this manner by various authors up to date, shows that the Lœffler bacilli were found in 307 out of a total of 342 examined—about 90 per cent. The suggestion that the cause of diphtheria in America differed from that in Europe had been previously disproved by Prof. W. H. Welch and Dr. A. C.

Abbott, who, in a most exact study of eight cases of typical diphtheria occurring in Baltimore, found the Lœffler bacilli present in every instance.—(*Johns Hopkins Hosp. Bulletin*, Jan. 1890.)

That diphtheritic membrane can be produced amongst other causes by the streptococcus pyogenes is shown by the uniform presence of this organism in the diphtheritic endometritis occurring in puerperal fever. The only statements as to the absence of the Lœffler bacilli in diphtheritis of other regions than the throat is that furnished by Paltauf and Kolisko, who were unable to find the bacilli in this condition or in intestinal or cutaneous diphtheritis phagædena.—(*Wiener med. Wochenschrift*, No. 8, 1889.)

I have tabulated below the cases collected by Dr. Prudden and added my own cases. (I have not included in this table the cases where I was not able to obtain satisfactory material for examination, as I found in cases A 2 and A 3 that the bacilli might easily be missed if the secretions only were examined, and although found in portions of actual membrane).

Observer.	Reference.	No. of Cases Examined.	Lœffler Bacilli.	
			Present in.	Absent in.
Babes	Zeitschrift f. Hygiene, Bd. 5	42	42	0
D'Espine	Revue Medicale de la Suisse, Romande, 1888, No. 1	14	14	0
Ortman	Berl. Klin. Wochen., 1889, No. 10	16	15	1
Spronck	Central. f. Pathol., Bd. 1, p. 218.	7	7	0
Roux and Yersin	Annales de L'Institut Pasteur, Dec. 1888	15	15	0
Paltauf and Kolisko	Wiener Klin. Woch., 1889, No. 6	50	50	0
Zarniko	In Aug. Dissertation, Kiel, 1889	20	18	2
Beck	Zeitschrift f. Hygiene, Bd. 8	52	50	2
Sorensen	Nordiskt Medicinskt Archiv, Bd. 18, No. 25	10	7	3
Escherich	Cent. f. Bacteriologie, Jan. 2, '90	22	20	2
Tangl	Cent. f. Pathologie, Bd. 1, p. 705.	18	18	0
Briegn and Fraenkel	Berl. Klin. Wochen., Mar. 17, '90	22	22	0
Prudden (1st series)	Am. Jour. Med. Scie., May 1889.	24	0	24
Welch and Abbott	Johns Hopkins Hosp. Bul., vol. 2, No. 11	8	8	0
Prudden (2nd series)	N. Y. Med. Record, April 18, 1891	12	12	0
Johnston	Montreal Med. Jour., Sept. 1891.	10	9	1
		342	307	35

The method of examining is very simple, and no one having an elementary training in bacteriology would have any trouble in carrying it out. Following the directions of Roux and Yersin

(*Annales Pasteur*, July 1890), I employed sterilized serum, obtained either from ox-blood or from hydrocele or pleuritic exudation. I can quite confirm the statements of these observers as to the advantage of this medium over agar-agar jelly, since by employing serum the colonies of the diphtheria bacilli are readily recognizble at the end of twenty hours, or even earlier if the serum be "improved" in the manner recommended by Lœffler, through the addition of one-fourth its bulk of a broth containing peptone, beef-tea and sugar. On the other hand, if agar-agar be employed, the colonies are never recognizable before the end of forty-eight hours, and show nothing strikingly characteristic before the fourth day. As the essential object of the examinations is to make an early and positive diagnosis, the saving of twenty-four hours would seem to be of vital importance in itself, but the serum method has also the advantage of permitting the diphtheria bacilli to bring their colonies to maturity before the other bacilli which are present have even commenced to form visible colonies. With agar, on the other hand, the two days needed for the appearance of the diphtheria colonics affords ample time for the development of the putrefactive forms, if these are present in any large number. The only advantage of the agar method is that the pyogenic staphylococci and streptocacci which are usually present have more characteristic growth than on serum—a matter of secondary importance.

There is a current impression that the serum is difficult and troublesome to prepare, and this has led to its use being avoided in many laboratories when any other medium can be substituted. This idea is quite erroneous, as serum is as easily made as any of the other nutrient media—in fact, far easier than gelatine, if prepared according to the method given by Hueppe (*Centralb. f. Bact.*, July, 1887), which consists in coagulating and sterilizing the serum at once simultaneously. After the tubes are filled to a depth of one to two inches they are laid obliquely in rows in a thermostat, which is then heated till the inner temperature reaches 68° to 75°C. After half an hour or more at this temperature the tubes will be found to have coagulated, leaving the serum nearly transparent. The temperature can now be raised

to about 90° by bringing the water in the jacket to the boil, and the tubes should be exposed to this heat for half an hour each day on three or four successive days, when, on placing them in the incubator in the usual manner, all but a few will be found to remain perfectly sterile. If Lœffler's serum is required, the serum is mixed with one-fourth its volume of Lœffler's bouillon before filling into the tubes. This addition does not interfere with the property of coagulating and remaining transparent. If blood serum is employed, care should be taken in collecting it that the clot is allowed to form before transporting the jars of blood. After standing forty-eight hours in a refrigerator or in a cool cellar, an abundant supply of clear serum can be obtained. The presence of small traces of hæmoglobin in the serum does not much impair its translucency, certainly not enough to render it unsuitable for the isolation of diphtheria bacilli.

The serum tubes could be prepared and kept in stock by druggists if the method ever comes into general use, which seems unlikely. The examination may be made from material taken direct from the throat by scraping the membrane, or, preferably, a piece of membrane may be detached by a pair of forceps or a swab of cotton wool. If the membrane has to be transported, it may be put into a clean, dry test-tube or folded up in blotting paper. To examine, it need only be moistened by a drop of sterilized water. A microscopical examination can be made by smearing the piece of membrane over the surface of a cover-glass, passing it three times through a flame. After drying and staining with a drop of any aniline stain, but preferably by Gram's method, the diphtheritic bacilli are seen as short thick rods (about the same length as tubercle bacilli), lying in little groups. These bacilli are present in enormous numbers in the early stages of diphtheria, but diminish rapidly in number as the membrane softens. The highly characteristic involution forms, which assume comma or club shapes with swollen ends, and present a protoplasm broken up into small granules, are only recognizable with a good immersion lens.

The cultures are made by drawing the infected needle in parallel lines along the surface of the serum. By treating two

or three tubes in this fashion, the infecting material at first abundant and producing a continuous dense growth, will in the second or third tube only implant a very few bacteria, so that the colonies can be studied separately. The tubes are to be kept at about body temperature for twenty-four hours, when minute white points appear on the surface of the serum, attaining about the size of pin-heads or of split peas. These, if found under the microscope to consist of bacilli, are probably the diphtheria bacilli, as the other forms would not have attained such size in so short a time.

The other colonies which attain such proportions in twenty-four hours are almost invariably found to be micrococci, usually the staphylococcus pyogenes.

I will not go further into the culture experiences with the bacillus than to say that I have been able, in all my cases, to confirm the statement first made by Welch and Abbott, of the Hygienic Institute, that the bacilli form an abundant invisible growth on potatoes, a medium stated by previous observers to be unsuitable for its cultivation. I also can substantiate the statement of Beck, that the agar cultures do not show the characteristic involution forms.

The method, however, has a serious defect, since an organism exists which is identical in size and appearance with the Lœffler bacillus, and grows on serum in a similar manner, though totally devoid of pathogenic properties. This is called the pseudo-diphtheria bacillus, and has been frequently found in the throats of healthy persons, as well as in follicular tonsillitis. The proof, therefore, is not absolute until substantiated by the inoculation of some susceptible animal. How far this pseudo-diphtheria bacillus would interfere with the method in practical work is not yet certain. Personally I have so far only met with the virulent or true bacillus.

The results of my examinations are as follows:—

Of nine cases examined in which the diagnosis of diphtheria could be made without much difficulty from the symptoms and the appearance of the throat, the Klebs-Lœffler bacillus was found in eight. In the case where it was not found, the con-

dition was one of an undoubted false membrane which contained, microscopically, large numbers of bacilli which appeared to be the organisms in question but did not appear in the cultures. When the specimen was taken the throat had just been freely sprayed with a solution of hydrogen peroxide, and the negative result may have been due to its disinfectant power of inhibiting the growth, though the fact that colonies of staphylococcus aureus appeared make that less likely. There was an anomalous course in this case, since the throat was found perfectly clear of membrane on the following day, preventing my repeating the experiment. In spite of this the disease appears to have been true diphtheria, as the nurse stated that the patient had a distinctly nasal voice when he left the hospital, ten days later. The bacillus was also found in an anomalous case where an extensive false membrane existed with almost no disturbance of the general health. In this case there was no paralysis.

In six cases where the diagnosis was doubtful, the bacilli were not found. Several cases of follicular tonsillitis and a case of scarletinal sore throat were examined with negative results.

I have divided the cases into two groups—(*a*) where the condition was clinically like diphtheria, and (*b*) anomalous cases. The cases are as follows, the first being given in detail and the remainder summarised in a table at the end of the article.

Case 1.—M. L., female, aged 21, admitted April 9th, 1891, into the female medical ward of the Montreal General Hospital, under Dr. Ross, with a suspicious-looking patch of membrane in both tonsils and a tiny membranous patch on the side of the uvula; temperature 102½°. Next day an extensive patch of dirty gray membrane was seen in the uvula and soft palate. Transferred to diphtheria ward. Seen on April 11th; temperature 100°; had a patch of membrane on left anterior pillar of fauces. Discharged May 2nd; no paralysis.

Microscopic examination of the membrane showed an enormous number of short, thick bacilli, a few larger bacilli, and some clusters of micrococci.

Cultures in serum made on May 11th and kept at 35°C. showed on the following day numerous small, flat, white colonies

composed of short, thick bacilli, having the typical appearance of the Lœffler bacilli. Involution forms numerous. In agar plates small, flat colonies appeared on the second and third days, showing superficial thin concentric growth from a small central white spot. In stale culture in agar the growth, on first transplantation, was barely visible, but in subsequent generations the colonies became more distinct, forming always flat, circular, concentric, white growths on surface about the entry point of the needle. Microscopically the bacilli in the agar cultures were short, thick rods with rounded ends; their length was about 0.0015 to 0.002 mm. (one and a half to two-thousandths of a millimetre). None of the agar cultures showed involution forms.

April 28th, 1891. — Inoculated a very large, full-grown female guinea-pig with 0.7 ccm. of a watery suspension obtained from washing a seven days old culture in six per cent. glycerine agar-agar. Injected subcutaneously in right flank.

May 6th.—Found dead in cage; had been quiet and refused food for past three days. In right flank, at site of inoculation, induration and gray opacity of skin and subcutaneous tissue, with some hemorrhage; marked œdema extending from this spot to right axilla and backward to right groin.

Microscopically, a few bacilli corresponding in size to Lœffler bacilli found in the œdematous fluid. Microscopic examination of blood, lungs, liver, kidneys and spleen negative. Kidneys swollen, opaque and grayish in sections; extensive swelling and degeneration of epithelium in convoluted tubes; a few casts seen in the tubules. From œdematous fluid cultures in two serum tubes showed on following day numerous small colonies of a bacillus corresponding to Lœffler bacillus with well marked involution forms, staining well by Gram's method. Cultures from blood, spleen, lungs, liver and kidneys, all remained sterile.

The remaining cases, except a few of special interest, are briefly summarised in a table, as they were for the most part practically repetitions of the one given above. One case was of special interest and occurred in the private practice of Dr. Major. In this case, which I saw for the first time on the seventh day,

the membrane had nearly all gone, and on the first examination no Lœffler bacilli were found, although two agar tubes and two serum tubes, as well as three Petri dishes, were all seeded from a piece of membrane. In this case an oïdium appeared strongly resembling that of thrush, and I considered the case to be thrush complicated with staphylococcus infection. Dr. Major stated, however, that at the onset the condition was unmistakeably that of diphtheria, and the correctness of his diagnosis was proved some days later by the onset of severe and persistent paralysis of the palate. In the interval I had obtained a fresh piece of membrane which yielded two colonies of the Lœffler bacillus. This case shows that a negative result is of no diagnostic value when the membrane is clearing, even when a fairly exhaustive examination has apparently been made. In another case I found an oïdium growth, readily distinguishable, however, from thrush, associated with a large number of Lœffler bacilli.

The uncertainty of examinations made at a late period in cases going on to recovery was shown in another case, for the opportunity of examining which I am indebted to Dr. W. S. England. In this case I saw the patient on the seventh day. A distinctly membranous exudation had been present, returning within twenty-four hours after being scraped off, but always confined to the tonsils. In this case smear cultures on five glycerine agar tubes failed to show any bacilli, the seeding being done directly from the membrane in the throat. In this case a tiny particle of membrane which had been obtained at the time of examination was seeded on serum a week later, and yielded two colonies corresponding to the Lœffler bacilli, one of which was tested and found to kill a guinea-pig in the typical manner.

In two cases I had great difficulty in obtaining suitable material for examination, owing to the affection being confined to the posterior nares, and where a prolonged local examination seemed unjustifiable owing to the profound exhaustion of the patient. In these cases I made cultures daily from the nasal discharge for several days, but without in any case obtaining the Lœffler colonies. In one of these cases (Case A 2) the nurse afterward obtained for me a small fragment of membrane

from which I obtained colonies having the characteristic culture appearances, and which killed a guinea-pig in the typical manner. In the other case (Case B 2), staphylococcus aureus and citreus were present in enormous number, together with a few streptococci, but no Lœffler bacilli were found.

In a fatal case (Case A 9), where an autopsy was performed by Dr. Finley, who kindly sent me the respiratory organs for examination, the larynx and trachea showed an extensive sheathing of diphtheritic membrane extending down to the main bronchi. Large numbers of the Lœffler bacilli were obtained from this membrane, and an area of pneumonia from the lung showed a small number of the bacilli associated with a large number of small diplococci. No streptococci were found.

In an anomalous case already cited, brought to my notice by Dr. H. S. Birkett (Case B 5), an extensive soft, yellowish membrane covered the posterior nares and extended over the epiglottis and into the larynx. This membrane could be readily removed without causing bleeding, but tended to recur. The general health of the child was unaffected. Cultures on agar yielded an abundant growth of a single bacillus form, forming prominent yellow white surface colonies. Inoculated into the conjunctiva of rabbits, no effect was produced, and I was inclined at the time to regard the bacteria as being possibly the pseudo-bacillus, but on re-investigating the cultures some three months later I found that they gave the typical Lœffler colonies on serum, with an abundant invisible growth on potatoes, and showed on both these media most characteristic involution forms. Inoculated into a guinea-pig, this organism showed a high degree of toxic virulence, killing the animal in thirty-six hours. The autopsy on this animal showed an opaque hæmorrhagic, reddish-gray indurater area at the spot of inoculation, surrounded by a zone of serous œdema. Cultures from the organs and from the serous exudation remained sterile, but typical Lœffler colonies were obtained from the circumscribed hæmorrhagic patch at the site of inoculation. These colonies, on being transplanted on agar, gave only the characteristic, flat, compact, concentric surface growth of the Lœffler organism, and not the abundant

prominent growth of the original tubes. For this reason it appears probable that the original colonies were impure, containing both the Lœffler colonies and some other form, which latter was subsequently eliminated in passing through the serum culture.

In cases which were examined at an early stage the Lœffler bacilli were found almost in pure culture. In most cases the staphylococcus aureus was found, but it was usually scanty. The colonies closely resemble those of the Lœffler bacilli for the first day, but can be distinguished at the end of forty-eight hours. Streptococci were seldom met with, and then only in isolated colonies, the scarcity of this organism being contrary to what I had been led to expect from a study of the literature. In the anomalous or doubtful cases, on the other hand, either staphylococcus and streptococcus, or both together, were present abundantly in all instances. The influence of these pyogenic bacteria on the course of true diphtheria is a point greatly needing investigation.

In cases examined during the period when the membrane had begun to soften—that is to say, after the third day in ordinary mild cases—the falling off in the number of diphtheria bacilli was most marked. This did not seem to be due here to the antagonistic action of saprophytic bacteria, as has been commonly assumed, since, as a rule, the cultures made in late stages in cases going on to recovery remained perfectly sterile, except for the few scattered colonies of Lœffler bacilli. This result I attribute to the inhibitory action of the local disinfectant applications—in most of the cases a spray of peroxide of hydrogen was employed—but I have made no experiments to determine this point.

A matter of considerable interest is the question whether this method of examination will ever come into general use, and if such be the case, what class of practitioners could carry it out to the best advantage. This point is considered by Prof. Welch of Baltimore in an address delivered before the ninety-third annual session of the Medical and Chirurgical State Faculty of Maryland in April, 1891 (*Medical News*, May 16, 1891). In

this address, which by a masterly treatment of the facts places the prophylaxis and treatment of diphtheria on a broad scientific basis, Professor Welch does not think that the hopes of Roux and Yersin, that the method may yield good results in the hands of unskilled persons, are likely to be realised. He thinks that an elementary training in bacteriology is needed in those carrying it out, and suggests that as elementary courses on bacteriology have now become so common that it will not be long before most communities will possess at least one person capable of doing the work satisfactorily. My own experience has shown me that doubtful cases, which are those in which the value of the method should be best shown, the chief difficulty is in obtaining suitable material for examination are of two kinds—first, those where the condition resembles tonsillitis and the exudation is confined to the tonsils; and, second, those where the local disease is situated in some part of the respiratory tract not readily examined, or where the severity of the constitutional symptoms renders a prolonged examination difficult. As the procuring of a small piece of the false membrane at the earliest period possible is the *sine qua non*, and to do that in this latter class of cases requires a special manipulative skill only possessed by a skilled laryngologist, the task will properly fall to this individual. The aid of a throat specialist seems indispenable in cases where the local examination presents much difficulty. Possessed of suitable material, the best results in the further examination would certainly be obtained in a properly equipped laboratory.

In the cases where the confusion arises from the membrane being confined to the tonsils, the case is much more simple, and there is less need of the services of the laryngologist; all that is required being to detach a small piece of the exudation, wrap it in paper, and send it to some laboratory for examination.

Considering the gravity of the interests at stake in the prompt recognition and isolation of cases of diphtheria, one would naturally suppose that a method which enabled a positive diagnosis to be made within twenty-four hours, in cases seen during the first days of the illness, would be welcomed eagerly by the profession. That this has not happened is due probably in great part

to the wise precaution of treating all doubtful cases as if they were cases of diphtheria, and possibly, too, in some degree to a tendency to pride ourselves upon our sagacity, valuing the result of a clever guess more than that obtained by a less brilliant, though more certain, method. As a matter of experience, a large proportion of the doubtful cases, especially the tonsillitis group, declare themselves to be one thing or the other by the time the doctor makes his visit on the following day.

In conclusion, it may be stated :

(1) That in almost all cases where strong clinical grounds exist for the diagnosis of diphtheria, the bacteriological examination has shown the almost invariable presence of the malignant Lœffler bacilli.

(2) That, excepting in connection with scarletina, measles or erysipelas, the number of cases of diphtheritic sore throat due to other causes is very small.

(3) That in doubtful cases the accuracy of the method depends chiefly upon obtaining suitable material at an early stage of the disease.

(4) That the method is not of much service in doubtful cases where the difficulty is due to the infection occurring in localities difficult to examine without skilled manipulation, unless suitable material is obtained for examination.

Summary of Method for Detecting Lœffler Bacilli.

Microscopical Examination.—Stain a cover-glass smeared by a bit of membrane with any aniline dye. The bacilli are arranged in small clumps, and are short, thick rods, about same length as tubercle bacili, but much thicker; numerous beaded and drumstick shapes met with —in solution forms. Gram's staining method can be employed.

Cultures.—Can be made direct from membrane in throat or from small bit of membrane folded dry in clean paper. No special antiseptic precautions necessary. Touch or scrape membrane with a sterilized platinum needle and draw it in parallel streaks over the surface of a serum tube, using two or more successive tubes before reinfecting the needle. Keep the tubes at body temperature. In 20-24 hours the Lœffler bacilli appear as small grayish-white points, size of pin-heads, showing under the microscope the characteristic appearances of the bacilli in the original membrane.

Diagnosis.—(*a*) Other bacilli do not form visible colonies at twenty-four hours. (*b*) Staphylococcus colonies resemble those of Lœffler bacilli to naked eye appearance, but recognized on microscopic examination. (*c*) Pseudo-diphtheritic bacilli have microscopic and culture characters of the Lœffler bacilli, but have no pathogenic properties.

Pathogenesis.—Subcutaneous inoculation of guinea-pig kills in two to five days, with haemorrhagic necrosis and œdema at site of inoculation. The bacilli can be recognised microscopically and by culture near spot of inoculation, but blood and viscera give negative results. Disseminated parenchymatous degeneration of liver and kidneys.

The following is a tabular analysis of the cases, divided into two groups :—

GROUP A.—Cases evidently diphtheria.
Number of cases examined, 9. Result: Positive 8; Negative, 1.

No.	Name.	Sex.	Age	Service.	Course.	Result of Examination.	Remarks.
1	M. L.	F	21	M. G. Hospital.	Recovery	Typical.* Oidium also found.	Began as Tonsillitis,
2	L. E.	M	2	Do.	Death.	Typical.	Nasal diphtheria. Bacili in bit of membrane. None in nasal secretion.
3	Mrs, T.	F	28	Dr. England.	Recovery	Two colonies only.	
4	W. P.	M	16	M. G. Hospital.	Do.	No Lœffler bacilli; abundant staphylococcus pyogenes aureus.	Rapid disappearance of membrane, (nasal voice).
5	L. M.	F	10	Dr. Major.	Do.	Only two colonies found. Oidium present	Marked diphtherial paralysis.
6	F. P.	M	8	M. G. Hospital.	Do.	Typical.	
7	E. G.	F	10	Do.	Do.	Typical.	
8	L. P.	M	13	Do.	Do.	Typical.	
9	M. G.	F	4	Do.	Death.	Typical.	

* In cases marked "typical" a large number of the virulent Lœffler bacilli were found.

GROUP B.—Anomalous cases.*
Number of cases, 5. Result: Positive, 1; Negative, 4.

1	O. P.	F	20	M. G. Hospital.	Recovery	Negative.	Exudate confined to Tonsils.
2	C. W.	F	24	Do.	Do.	Negative.	Nasal diphtheria? Nasal voice. Local examination unsatisfactory.
3	A. C.	M	1	Do.	Do.	Negative.	Local examination unsatisfactory.
5	W. J.	M	8	Do.	Do.	Negative.	Scarletina.
6	S.	M	8	Dr. Birkett.	Do.	Positive.	

* Cases 1, 2 and 3 of this series were placed in the diphtheria ward of the Montreal General Hospital. Several cases of follicular tonsillitis were also examined, always with negative results.

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A NEW METHOD FOR THE CULTURE OF DIPHTHERIA-BACILLI IN HARD-BOILED EGGS.[1]

BY WYATT JOHNSTON, M.D.,
OF MONTREAL.

ALL who have had experience in the diagnosis of diphtheria by culture-methods agree in praising their accuracy and promptitude. Unfortunately, the general practitioner, who must feel most of all the need of some accurate method for the prompt diagnosis of doubtful cases, does not seem disposed to avail himself of the new process, and the prophecy of Roux and Yersin, that the method would come into general use, appears still to be far from fulfilment.

Thinking that the chief obstacle lay in the difficulty of obtaining serum for the culture-medium, M. Sakharof[2] recently suggested a simple plan by which slices of hard-boiled eggs, cut with a sterilized knife and placed in sterilized tubes, could be made to replace the serum.

Of this method I have no personal experience, but should imagine that the main objection would still exist, as the physician might not have test-tubes about him at the time when they were most needed.

[1] From the Pathological Laboratory of McGill College.
[2] Annales Inst. Pasteur, June, 1892.

I have, during the past two months, made use of a method which may be regarded as a modification of Sakharof's, and which does away with the necessity both of test-tubes and the preparation of media before they are actually needed for use.

I employ hard-boiled eggs, from which a part of the shell is removed with ordinary forceps, after being tapped so as to break it. In this way shell and shell-membrane can readily be peeled off from one extremity (by selecting the narrow extremity the air-chamber is avoided), leaving a smooth, glistening, moist surface, which offers a most tempting spot for making cultures. These are made, as in the case of serum, by touching the diphtheritic exudation with a sterilized needle and drawing the latter lightly from three to six times across the exposed white of the egg. Instead of the regulation platinum needle mounted in a glass rod, I employ either an ordinary needle or a bit of silver suture-wire held in an artery forceps. To guard the culture against contamination the egg has only to be placed upside down in a common egg-cup; it can afterward be wrapped in paper and transported, if necessary. The interior of the cup can be sterilized, if desired, by allowing a flame to enter it for a second or two, though I have not found this necessary, as the nutrient surface does not come in contact with the inside of the cup. The egg and shell are, of course, both sterilized by the act of boiling.

Five minutes' boiling suffices, and if the operation has to be done "while you wait," the egg can be cooled in a still shorter time by placing it in cold water. Strict attention to aseptic details is

unnecessary, as the diphtheria-bacillus outstrips in its growth the contaminating organisms likely to lead to confusion. The appearance of the diphtheria-colonies at the expiration of twenty-four hours is the same as when they are grown in serum, but I have found the growth even more rapid, so that a colony is already visible in twelve hours. Confusion with micrococci is, of course, to be guarded against. The reliability of this method seems to be the same as that of the methods of Haffter and E. Roux. I have found one bacillus which attains visible dimensions within the same period, but as this also grew on blood-serum in the manner characteristic of the diphtheria-bacillus, the great value of the method here described is not invalidated by that fact.

Although this minor modification of a now well-tried procedure might enable it to be employed by those destitute of laboratory outfits, I do not think it likely that this means of diagnosis will be utilized by physicians not habituated to laboratory methods.

It may be of interest to state here that the constant temperature of about 35° C., needful to insure the rapid and characteristic growth of the diphtheria-bacillus, can readily be obtained by placing in a cupboard or box with the culture, a large jar or pail of warm water, which is renewed from time to time, thus making an impromptu thermostat.

CORONERS AND INQUESTS.

CORONERS AND INQUESTS.

Where they fail to meet the requirements of the case.

The Medico-Chirurgical Society makes a Study of the Law and suggests Change.

From "THE GAZETTE," February 8th, 1894.

Some time ago the Medico-Chirurgical society of Montreal appointed a committee consisting of Drs. G. P. Girdwood, J. G. Adami, E. P. Lachapelle and Jas. Bell to study the law and manner of conducting inquests in this province. The result has been reported to the committee, and the report transmitted to the Attorney-General and the medical members of the Legislature. Among other things it says :—

The present provincial laws respecting enquiries into the mode and cause of death are based essentially upon the old English common law. The enquiries are placed in the control of coroners appointed by the provincial Government, a coroner for each judicial district. The coroner need not be a member of either the legal or the medical profession, although in the great majority of cases he belongs to one or the other.

Upon receiving notice of a case of death following upon any act of violence, or of death attended by suspicious circumstances, it is his duty to make a preliminary enquiry.

If, with or without medical aid, he comes to the conclusion that the cause of death is to be made out without the assumption of there having been either criminal act or criminal neglect, he can order the interment of the body. If, on the other hand, he is led to suspect that death has been due to violent or unfair means, or culpable or negligent conduct of others, under circumstances calling for investigation by a coroner's inquest, then, having made a sworn deposition to this effect before a magistrate, he is empowered to hold an inquest. What these "circumstances" are which call for investigation is not defined in our statutes, they being left to the coroner to determine. Having made the deposition, he now can summon a jury and hold a coroner's court. He is empowered to call before him such witnesses as in his opinion can throw light upon the cause of death.

The jury must view the body of the deceased, and, if the majority of the jury desire it, the coroner is directed to instruct that an autopsy be performed to throw some light upon the cause of death. Having heard all the evidence, the coroner sums up, and leaves it to the jury to bring in a verdict, and, when this has been delivered, the coroner gives an order for the interment of the body.

The coroner is paid $6 for every inquest, and if an inquest occupies more than two days, $3 for every succeeding day. The practitioner of medicine making an external examination of the body receives $5, making an autopsy he receives $10. There are further fixed charges for the constable who summons the jury and the witnesses, for chemical analyses, for hire of room to be used for the inquest, and for guarding the body.

THE COST.

Taking the returns for Montreal alone, as shown by Dr. Wyatt Johnson, the cost per inquest—that is to say, per case—is decidedly greater than in London, New York or Massachusetts. The rate would seem to be $22 in Montreal, $15 in London, $16.90 in Boston, $12.80 in Massachusetts generally, $10 in New York; and this notwithstanding the fact that autopsies, the most expensive individual item in the investigation of suspicious deaths, are from three to four times as frequent in the other cities as they are in Montreal. Here, in Montreal, it costs more to maintain a dead body in the care of the coroner than it does to maintain an ordinary live individual with healthy appetite at a first-class hotel for the same period. Some of the items permitted by law in the coroner's accounts ought to be lessened or removed altogether, others ought to pass into general police accounts. But the fact remains that the system is as expensive as its results are unsatisfactory, and that the chief source of expense is the legal investigation of cases which do not call for legal investigation at all, owing to the fact of death not having been due to violence. The exclusion of cases not calling for inquest by means of a preliminary medical examination seems to be the most rational means of reducing the expenses.

Your committee is of opinion that, as a matter of principle, the payment of the coroner according to the number of inquests held by him is most unsatisfactory, and is inimical to the proper carrying out of enquiries into the cause of death.

FIXED SALARIES INSTEAD OF FEES.

Your committee find that of the cases of death calling for a coroner's investigation occurring in the various large towns, from 50 per cent. to 75 per cent. can upon preliminary investigation be found to be due to natural causes. That is to say, the more careful the preliminary investigation made by the coroner, and the more conscientious and expert he shows himself in the performance of his duties, the fewer the inquests he finds it necessary to hold, and the less his income if he be paid so much per inquest. While if it so happens that his enquiries lead him to suspect the frequent occurrence of any one form of crime at any period, as, for example, child murder, and so to hold an increased number of inquests upon certain classes of cases, immediately he lays himself open to the charge of seeking to increase his income. This ought not to be. In the cities, at least, the coroners ought to receive fixed salaries.

Under the present system, the jury in Montreal, with rare exceptions, certainly cannot be said to be a capable and representative assembly of citizens. Men engaged actively in any form of business prefer to employ any subterfuge rather than sit for what may be many hours in a morbid atmosphere, for no return whatsoever save discomfort and loss of time. The consequence is that too often the jury is composed of a heterogeneous collection of incapables, gathered from the highways and byeways and bar-rooms of the neighborhood. The verdict of such incapables is, time after time, at variance with the evidence presented.

VIEWING THE BODY.

The custom of viewing the body is as old as the coroner system. It arose at a time when violent deaths were as many as doctors were few, and when population was everywhere so sparse that the jury had an important part to play in determining by external examination that death was due to violence, and, again, in identifying the corpse. Now-a-days, in a large town, it is highly probable that not one of the jury will have known the deceased, and the determination of the cause of death may more safely be left to medical men. In any case, it is easy to obtain identification by means other than the irruption of a strange, unseemly rabble into the house of mourning. The

general feeling throughout the community is that this intrusion into the circle of bereaved relatives in the very depth of their trouble, permitted by the present law, ought to be prevented, and your committee urges strongly that it is as unnecessary as it is unbecoming. It has been superseded in many states by a system of sworn affidavit of the fact of death and the identity of the body, and this course should be followed here.

The existing law does not demand inquest in cases of *felo de se*. This your committee, on the whole, is inclined to consider a disadvantage. The general opinion of the community is strongly opposed to suicide, and were it to be recognized that this mode of death necessarily involved a public investigation, there is little doubt that the unpleasant publicity of the subsequent proceedings would act as a deterrent in not a few cases. As a matter of fact, suicide is on the increase in those states where this deterrent does not exist or has of late years been removed.

MEDICAL EVIDENCE.

A study of the verdicts brought by the coroners' juries shows clearly that the decision of points of medical evidence is a matter that should not be left to non-medical persons. Statements utterly at variance with the cause of death assigned have been time after time accepted blindly by coroner and jury. The appreciation of medical facts, and the opinions to be formed from these facts, come properly within the domain of the medical expert. It cannot be expected that the legal coroner and the jury should without fail form correct opinions upon delicate medical problems.

Your committee strongly approves of the plan adopted in many of the United States, of admitting a written medical deposition of fact or opinion as evidence at inquests in cases where the personal attendance of a medical witness is not considered necessary by the coroner.

THE PERFORMANCE OF AUTOPSIES.

In all the large class of cases now investigated before juries where sudden death occurs without the slightest external lesion, an autopsy is advisable. Nevertheless, with an exception to be presently noted, no autopsy can be performed unless it be demanded by the majority of the jury. That is to say, the jury has to express itself willing to waste an hour or more in the middle of its proceedings, so that a competent medical man may be called, who shall make an examination into the state of the viscera. As a consequence, the jury, in the first place, shows the greatest unwillingness to allow the performance of autopsies, and will the rather return a wholly unreliable verdict. In the second place, the medical man performing the post mortem is at a great disadvantage, for he is expected to keep the jury waiting as little as possible, and his examination, instead of being deliberate and careful, is hasty and liable to be imperfect. Your committee feel assured that were the coroner allowed full power himself to order an autopsy in all doubtful cases a very large proportion of cases would be discovered in which there would be no necessity for holding an inquest and summoning a jury. Thereby a very large expenditure would be prevented, and at the same time the cause of death would be satisfactorily established. The exception referred to above is that by the present law the coroner is permitted to order an autopsy if he makes an affidavit that he holds the autopsy to be necessary. Unfortunately, coroners do not seem to have taken advantage of this permission, but prefer to shelter themselves by leaving the matter wholly in the hands of the jury.

PRELIMINARY INVESTIGATIONS.

In all cases of suspicious death the first question to be settled is what has been the immediate cause of death. In all cases, therefore, the first point to be investigated is purely medical. It is true that frequently the question is one that can be answered by any individual endowed with common sense, as, for instance, when a corpse is discovered upon the railroad track minus its head, though even in such cases serious mistakes have occurred through the bodies of murdered persons being so placed as to give an impression of accidental death. But if the question in certain simple cases can be answered by a layman as well as by a

professional man, there is a very large number of cases, and these often the most important from a medico-legal aspect, where a correct determination can only be reached by a well-qualified medical man, and where it is all important that a correct answer be gained at the outset, not only for the benefit of the relations of the deceased (that they be sheltered from the least breath of unnecessary suspicion), but also for the benefit of the provincial exchequer, that the province be not saddled with the cost of an inquest leading to no result. When more than 50 per cent. of all deaths which coroners are called upon to investigate are found to be from natural causes, it is evident that the majority of deaths now investigated require no legal investigation whatever, while, on the other hand, as indicated above, all such deaths demand an initial investigation by a medical man.

CRIMINAL CASES.

Under the existing law, when his jury brings in a verdict of murder or manslaughter, or of being accessory to murder before the fact, against any person or persons, the coroner must issue a warrant against such person or persons, and send him or them before a magistrate or justice, if this has not already been done. He must at the same time transmit the depositions taken before him in the matter. To all intents and purposes, the trial before the magistrate proceeds as though no previous enquiry had been held. The coroner's depositions are not employed as evidence. In fact, the magistrate treats the case as though he were proceeding under an ordinary warrant. If the magistrate confirms the charge, the case is sent up to the grand jury, and here again all the witnesses are once more summoned and the evidence is repeated, and the grand jury finding a true bill, the case goes before the petit jury, and again the evidence is repeated.

It appears to your committee that this proceeding is singularly cumbrous, and that, besides harassing the witnesses, it allows an unduly large number of loopholes of escape for those guilty, upon some legal technicality or faulty observance of legal procedure. Your committee, considering that the problem of how this procedure may be simplified is a purely legal one, does not offer any suggestions on the matter.

Taking all these disadvantages into consideration, and being especially impressed by the fact that the earliest stages in the investigation of suspicious death must of necessity be of a medical nature, and by the further fact that where the legal proceedings of the coroner lead to a definite charge against an individual or individuals, those legal proceedings are practically passed over unnoticed by the higher courts, your committee have come to the conclusion that a drastic change in the mode of investigation of suspicious deaths is advisable in this province.

THE CORONER'S PERSONALITY.

There are two questions which naturally suggest themselves prominently in connection with questions of coroners' reform. The first is, Should the coroner be a physician or a lawyer? and the second, Should the office of coroner be abolished?

With regard to the qualifications necessary for coroners, your committee does not think it necessary to dwell upon the relative advantages of having medical or legal coroners, although this is a subject of dispute which has now been fruitlessly discussed for more than a century, and will in all likelihood continue to be so as long as the coroner system lasts.

In London a settlement of the question has been attempted by selecting as far as possible coroners who have obtained both legal and medical qualifications. This plan of expecting the coroner to be a jack-of-all-trades has not much to recommend it; and the fact that in London, in addition to the doubly qualified coroner, there are the deputy coroners, who are obliged by law to be barristers, and all the medical expert work is done by outside men, shows that matters are not in any way simplified even by having the coroners who are at once both lawyers and physicians.

The only rational plan, and one whose advantages appear never to have been questioned, is that adopted on the conti-

nent, as well as in those states which are now under the medical examiners system, of separating as far as possible the medical and legal side of the investigation, and entrusting these to physicians and lawyers respectively. Your committee is just as firmly convinced that all legal questions should be left wholly to lawyers, as that all medical ones should be entrusted to medical men.

ABOLITION OF THE OFFICE OF CORONER.

Your committee finds that in those states where this has been done, the previous difficulties seem to have been promptly and permanently removed, and it does not appear to have been necessary in any instance to revive the office. The office of coroner was created in England while that country was in a lawless state, and when police regulations and courts of justice were almost non-existent. Since the development of the judicial and police system, the coroner's office has gradually come to fill the important function of fifth wheel to the car of justice. It has been retained through that conservative spirit which retains the cumbrous system of pounds, shillings and pence for the national currency. Many of the United States are still in that primitive and lawless condition, which makes the office of coroner a useful one. In the more highly civilized states the old coroner system is rapidly disappearing, and it is practically obsolete in five, viz.: Massachusetts, Rhode Island, Connecticut, New Jersey and New Hampshire.

As to whether the office of coroner should be abolished in our own province, we have no hesitation in stating, as medical men, that, from a medical point of view, the office is simply an absurdity, which constantly interferes with the proper employment of medical science for judicial ends, and that it could be abolished to-morrow with marked benefit to the medical side of criminal cases.

The fact that the appointment of competent medical experts as consultants to the coroner's court of Montreal during the last year has neither prevented nor greatly diminished the number of those palpably absurd and unsatisfactory verdicts, which have made this court a public laughing-stock in past years, shows that something must be radically wrong with the system, which must be remedied, even if this necessitates abolishing the office.

On the other hand, we do not feel, as medical men, competent to decide as to the possible effects which would be produced by this change from a judicial point of view. If the office of coroner were abolished, the legal duties would have to be provided for in some way, the details of which can only be decided by persons thoroughly conversant with the workings of our criminal law. Furthermore, the abolition of the office of coroner does not appear to your committee to be absolutely necessary in order to secure the necessary medical reforms. All that is really necessary is to do away with the medical functions and responsibilities of the coroner and to make the office a purely judicial one, only dealing with those cases where there are definite grounds to suspect death from violence or negligence and these grounds are either strengthened or not removed by the examination of a medical expert.

A COMPROMISE IDEA.

We would therefore recommend :

1. That salaried medical examiners be appointed to investigate all deaths occurring under circumstances calling for medico-legal investigation under any act, and that these officers be given authority to make such medical examination of the body as may be necessary to determine whether death was due to violence or not ;

2. That in every case the medical examiners report the result of their examination to the coroner or other judicial officer charged with investigating the legal side of such cases, who, in case of violent death, shall make such investigations and take such measures as are necessary for the proper administration of the law.

STATISTICS

—OF THE—

CORONER'S COURT FOR THE DISTRICT OF MONTREAL

1893.

BY

WYATT JOHNSTON, M.D., AND GEO. VILLENEUVE, M.D

(*Reprinted from the Montreal Medical Journal, March,* 1894.)

STATISTICS OF THE CORONER'S COURT FOR THE DISTRICT OF MONTREAL, 1893.

By WYATT JOHNSTON, M.D., and GEORGE VILLENEUVE, M.D., Montreal.

The number of deaths investigated during the year was 386. Calculated from the census of 1891, the population of the judicial district of Montreal is rather more than 350,000, of which about 280,000 is urban, making a yearly rate of about 1.1 inquests per 1,000 inhabitants of the district.

In 184 of the 386 cases we were summoned to testify as experts. In the remaining 202 cases the information is obtained from the very complete public records of inquests kept by Coroner McMahon. Of these there were 36 cases in which no medical testimony was taken. In the remaining 166 cases, the medical evidence was given by other physicians, who in 60 cases had either not seen the deceased professionally during life or only at a time remote from the death. In 88 of the cases the evidence was taken before the coroner alone, without a jury.

We give the following details which seem of interest.

Of the bodies, 276 were males and 109 females. In one case, that of a newborn child, the sex is not stated.

Exclusive of the new born infants, the identity of 5 bodies remained unknown, being in each case persons found drowned.

CLASSIFICATION BY AGES.

Age...	New-born.	Under 1 yr.	1 to 5 years	5 to 10 yrs.	10 to 20 yrs.	20 to 30 yrs.	30 to 40 yrs.	40 to 50 yrs.	50 to 60 yrs.	60 to 70 yrs.	70 to 80 yrs.	80 to 90 yrs.	90 to 100 yr.	Unknown.	Total.
Cases.	14	11	23	14	25	55	46	58	44	34	17	15	3	27	386
Per ct.	3·6	2·8	5·9	3·6	6·5	14·3	11·9	15·1	11·4	8·8	4·4	3·9	0·8	7·0	100·0

CLASSIFICATION BY MONTHS.

Month.....	Jan.	Feb.	March.	April.	May.	June.	July.	August	Sept.	Oct.	Nov.	Dec.	Total.
Cases.....	23	35	35	28	36	44	33	40	27	41	26	18	386
Per centage	5·9	9·1	9·1	7·2	9·3	11·4	8·5	10·4	7·1	10·6	6·7	4·7	100·0

NUMBER OF AUTOPSIES HELD, WITH THEIR PROPORTION TO THE NUMBER OF INQUESTS IN EACH MONTH.

Month.....	Jan.	Feb.	March.	April.	May.	June.	July.	August	Sept.	Oct.	Nov.	Dec.	Total.
Autopsies..	3	2	1	6	4	10	5	9	5	8	2	1	56
Percentage	13·1	5·4	2·8	21·4	11·1	22·7	15·1	22·5	18·1	19·5	7·7	5·5	14·5

CLASSIFICATION BY VERDICTS AND CAUSES OF DEATH.

I.—HOMICIDES.

Firearms	1
Blows and falls	4
Criminal negligence	4
Infanticide	3
Total	12

II.—SUICIDES.

Firearms	5
Cutting throat	3
Hanging	2
Drowning	1
Jumping from height	1
Railways	2
Poisoning	9
Total	23

III.—ACCIDENTS.

Firearms	4
Machinery	3
Elevators	3
Railways	23
Street railways	8
Horse and carriage	14
Tobogganing	1
Burns, scalds, burning buildings	22
Explosions	3
Falls from height	11
Falling bodies	11
Drowning	41
Choking by food	3
Overlaying	3
Illuminating gas	7
Administration of chloroform	2
Poisoning	11
Exposure	2
Sunstroke	1
Other causes	1
Total	174

IV.—NATURAL AND UNKNOWN CAUSES.

Circulatory System (40)—	Heart disease	39
	Aortic aneurism	1
Respiratory System (35)—	Pneumonia	15
	Phthisis and hæmoptysis	17
	Bronchitis	1
	Pulmonary embolism	1
	Other diseases	1
Digestive System (12)—	Diarrhœa	5
	Peritonitis	2
	Other diseases	5

Nervous System (18)—	Apoplexy	10
	Congestion of brain	2
	Other diseases	6
Genito-Urinary System (5)—	Chronic nephritis	1
	Urethral fever	2
	Puerperal fever	
Developmental Diseases—	Stillborn	5
Infectious Diseases—	Typhoid	1
	Diphtheria	1
General Diseases (10)—	Purpura and scurvy	2
	Senile debility	4
	Infantile debility	4
	Other diseases	2
Habits and Occupations—	Intemperance	5
Unknown causes		45
	Total	177

SUMMARY OF THE PRINCIPAL GROUPS OF CAUSES OF DEATH, GIVING THE PERCENTAGE WHICH THEY FORM OF THE TOTAL INQUESTS AND ALSO THEIR FREQUENCY, PER 10,000 LIVING.

Verdicts.	Number.	Per cent.	Per 10,000 living.
Homicide	12	3·11	0·34
Suicide	23	5·96	0·65
Accidents	174	45·07	4·96
Natural and unknown causes	177	45·86	....
Total	386	100·0	....

Deaths from Homicide.

A few remarks upon these different groups may not be out of place.

We have no case of conviction for murder or manslaughter to record. Of 12 verdicts of homicide, 3 were for infanticide by persons unknown. In none of these was the guilt brought home to an individual. Of the 9 remaining cases: in one the violence took place in Ontario and therefore has no bearing upon the criminality of this district; two charges, both for manslaughter, are still before the courts; one case was discharged by the police magistrate; in 5 cases the grand juries found no bill, and the only one which came before the Court of Queen's Bench was dismissed by the judge without calling for the defence. In none of the cases was there any evidence of malice.

The results of the incriminating verdicts of coroner's juries here appear to be of a surprisingly mild and harmless character. In a number of cases of accidental death, verdicts of "negligence not criminal" were found.

Deaths from Suicide.

SEX AND SOCIAL CONDITION.

MALES.		FEMALES.	
Married	12	Married	1
Unmarried	4	Unmarried	2
Not stated	2	Widows	2
	18		5
Total	23		

SUICIDE: CLASSIFICATION BY AGE.

Age	Below 20.	20 to 30.	30 to 40.	40 to 50.	50 to 60.	Above 60.	Total.
Number	1	4	4	8	4	2	23

SUICIDE: CLASSIFICATION BY MONTHS.

Month	Jan.	Feb.	March.	April.	May.	June.	July.	August	Sept.	Oct.	Nov.	Dec.
Number	0	0	2	2	4	4	1	3	1	2	2	2

The occupations of the male victims were as follows:—Lawyers, 1; farmers, 1; merchants and clerks, 4; workmen and servants, 12.

The apparent causes were as follows:—Business trouble, 1; family trouble, 1; unknown, 1; physicial disease and suffering, 2; love, jealousy, or dissipation, 4; drink, 5; mental disease, 9.

It is reassuring to note that the frequency of suicide, viz., 0·65 per 10,000, in Montreal, is low in comparison with most other large American cities, the rate per 10,000 living in 1891, having been as follows: Baltimore, 0·56; Philadelphia, 0·76; Boston, 1·11; New York, 1·57; Chicago, 1·87; St. Louis,

2·16; San Francisco, 2·66. In Paris, the annual rate of suicide is about 3·78 per 10,000.

Of 9 cases where poison was the means employed, 7 were by Paris green; and one from rough-on-rats. Some means should be taken to restrict the too ready sale of such deadly commodities. The suicides by firearms, cutting throat and hanging, were all in males.

Accidental Deaths.

The commonest causes of accident, in order of frequency, were:—

Drowning	41, or 23·5	per cent.
Railways	23, or 13·2	"
Burns, scalds and fires	22, or 12·6	"
Horses and carriages	14, or 8·2	"
Falls from height	11, or 6·3	"
Falling bodies	11, or 6·3	"
Poisoning	11, or 6·3	"
Street railway	8, or 4·4	"
Illuminating gas	7, or 4.1	"

Drowning.—The number of drowning accidents is sufficiently high to attract attention. Of the 41 cases, the accidental nature of the drowning was well established in 32 cases only, all that could be stated about the remaining 9 bodies being simply that they were found in the water.

Illuminating Gas.—The number of deaths from illuminating gas is also surprisingly large. The danger of gas depends upon the proportion of carbon monoxide it contains, and this again depends upon the mode in which the gas is prepared. It has always been asserted that the gas supply of Montreal contains a very small proportion of this dangerous ingredient, but the large number of deaths from this cause would seem to render the subject worthy of investigation by our health authorities.

The accidents happening in connection with public travel naturally have a great public interest.

Fatal Railway Accidents.—The victims were trespassers in 10 cases; employees (brakesmen or switchmen) in 6 cases; travellers in 4. In 4 cases the accidents occurred at level crossings.

Street Railways.—Of 8 fatal accidents during the past year, 5 occurred in connection with electric cars and 3 with horse

cars. Two of the victims were employees; 1 was a child playing upon an empty car on a siding; 3 were persons crossing the street, and 2 were passengers (one of whom got off the car, while in motion, on the wrong side and was struck by an approaching car on the other track; the other was killed while trying to pass from one car to another while in motion). None of the accidents were shown to be due to the use of the trolley system or the over head wire.

Elevators.—Three deaths which occurred from elevator accidents were all due to imprudence of the persons killed, none arising from any defect in the machinery.

In most of the accidental deaths the cause was clearly established by the evidence of eye witnesses, and the investigations were conducted chiefly with a view of deciding whether there had been criminal carelessness or not.

Deaths from Natural Causes.

In 130 cases definite natural causes were assigned, and in 47 the finding was either "natural causes," "unknown causes," or "unknown natural causes." The nature of the cause of death was almost always made out when autopsies were made out, and, therefore, the expression unknown means that, in most cases, no attempt was made to find out the cause of death. It has not seemed advisable to attach any importance to the relative frequency of the known and unknown causes shown in the tables, because, in a large number of cases, definite causes were assigned for the death without any proof at all being adduced that the alleged causes really existed.

There is no doubt that a large number of the deaths investigated during the year were, directly or indirectly, due to drink, but as the conclusions of the juries on this point appear to have been arrived at by intuition rather than by investigation and weighing of the evidence, definite statements on this head based on the verdicts would be misleading. We have, therefore, refrained from attempting to estimate the exact proportion of deaths due to intemperance.

A BIOLOGICAL ANALYSIS

—OF THE—

MONTREAL WATER SUPPLY DURING THE PERIOD FROM NOVEMBER, 1890, TO NOVEMBER, 1891.

BY

WYATT JOHNSTON, M.D.,

Lecturer in Bacteriology, McGill University: Bacteriologist to the Quebec Provincial Board of Health.

(*Reprinted from the Montreal Medical Journal, August,* 1894.)

A BIOLOGICAL ANALYSIS OF THE MONTREAL WATER SUPPLY DURING THE PERIOD FROM NOVEMBER, 1890, TO NOVEMBER, 1891.*

By WYATT JOHNSTON, M.D.

Lecturer in Bacteriology McGill University; Bacteriologist to the Quebec Provincial Board of Health.

The following account of a biological analysis, made three years ago, has been abridged from my report addressed at the time to Mr. B. D. McConnell, then Superintendent of the Montreal Water Works, who took a deep interest in the investigation. Chemical analyses were made at the same time by Prof. R. F. Ruttan and Prof. Phister.

PLAN OF INVESTIGATION.

I. Regular monthy examinations of samples of water from the following four localities :

1. The lower reservoir.
2. The settling basin.
3. A point near the intake of the St. Cunegonde Water Supply.
4. A point in the middle of the River St. Lawrence south of Nun's Island.

These examinations were made at the express order of the Water Committee with a view of determining whether the water

* Published by permission of the Water Committee of the Montreal City Council.

obtained from localities 3 and 4 would be preferable to that furnished by the present intake on the north shore of the St. Lawrence, just above the Lachine Rapids.

In addition, I found it necessary to make :

II. Examination of tap water obtained from various points within the city, from the upper reservior, and from the aqueduct,

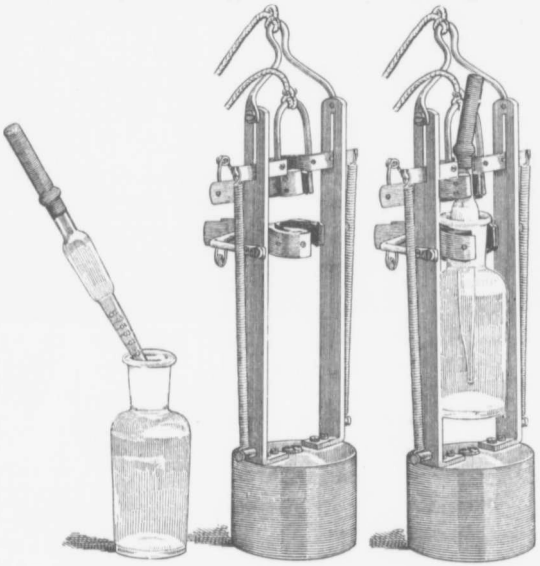

Fig. 1.

to see whether evidences of local contamination existed and to trace the effect of temperature, rainfall and water level.

III. Examination of the water of the St. Lawrence and Ottawa rivers at points above Montreal, to see whether the influence of the sewage from the towns along their banks was perceptible.

IV. Examination of surface waters from other parts of Canada, and especially from uninhabited districts.

METHODS.

A large proportion of the work consisted in the estimation of the number of bacteria present. (Quantative bacterial analysis.) The nature of the bacteria was also studied, as far as the time limits of the analysis permitted. Cultures, for quantitative work, were for the most part made in slightly alkaline, 10 p.c. beef peptone gelatine, made after Lœffler's formula, and grown at 20°C. The samples were taken 10 to 20 feet below the surface, by means of an apparatus shown in figure 1, and were plated in flat glass vials. The cultures were, as a rule, made within a few minutes of the time of taking the samples, and in a few instances, when about an hour or two intervened, the samples were kept in an ice box.

The sediments were all examined microscopically, and during four months the microscopical organisms present were estimated quantitatively by the Sedgwick-Rafter method.

SOURCE OF SUPPLY.

Before giving the details of the analysis, it might be well, in order to make the report intelligible to those who are not familiar with the local conditions of the Montreal water supply, to briefly mention the character of the water, and the topography of the district from whence it is obtained.

Although taken from the north shore of the St. Lawrence river, the Montreal water supply is derived, during the greater part of the year, from the Ottawa, which enters the St. Lawrence from the north at a point about 20 miles above the intake, and forms a belt of dark water close to the shore, the border between this water and the clear green of the St. Lawrence proper being very distinct, though varying in position with changes in the direction and force of the wind and the relative level of the water in the two rivers. During the winter owing apparently to an ice-jam, the Ottawa passes to the north of the island of Montreal, so that the Montreal supply during the months of January, February and March consists of nearly pure St. Lawrence water.

Ottawa River.

The Ottawa river drains an area of over 60,000 square miles

(rather less than the Danube), most of which is entirely uninhabited. Its discharge has been estimated at 60,000 cubic feet per second. Its average width for the 100 miles above Montreal is somewhat over half a mile. At 25 miles above the city it expands into the lake of Two Mountains, varying from 2 to 4 miles in width, and 4 miles above the intake, into Lake St. Louis, 4 to 7 miles wide. There are rapids and falls 60 and 30 miles above Montreal. At many points between Ottawa and Montreal navigation is impeded by enormous sawdust beds from the Ottawa saw mills.

The population along its course, according to the census of 1891, is about 300,000, or 6 per square mile, of which about 100,000 is comprised in cities or towns of over 1,000 inhabitants, the remainder being rural. The chief centres of population and their distances above the Montreal intake are as follows:

Pembroke	4,401	220 Miles.
Renfrew	2,611	190
Perth	3,136	180
Smith's Falls	3,864	175
Aylmer	1,945	140
Ottawa (and Hull)	55,429	125
Buckingham	2,239	100
Hawkesbury	2,042	60
Lachute	1,751	50
St. Anne	1,500	20
Lachine	3,167	4

The Ottawa water is dark, and contains a large amount of peaty pigment, giving the water, when in a deep column, a tint suggesting that of porter. Apart from this it is stated by Prof. Ruttan to contain almost no organic matter. It is much softer than the St. Lawrence water.

St. Lawrence River.—The St. Lawrence drains an area of 510,000 square miles (half as much as the Mssissippi). Its discharge, before receiving the Ottawa, has been estimated at 500,000 cubic feet per second. Apart from the cities and towns, situated upon the Great Lakes or on streams draining into them, the total population of the towns and villages of over 1,000, situated upon the river proper, amounts to about 55,000, of which Kingston (20,000) is really in Lake Ontario. The

populations and distances above the intake at Montreal are as follows :

Kingston	20,000	185 Miles.
Gananoque	3,669	150
*Clayton	4,400	130
*Prescott	2,920	120
*Ogdensburg	11,662	120
Cornwall	6,085	70
*Valleyfield	3,315	35
*Beauharnois	1,590	20

Towns marked * are on the south side of the river.

The river averages fully one to two miles in breadth during the whole of its course, and expands into Lake St. Louis, 4 to 7 miles wide, just above the intake, and into Lake St. Francis, 8 miles wide, 35 miles above. There are rapids at points 20, 25, 30, 35, and 80 miles above the intake.

The St. Lawrence water is clear and light green in colour, and is fairly hard.*

In both these rivers the temperature falls to the freezing point in winter, even at points near the bed of the stream.

I. Monthly Examination of Water Supply.

Microscopical Analysis.—The method employed was, at first, that of simply allowing the sediment to settle in a conical glass, and by means of a pipette placing a little of it under a microscope. This gives a general idea of the constituents of the sediment, but affords no information as to the quantity in which the different organisms are present. In the Sedgwick-Rafter method (which unfortunately only became known to me after the analysis was completed) a given

* The following table compiled from Dr. Ruttan's analyses shows the average chemical composition of Ottawa and St. Lawrence water (quantities in part per million) :

	Color. Lovibond scale.			Solids.			Nitrogenous Matter.			Oxygen consumed on 80°F.		Hardness (as Calcium Carbonate.)	Chloride (as Chlorine.)
	Red.	Yellow.	Blue.	Total.	Loss on ignition.	Ash.	Free Ammonia.	Albumenoid Ammonia.	Nitrates.	15 min.	4 hrs.		
Ottawa.	1.7	5.4	0 04	52	24	28	0.02	0.12	0.03	3.7	6 4	55	1 5
St. Lawrence.	0.1	1.0	0.47	142	69	74	0 01	0.09	0.09	0 5	1.2	102	3.5

quantity of the water, usually 500 cc., is filtered through sand and the sand with the organisms retained in it shaken up with a definite quantity of distilled water, 1 cc. of this is then placed in a glass cell, leaving a superficial area of 1,000 square millimetres and a depth of 1 millimetre. By examining under a microscope, into the eye piece of which a diaphragm has been fitted covering exactly 1 square mm. with the objective employed, each microscopic field represents a fixed unit of measurement with reference to the original water, and the number of each different organism per cc. can be calculated from the average number present in each field. As a rule the genera only are determined. This method is not applicable for determining the number or character of the bacteria.

During the period from March to November, 1891, the presence of the following organisms was noted. The numbers represent the number of different genera found in one sample and not of individual organisms per c.c. :

Month 1891.	Mar.	Aprl.	May.	June.	July.	Aug.	Sept.	Oct.	Nov.
Sample from :									
Reservoir	*	6	12	16	*	8	8	5	9
Settling Basin.	5	3	15	9	*	10	5	*	8
St. Cunegonde.	4	4	31	18	13	11	5	5	12
St. Lawrence..	*	3	3	12	18	14	5	5	9

* Not estimated.

Of these, the following genera were the most frequent :

DIATOMACEÆ. — *Acnanthes, Amphora, Asterionella, Cyclotella, Diatoma, Encyonema, Epithemia, Fragilaria, Gomphonema, Melosira, Navicula, Nitzschia, Pleurosigma, Stauroneis, Surirella, Stephanodiscus, Synedra, Tabellaria.*

CYANOPHYCEÆ.—*Anabæna, Oscillaria.*

OTHER ALGÆ.—*Chara, Cladophora, Œlospharium, Conferva, Cosmarium, Palmella, Pleurococcus, Pediastrum, Vaucheria, Volvox, Penium, Protococcus, Scenedesmus, Tetraspora, Zygogonium.*

FUNGI.—*Crenothrix.*

RHIZOPODA.—*Actinocyclus, Actinophrys, Amæba, Gromia,*

INFUSORIA.—*Bursaria, Carchesium, Dinobryon, Epistylis, Euglena, Heteronema, Monas, Paramœcium, Trachelocerca, Trachelomonas. Vorticella.*

SPONGIARIA—*Sponge spicules.*

VERMES.—*Anguilcula, Monostylus, Rotifer, Stylonychia. Stentor.*

CRUSTACEA.—*Alona, Cyclops, Daphnia.*

As I had not been able to employ the quantitative method during the year of analysis, I give the results obtained, per c.c., from tap water during the period from April 10th to June 4th, 1892, in the following table :

Date of examination	April 30.	May 6.	May 15.	May 28.	June 4.
Number of sample	62	63	64	65	66
DIATOMACEÆ.	**64**	**84**	**56**	**42**	**322**
Acnanthes	2	0	0	0	0
Amphora	3	pr	0	0	0
Asterionella	21	36	18	12	20
Cocconeis	0	0	0	1	0
Cyclotella	pr	pr.	2	0	1
Cymbella	1	pr.	0	0	0
Diatoma	2	0	pr.	0	0
Encyonema	0	0	2	0	0
Fragilaria	pr	6	pr.	2	0
Gomphonema	1	pr.	pr.	0	0
Grammatophora	0	0	0	pr.	0
Melosira	23	24	21	2	5
Navicula	9	9	pr.	1	300
Nitzschia	0	4	11	2	0
Surirella	0	0	1	0	0
Synedra	2	4	0	22	1
Tabellaria	0	3	0	0	0
ALGAE.					
Chlorococcus	0	32	0	0	0
Protococcus	0	2	1	0	0
Zoospores	pr	pr.	pr.	pr.	10
INFUSORIA.					
Monas	0	2	pr.	0	0
MISCELLANEOUS.					
Starch grains	3	2.5	2	2	4.5

I have omitted from the table the following genera which, though occasionally seen, were never present in an amount equal to 0.5 per c.c. : —*Coscinodiscus, Pleurosigma, Stauroneis, Stephanodiscus, Oscillaria, Arthrodesmus, Cladophora, Calosphærium, Conferva, Pediastrum, Pleurococcus, Beggiota, Amœba, Cercomonas, Trachelomonas, Spongilla and Cyclops.*

The organisms were more numerous in the warm than in the colder months. The higher animal forms being only met with during the summer.

Pollen grains (most commonly from the pine) and vegetable

fibres were usually present in traces, and were most constant in the samples from the reservoir.

From the above results it will be seen that while the waters contain small amounts of the non-bacterial organisms common to all surface water, these were never found in sufficient quantity to affect the odor, taste, or hygienic quality of the water. Of the organisms, the diatoms Melosira and Asterionella were the only ones occurring constantly in any appreciable quantity.

The green organism (Anabœna) which abounds in the water of Lake Ontario and the Bay of Quinte during the summer, was scarcely detected at Montreal, though owing to the infrequency of the periods of collecting samples it may have been missed. Though present in the reservoir during August and September very little appeared to enter the supply pipes.

The results of examination of sediments, on the whole, were decidedly satisfactory from a hygienic point of view.

Starch Grains.—The only anomalous features presented by the sediments was the constant occurrence of starch grains in the sediment of most of the samples. These I first noticed in the May samples, they being present in the water from the reservoir, settling basin and St. Cunegonde, but not in that from the St. Lawrence.

These grains were usually round or slightly oval, or in some cases presented blunted angles. They measured 12 to 30 microns in diameter, stained blue with iodine solution and polarized with a central cross. Some showed a central fissure in the form of a slit or cross, and often lamination could be distinctly made out.

I was at first disposed to regard them as an accidental contamination, due to the entrance of dust into the samples, but this was shown not to be case by the fact that upon filtering water directly from the tap through glass wool, compressed into a small strainer, the starch was invariably detected, while the materials employed as well as the glass-ware used, showed no signs of it.

Upon consulting the standard works on water analysis, I was unable to find any reference to the presence of starch in water otherwise than as a consequence of contamination by sewage proper, kitchen refuse, or the waste of industrial establishments. On the other hand, all the other results of my analysis were strongly opposed to the theory of contamination of the water.

Being myself unable to identify the grains satisfactorily with any of the known starches, I consulted Prof. D. P. Penhallow, of McGill University, who examined them carefully and called my attention to the fact that they corresponded in size and shape and structure to corn starch grains, and were much larger than any of the starch grains found in aquatic plants.

He stated that, in his opinion, the only starch bearing aquatic plants at all likely to lead to dissemination of starch grains in the water were the yellow and white water lillies (*Nymphæa* and *Nuphar*) the starch grains of which, however, never exceeded 13 microns in diameter, and were readily distinguished, by their form and arrangement, from the granules under consideration.

If the grains were corn starch then they must have come from some starch factory or grist mill.

There were, however, no starch factories or large milling industries along the banks of the Ottawa, and though some starch factories are situated upon the St. Lawrence, none of the grains had been found by me in that water.

Upon estimating the number of starch grains per cc., I obtained the following results, for different seasons of the year, from samples of the water which happened to have been preserved :

Month.	Mar.	Aprl.	May.	June.	July.	Aug.	Sept.	Oct.	Nov.
Sample.									
Reservoir.....	*	pr.	*	2	*	*	*	3	2
Settling Basin.	0	*	*	pr.	*	1	*	*	2
St. Cunegonde.	0	*	0,8	4	4	*	*	5	2
St. Lawrence..	*	*	*	0	0	*	*	0	0

* Not examined.

The largest amount of starch ever found in any sample was 7 granules per c.c., in a stagnant rusty sample, obtained from a street hydrant.

The presence of the starch in the Ottawa water and its absence from the St. Lawrence, was a matter which completely puzzled me. Examination of the starch granules of the sweet

Fig. 2—Starch grains from water.

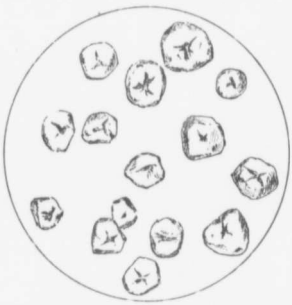

Fig. 3—Starch from white pine bark.

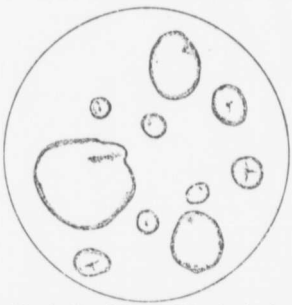

Fig. 4—Starch from white pine bark after soaking in water.

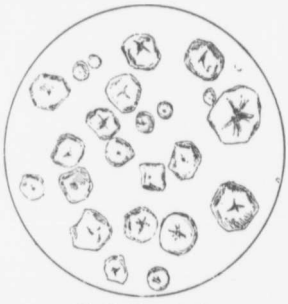

Fig. 5—Corn starch.

flag root and wild rice, showed that these grains were altogether too small to be thought of as a possible source.

At this point, Prof. G. P. Girdwood, of McGill University, suggested to me the possibility that as starch is present in the

bark of some of the coniferous trees, it might be derived from the white pine lumber which, as already stated, is sawn in such large quantities as to block the Ottawa river in places with vast beds of sawdust. Upon my examining white pine bark, I was delighted to find not only that it contained large quantities of starch, but that these, though somewhat more angular, closely corresponded in size, shape and structure with the grains found in the water (and closely resembled corn starch).

Upon soaking pine bark for two months in water, many of the starch grains in it assumed the rounded outline typical of the starch of the water sediments, whereas corn starch grains, after the same period of maceration, became fissured and tended readily to disintegrate upon slight pressure.

The appearance of the various grains may be better understood from the accompanying illustrations, figs. 2, 3, 4 and 5.

Starch grains similar to those of the pine were found, though less plentifully in the bark of the cedar, hemlock and spruce.

The following table gives the diameter in micro-millimeters of the various starches examined :

	Diameter in microns.
Water Sediments	11.4 to 28.0
White Pine Bark	8 to 28.0
Corn	5.8 to 27.0
Sweet Flag	6.0 to 13.0
Wild Rice	5.7 to 13.0
White Water Lily	1.9 to 7.6
Yellow Water Lily	3.8 to 13.3

There is nothing to show that the starch forms a dangerous ingredient of the water. I have also found somewhat similar grains under circumstances which did not show any possibility of sawdust pollution, and unless great care is exercised one is liable to meet with them as a result of contamination of the glass-ware, etc., by dust.

My excuse for giving the above results at such length, is that it does not seem to have been recognized as yet that starch

grains may be observed in water independently of sewage or industrial pollution on the one hand, and of errors in manipulation on the other.

Bacterial Analysis.—The opinion entertained by chemists of the Montreal water supply, at the time when this examination was undertaken, is fairly well expressed in Bulletin No. 15 of the Inland Revenue Department at Ottawa, which in referring to the relatively high proportion of organic matter, speaks of it as " capable of sustaining and nourishing, to a much greater degree than in most water supplies, those minute organisms which, while in most cases harmless, are closely related to others known as disease germs. A water so largely impregnated with organic matter, as that of the Ottawa, would become a very efficient nidus for the propagation of morbific bacteria were such organism to find an entrance to it."*

It may be stated in a general way that a pure water should not habitually contain large numbers of bacteria. Although no hard and fast rule can be set, Miquel's scale fairly expresses our present ideas upon the relation of the number of bacteria to the purity of water :

Exceptionally pure water	contains	0	to	10 per c.c.
Very pure	"	10	to	100
Pure	"	100	to	1,000
Mediocre	"	1,000	to	10,000
Impure	"	10,000	to	100,000
Very impure	"	100,000	and over.	

The number of bacteria in filtered water should not, according to Koch, habitually exceed 100 per c.c.

I was agreeably surprised to find that the Montreal water, instead of teeming with bacteria, was conspicuously free from them, as compared with other bodies of running water, so that whatever might be the nature of the organic matter present it did not appear to be specially favourable to bacterial growth.

The following table shows the average number of bacteria found in some well known surface waters, most of which are

* McGill, Bulletin No. 15, Department of Inland Revenue, Ottawa.

used as sources of drinking water. These marked * are filtered before being distributed :

		Locality.	Authority.
Ottawa	220	Montreal	Johnston.
St. Lawrence	360		
Mississippi	800	St. Louis	Currier.
*Danube	2,000	Vienna	Kowalsky.
Seine	33,000	Above Paris	Miquel.
*Thames	19,750	Above London	P. Frankland.
Croton Aqueduct	4,280	New York	Health Report.
Hudson	3,065	Albany	Prudden.
Potomac	3,774	Washington	Thos. Smith.
Neva	5,772	St. Petersburg	Poehl.
Rhone	75	Geneva	Fol.
Rhine	20,300	Mulheim	Moers.
*Main	2,050	Frankfort	Rosenberg.
*Spree	65,000	Above Berlin	Frank.

Number of Bacteria found each month.—The following table shows the average number of bacteria per c.c. found each month in the reservoir, settling basin, St. Cunegonde and St. Lawrence samples :

Date.	Temperature of water C.	Level of water at Lachine in feet.	Bacteria per c.c.				
			Reservoir.	Settling Basin.	St. Cunegonde.	St. Lawrence.	Combined Average.
December 1st,'90	4°.	11·1	8	313	473	265	284
January 5th, '91.	0°.	12·0	31	44	30	61	41
February 2nd	0°.	10·9	20	89	63	29	50
March 5th	0°.	12·2	185	164	316	577	310
April 13th	0°.	13·0	171	347	363	161	260
May 4th	10·9	15·0	79	121	156	324	167
June 2nd	13·0	13·0	42	189	130	210	142
July 2nd	18·3	11·5	30	481	197	81	275
August 3rd	21·0	11·5	92	119	101	85	99
September 7th	18·3	10·1	21	81	53	53	52
October 1st	13·1	10·1	40	55	29	43	42
November 25th	4°.	10·5	143	1132	1883	363	930

The following summary shows the maximum, minimum and average number of bacteria per c.c. for each sample throughout the year, together with the dates upon which the maximum and

minimum numbers occurred, and the total number of samples examined from each source :

Number of samples examined.	Source.	Bacteria per c.c.		
		Max.	Min.	Average.
70	Reservoir	286 (Nov.)	9 (Feb.)	78
67	Settling Basin	1900 (Nov.)	32 (Oct.)	278
73	St. Cunegonde.....	2260 (Nov.)	12 (Oct.)	316
71	St. Lawrence	600 (Nov.)	18 (Oct.)	189
281				

The above tables show that during the greater part of the year the number of bacteria per c.c. of the water varies between 100 and 200. During the early part of the summer and in midwinter this number falls considerably below 100, and during the spring and early fall it rises for a short period to between 1,000 and 2,000. These temporary elevations coincide with a period of heavy rainfall which ushers in the winter, and with the melting of the snow in the spring, on both of which occasions the river level rises considerably.

The interval of one month between the taking of samples is so great, that the temporary rise in the number of bacteria might pass unnoticed, if this sample did not happen to be taken exactly at the time when it occurred. Suspecting that this was the case in 1891, I made private examinations of the tap water at intervals of one week, with the result that a rise to 1940 per c.c. (compared with 347 per c.c. in the official sample taken a few days before) was observed, the number falling to 117 by the time the next official collection became due. The number obtained in the official settling basin being 121.

It is evident that the 12 months covered by the analysis comprises the early winter increases in bacteria for both 1890 and 1891, which makes the average number for the year higher than would otherwise be the case.

This spring contamination of the water was also studied in

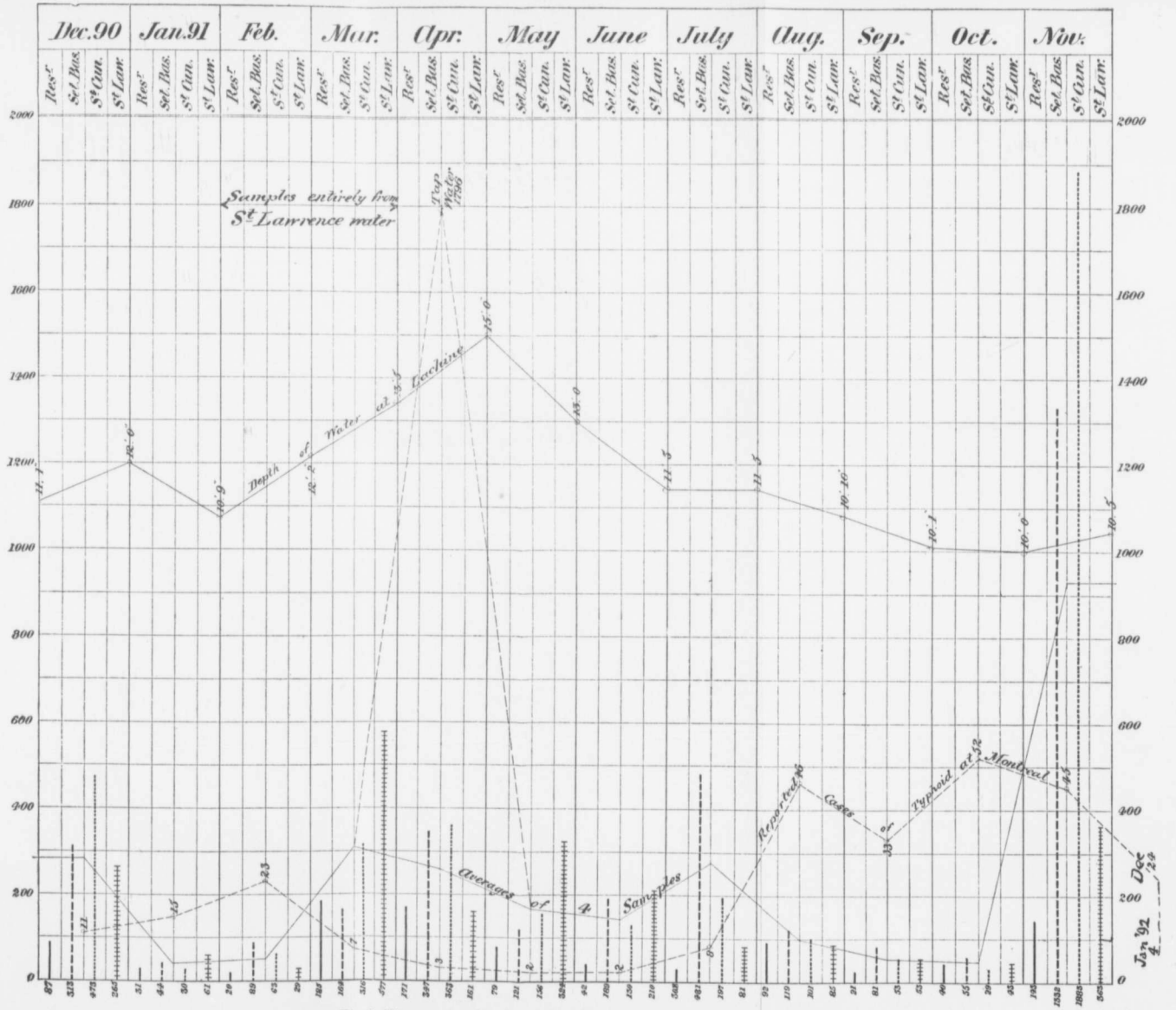

Fig. 6—Diagram showing the results of the monthly examination of water samples.

f water samples.

tap water during April, 1892. The following table shows the variation in the number of bacteria :

Date.	Bacteria per c.c.
April 2	112
9	830
16	2400
26	122
30	46

The two periods characterized by low numbers of bacteria (midwinter and early fall) correspond with seasons when the level is very low.

These relations are shown graphically in Fig. 6.

Although rainfall, when sufficient to produce a marked rise in the water level of the rivers, was found to be associated with an increased number of bacteria, due no doubt to the washings of the soil, no increase was noted corresponding to the ordinary local rainfall.

Comparison of the Four Samples Examined.

Reservoir.— One is struck by the marked superiority of the reservoir water shown by its small number of bacteria, as compared with the other samples. During 9 months of the 12, the number of bacteria was below 100, while the average number was less than one-third of the number found in the settling basin. This, apparently, is due to the beneficial effects of sedimentation, although the reservoir is not well constructed for that process (not having separate inlet and outlet pipes), but chiefly serves to secure a head of water with constant pressure and to form a reserve in case of need. That the reservoir water does not deteriorate, and that its quality remains unimpaired in spite of a large accumulation of mud and slime at the bottom, is a matter which can be readily accounted for. We know now that the agencies which produce the series of oxidative and nitrifying changes, leading to the self purification of waters, are a special class of organisms (nitro-bacteria) which are most abundant in that very slime which is generally regarded with so much suspicion by the public. To secure the proper perform-

ance of this beneficial process, by which the albumenoid and ammoniacal bodies, products of pollution, are (perhaps after being first decomposed into more readily assimilable forms by the agency of the water bacteria) changed into the more stable forms of nitrates, it is necessary that there shall be a sufficient supply of dissolved oxygen in the water and a sufficient circulation to promote oxidation and check any tendency to anærobic putrefaction. For this reason shallow reservoirs of 15 to 30 feet in depth are better than deeper ones.

Sunlight has been supposed to act powerfully in keeping in check any tendency to bacterial overgrowth, but although I have not yet been able to practically test the matter, it seems probable that the opacity of the Montreal water supply in summer would render the effect of sunlight very slight.

That the improvement which reservoir waters undergo during sedimentation is not merely due to a mechanical sinking of the bacteria, is shown by the fact that the number found in the deeper strata does not show any corresponding increase. This was seen in the following observations :

		Depth.		Bacteria per c.c.		
		From surface.	Above bottom.	Max.	Min.	Average.
Lower Reservoir..	(South Basin)..	10	15	66	43	54.3
No. 564.....	Oct. 2, 1891..	24	1	86	55	67.3
Lower Reservoir..	(North Basin)..	10	15	.....	.....	16.0
No. 564a.....	Oct. 2, 1891...	24	1	20	15	17.5
Lower Reservoir..	(South Basin)..	5	20	236	180	203.0
No. 570.....	Nov. 20, 1891.	10	15	246	214	238.0
		20	5	248	96	172.0
		24	1	182	148	165.0
Lake St. John.....	(Roberval).....	5	40	57	9	24.2
No. 555.....	Oct. 7, 1891...	40	5	27	8	17.6

From what we know of nitrification in waters, the ideal bed for a reservoir should be coarse sand or gravel rather than of bare masonary or cement, but as a matter of fact the natural sediment from the water furnishes abundance of the nitrifying agent.

Settling Basin.—This term as applied to the pond at the wheel house is a misnomer, as the current is always so rapid as

to allow of very little settling, and, as a matter of fact, the number of bacteria found there was never noticeably less than that in the aqueduct. From a biological point of view the plan suggested by the Superintendent of having a separate channel for the water used in obtaining power for pumping, and of greatly enlarging the settling basin seems to be an absolute necessity. At present the water is pumped into the mains with very little settling at all, while only a small proportion of it ever passes through the reservoir. I might point out that the question of what should be the proper dimensions of the settling basin is a biological as well as an engineering one, and a series of examinations should be made to find out what amount of surface area would be sufficient to secure, by sedimentation, the requisite reduction in the number of bacteria*.

St. Cunegonde.—The samples from the St. Cunegonde source in the Nuns Island Channel showed about the same number of bacteria as those from the settling basin, and were decidedly inferior in quality to both the reservoir and St. Lawrence water.

Evidently the theory of the supposed superiority of this water arose through a mistaken interpretation of the chemical analyses by the Inland Revenue Department, and simply consists in a lessened amount of organic matter due to larger dilution by the St. Lawrence water. As the organic matter, characteristic of the Ottawa water, has been shown by Dr. Ruttan to be of the nature of a harmless pigment (crenic and apocrenic acids), the most exact proportion in which it may be present is a matter of indifference from a sanitary point of view.

That the mere passage over the rapids in anyway improves the water by oxidation has never been demonstrated, and as we now know that the oxidation of water is not simply a matter of aceration, but is due to the action of the nitrifying bacteria, there is no longer this theoretical argument in favour of this point of supply.

On the other hand, a special investigation, made jointly by

* The question of the undesirable proximity of the garbage depot to the settling basin had not arisen at the time when this analysis was made, and I have since had no opportunity of investigating the matter.

Dr. Ruttan and myself in July, 1891, brought to light facts which show that the intake of the St. Cunegonde supply is not very favourably situated.

The discharge from the tailrace, which empties into the Nuns Island channel 150 yards above the St. Cunegonde intake, brings with it the contents of the river St. Pierre. This little stream receives the drainage of all the land lying to the north of the canal between Montreal and Lachine, with the result that its water half a mile west of Cote St. Paul was found to contain over 13,000 bacteria per cc. A little further on it receives the washings of the West End Abattoir. This addition gives the water a very offensive character, and I found it to contain 172,000 bacteria per cc. In examining the tailrace water upon several occasions I never failed to detect floating portions of offal and animal debris. After receiving the tailrace water this number was reduced to 92,500 per cc. owing to the dilution.*

As the discharge of a large volume of this filthy water at a point 450 feet above the St. Cunegonde intake which is situated, 900 feet from the shore, was so obvious an objection, I made, jointly with Dr. Ruttan, an examination of samples obtained on July 7th, 1891, at 5 points in the line between the shore and the intake in order to see how far out the zone of pollution extended. The wind was off shore and its velocity 15 miles per hour. The water level was fairly high in the channel. The water close inshore opposite the intake contained 69,000 bacteria per cc.; at 100 feet out it contained 669 per cc.; at 200 feet out it contained 238 per cc.; and at 400 feet 157 per cc. The number obtained from a sample of tap water at the pumping station was 127 per cc. which one would expect in pure water.

The chemical results obtained by Dr. Ruttan showed marked pollution inshore and at 100 feet, with slight pollutions at 200 feet and none at 400 feet, thus corresponding closely with the biological result.

It is evident that on that occasion the zone of pollution

* This contamination of the tailrace has no bearing upon the Montreal supply as the water only becomes polluted after leaving the settling basin.

ceased between 200 and 400 feet from the shore or 500 and 700 feet from the intake, and it is unlikely that under ordinary conditions the contents of the tailrace enter the St. Cunegonde supply. Still, as under altered conditions of the current, water level or bed of the river it is not impossible that this may occasionally happen, especially when the shallow flats lying inshore are packed with ice.

It would seem safer to divert the drainage of the St. Pierre into the city sewers, though I never found any evidence of such pollution in the samples examined.

I was not able to detect any evidence of pollution from the tanneries either in the water or the ice of this locality, but the probability that the Verdun shore may soon become densely populated is a further objection to the site.

An interesting point in the analysis was the increase in bacteria, was almost entirely caused by a species apparently identical with the colon bacillus. Corresponding with this increase there was a falling off in the proportion of the *Bacillus fluorescens liquefaciens*, which formed from 30 to 40 per cent. of all the colonies in the pure water of the river and only 0.5 to 1.0 per cent. of those in the polluted water of the tailrace. At 100 feet out the proportion of *B. fluorescens liq.*, rose to 12 per cent. at 200 feet to 25 per cent. and at 400 feet to 33 per cent. It would seem that any unusual deficiency of the proportion of this organism to the total colonies during summer should be regarded with great suspicion.

St. Lawrence Water.—The results of the examinations do not show that this water is better from a sanitary point of view than the present city supply, as far as can be judged from the number of bacteria and the nature of the sediment. Although informed that the line of the pure St. Lawrence water would always be met with at a point 800 feet south of Nun's Island, I have on two occasions seen the Ottawa water extend as far as 1500 feet south of the island. Of the St. Lawrence water it can safely be said that it is a perfectly clean and pure river water. One point in favor of the St. Lawrence is that it is far less

affected than the present city supply by temporary pollution due to heavy rainfall or melting snow.

II.—Examination of Local Conditions Affecting the Montreal Water Supply.

Tap Water.—In order to determine whether the water as supplied by taps was similar in quality to that of the mains, numerous samples were examined during July and August of 1891. The taps were in all cases allowed to run for at least 30 minutes before samples were taken and two or more samples were always examined, in order to make sure that the number obtained was typical for the day. Besides taking samples each day from one special tap which was allowed to run continuously, I made frequent examinations from taps in various parts of the city.

The tap water was found to contain practically the same number of bacteria as the water of the settling basin and, as a rule more than that of the reservoir. The number of bacteria was found as a rule remarkably constant, irrespective of the points from which the samples were obtained. Usually, but not always, the taps on the circuit supplied by the upper reservoir (the water from which is pumped up from the lower reservoir) contained fewer bacteria than those in the lower circuit. I have given the results in the following table.

Comparison of Upper and Lower Circuit

Date.	Number of Bacteria per cc.	
	Lower Circuit.	Upper Circuit.
1891.		
May 1		
" 8	306	117
" 14	210	66
" 22	146	105
June 23	50	48
" 30	30	22

As far as it goes this supports the view that the water is improved by standing in the reservoir.

During July the daily examination showed for the upper cir-

cuit a maximum number of 136 bacteria per cc. and a minimum of 28, the average being 68. During August the maximum was 160 per cc. the minimum 17 and the average 55.

A comparison was made of the water from the lower and upper circuits with the following results.

	Lower Circuit.		Upper Circuit.	
	Reservoir.	Taps.	Reservoir.	Taps.
Sept. 23	39	37	41	50
Oct. 2	53	49	29	54

Although this shows relatively slightly more bacteria in the upper than the lower circuits, the difference is not large enough to be outside the limits of experimental error.

Aqueduct.—Two examinations of samples taken at 5 points along the aqueduct gave :

	Aug. 7.	Sept. 12.
Maximum	173	102
Minimum	93	38
Settling Basin	224	113
Lachine Intake	115	80

The variation is not sufficient to show any material change in the water during its passage from Lachine.

Dead Ends.—In districts where the circulation in the mains is not complete complaints are often made of turbidity of the water. This turbidity appears to be due to rust from the mains but as the consumers are inclined to consider this condition as unwholesome, I made on Aug. 24th, 1891, an examination of the water from 11 different districts supplied from dead ends. The average number of bacteria found per cc. was 94, and therefore such as to exclude any idea of a polluted or stagnant state of the water. The vital statistics from the streets supplied by dead ends do not show any greater frequency of typhoid than other parts of the city. Iron rust is, as we know used as a means of precipitant for freeing water of organic matter.

III.—Study of the River Water at Points Above Montreal.

Ottawa Water.—In order to study the influence of the towns along the course of the river upon the character of the water, two sets of examinations were made in 1891, one on July 3rd, and the other on Sept. 24th. Samples were collected from the bow of a steamboat by means of a fishing rod and line to which small weighted bottles were attached, and the cultures made immediately. Duplicate samples were taken at 15 points on each trip, and a sample was also obtained from lake Des Chenes, 10 miles above Ottawa. Owing to an accident, several of the cultures made during the first trip could not be made use of. The results obtained are given in the following table together with the distances below Ottawa.

	Distances below Ottawa.	Bacteria per cc.
Above Ottawa (C.P.R. Bridge).....	0 Miles.	170
Gatineau.....	5 "	686
Cumberland.....	30 "	1530
Grenville.....	65 "	48
Carillon.....	80 "	60
*Como.....	90 "	72
*St. Anne.....	100 "	11
Lynch's Id.....	120 "	49

*In lake of Two Mountains.

These are shown graphically in Fig. 6.

This showed a marked increase in the number of bacteria below the city of Ottawa, diminishing to the normal for river water by Grenville and reaching a minimum in the lake of Two Mountains, and increasing slightly in the river channel below St. Anne. None of the smaller towns appeared to have any perceptible pollutory effect on the water.

In the second test on Sept. 24th and 25th, a much more thorough examination was obtained, but the results corresponded to a remarkable extent with those of the former examination. I have given the table in full in order to show the measure in which samples taken from the same points on two succeed-

ing days resembled one another in regard to the number of bacteria:

Locality.	Up Trip.			Down Trip.			Combined Average of both trips.
	Max.	Min.	Aver.	Max.	Min.	Aver.	
Lake Des Chenes				6	4	5·2	
Mil's below Ott'a.							
2	580	378	479	365	250	307	393
5	528	500	509	732	329	532	520
10	460	272	377				
15	155	123	140				
20 Cumberland	172	130	147	176	139	152	149
30 Thurso	314	131	257	172	72	122	204
50 Montebello	47	46	46	45	24	34	40
60 L'Orignal	40	26	33	45	24	34	33
65 Grenville	26	19	23				
80 Carillon	37	31	36	38	26	32	34
90 Como (Lake of Two Mts.)	18	10	14	28	16	22	17
92 Oka			17	8	5	6	12
100 St Anne	10	6	8	16	0	8	8
105 Lynch's Id.	21	15	18	22	14	18	18
120 Lachine	18	8	12				

This is shown graphically in Figs. 7 and 8.

A point to which Dr. Ruttan was the first to call attention is that the thickly settled agricultural district composed by the counties of Pembroke and Russell, having a population of about 100,000, drains into the Ottawa. An examination of water of one of the large streams for this district, the South Nation river, was made by us in May 1892, but no evidences of pollution were detected.

From this it is evident that any pollution due to the Ottawa or other sewage is effectually got rid of long before it reaches Montreal. The greatest improvement apparently takes place in the Lake of Two Mountains the bacteria being much fewer at the lower than the upper end. Attention may also be called to the fact that the number of bacteria in the water of the Lake of Two Mountain is lower than that of the present Montreal supply which on Sept. 23rd, gave 30 to 49 per cc.

That St. Anne, and upper Lachine with the intervening population along the banks of the St. Lawrence do not form a

possible source of infection for the Montreal water supply is by no means clear as our water is taken from the portion which flows by the north bank of the St. Lawrence. It seems advisable that a careful sanitary inspection of this district should be made

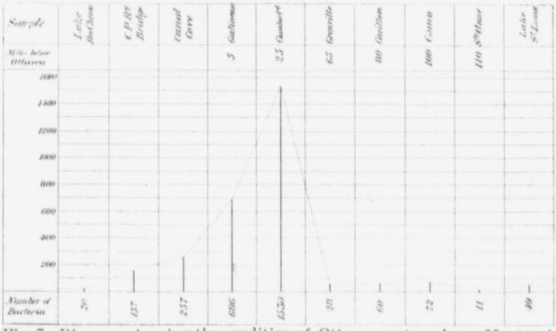

Fig. 7—Diagram showing the condition of Ottawa water above Montreal. (First examination, July 30th, 1891.)

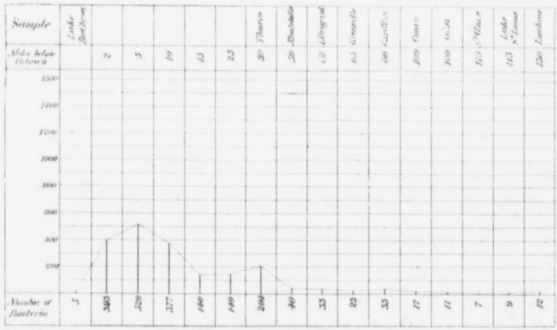

Fig. 8—Diagram showing the condition of Ottawa water above Montreal. (Second examination, Sept. 24th, 1891.)

and a map prepared showing the position of all privies, barns, etc., in order that any possible source of infection should be eliminated. The key to the safety of the Montreal drinking supply may be said to lie between St. Anne and the intake.

The entrance to the intake is confidingly placed so as to catch all washings from the adjacent portions of the lower Lachine road.

Question of typhoid infection.—The following inquiry into the possibility of water-borne typhoid in connection with the Montreal water may be of interest:

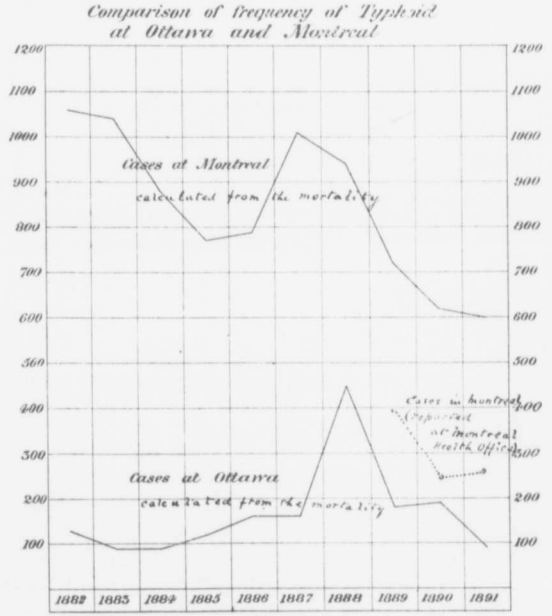

Fig. 9.

The number of cases reported each month at the health office are shown in figure 6. It is evident that if the frequency of typhoid fever depended upon general contamination of the water supply, it would, allowing for the period of incubation, be expected to appear in the month following the greatest contamination of the water. As no increase in typhoid occurred in these

months, that disease being most prevalent when the number of bacteria in the water reached its lowest point, it is evident that the turbidity and increase in bacteria which periodically affects the Montreal supply is not of such a nature as to cause or predispose to typhoid infection. In this connection it was interesting to see if there was any relation between the frequency of typhoid at Ottawa and at Montreal. Unfortunately non-fatal cases of typhoid are not reported to the Ottawa health office, and it is a well-known fact that less than half of the cases are reported at the Montreal office. As the deaths from typhoid are reported however, I have taken these as my basis, calculating the mortality at 10 per cent. As shown by figure 9 there is not only no constant relation between the frequency of typhoid in the two cities, but that even the severe epidemic of typhoid at Ottawa in 1888, was not accompanied by any increase in the number of cases in Montreal.

It appears therefore that general pollution of the Montreal water as may occur is probably of a harmless nature and does not form a source of infection.

St. Lawrence above Montreal.—A double series of observations was made in the same manner as in the case of Ottawa, the samples being taken on July 27th, 1891, between Brockville and Lachine, and on Sept. 30th, 1891, between Kingston and Lachine.

The results with the distances above the Montreal intake at which the samples were taken, are shown in the following table and in figures 10 and 11:

Sample from	Distance above intake.	Bacteria per cc.					
		July 27th			Sept. 30th		
		Max.	Min.	Average.	Max.	Min.	Average.
Lake Ontario near Kingston	190 miles.	.....	.....	.....	29	16	22
Long Point	180 "	.....	.....	.....	51	48	49
Clayton	175 "	.....	.....	.....	33	25	29
Brockville	125 "	76	14	44	.....	.....	.....
Galop Rapid	98 "	38	31	37	.....	.....	18
Head of Long Sault	75 "	210	70	121	.....	.....	76
Foot of Long Sault	68 "	156	141	151	.....	.....	.....
Cornwall	65 "	155	90	130	72	56	63
Coteau	35 "	77	26	47	15	10	12
Caughnawaga	2 "	74	49	61	33	20	27

This examination showed an interesting increase in the number of bacteria on both occasions in the swift and relatively shallow stretch of river below Prescott, the number falling again in Lake St. Francis and rising somewhat below Lake St. Louis.

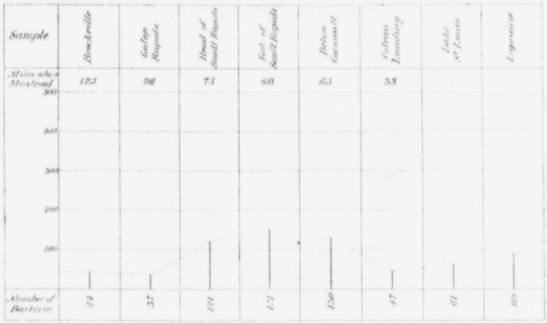

Fig. 10—Diagram showing condition of St. Lawrence water above Montreal. (First examination, July 26th, 1889.

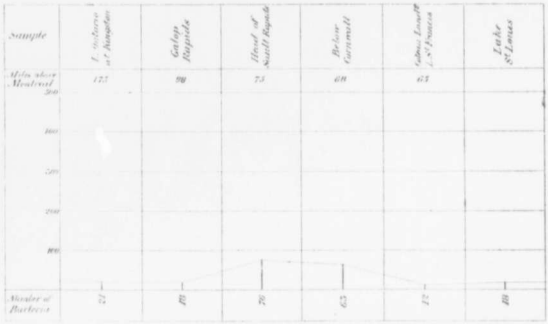

Fig. 11—Diagram showing condition of St. Lawrence water above Montreal. (Second examination, October 1st, 1891.)

It also shows throughout a relatively smaller number of bacteria in the water in September than in July.

IV. Surface Waters in other Parts of Canada.

Water from Uninhabited Districts.—A number of examinations made for the purpose of comparing the Ottawa water with

similar peaty waters for uninhabited districts, may be briefly recorded here by means of the following table, which shows that the water of the large rivers of the far north, coming from a desolate and almost unexplored country, contain as many bacteria as the Montreal water supply. It must be mentioned however that some of the samples were taken during a period of heavy rainfall late in the autumn.

Date.	Sample.	Bacteria per cc.			
		Max.	Min.	Average.	Temperature of water C.
1891.					
Aug. 30.	Saguenay above Chicoutimi	70	41	56	18°
Oct. 7.	Ouiatchouan	134	101	118	12°
"	Ashuap-Mouchuan	700	400	476	10°
"	Mistassini	694	400	474	10°

Other Canadian Water Supplies.—Finally it seems of some interest (in view of the scanty data available on the subject) to mention some analysis of other Canadian water supplies which I made during the summer of 1891, though the fact that these waters were not repeatedly examined makes it impossible to draw any definite conclusion as to their relative sanitary value. In each case several different samples were taken and the cultures were, in every case made upon the spot.

Locality.	Date	Number of samples.	Bacteria per cc.		
			Max.	Min.	Average.
Kingston, Ont.	Sept. 30th, 1891.	8	99	48	65
Quebec, Q.	Oct. 7th, "	5	112	86	90
Sherbrooke, Q.	July "	7	263	85	212
Halifax, N.S.	July 17th, 1892.	7	218	41	99

I mention these results partly in order to emphasize the fact that for a reliable analysis the water must be repeatedly examined and samples obtained at different seasons. In a recently published biological analysis of 21 Canadian water supplies,* made in the spring of 1894 very different results were obtained,

*E. B. Shuttleworth. Toronto Telegram, May 10th. 94.

he number of bacteria found in the Quebec water, for example, being stated as 1045 per cc. whereas it only contained 90 per c.c. on the occasion when I examined it.

Description of Species of Bacteria Found—Qualitative Bacterial Analysis.

From a sanitary point of view the most pressing questions in connection with my analysis of the Montreal water supply were those bearing upon the possibility of pollution. This can be determined as a rule better by quantitative than qualitative work, and I found that the study of the problems of this nature took up so much time that very little was left for the determining of the species of bacteria present.

Although I isolated over fifty different forms from the water, I was not able in all cases to study them thoroughly enough to identify them with existing species described by others. This identification is a matter of extreme difficulty except in the case of well known and easily recognized forms, and the difficulty is increased rather than diminished by the fact that some workers have published as new species forms which were already described, or described them in so vague and unsatisfactory a manner that it is impossible, from the meagre details given, to tell whether they are new or not. Microphotography does not seem to have greatly helped matters, and with the pigment producing (chromogenic) bacteria, it is impossible to tell from the descriptions given what the shade of colour really is and in how much its tint depends upon the medium employed.

For these reasons, although I detected and described several forms which I consider to be new species, I have hesitated to publish them for fear of adding to the existing confusion.

It seems to me that the great tendency which these organisms have to form varieties and races makes it really of less importance except in the case of pathogenic forms to multiply the number of new species by emphasizing minute points of difference than to study their points of resemblance, and so form them into definite groups in which, while the members might differ slightly from one another, their main characteristics

would enable them to be distinguished from the members of other groups. In other words, I would suggest the study of the affinities as well as the differences of the organisms. In that case, even if one might not be quite certain if the organism was a new species, he would know approximately where to place it.

It may, perhaps, not be out of place to quote the following passage from my report made in 1891 (though no doubt the idea has occurred to others) :

"The result of the large number of disjointed efforts made in the direction of systematic description of the water bacteria makes it clear that the matter can never be settled on paper or by the isolated observations of individuals. What is wanted seems to be more co-operation among those working on the subject. This would lead to a sounder basis of classification of the water flora and seems really to be the only feasible means of attaining that end. If a society or committee of those engaged in water analyses in different localities could be formed, and each member allotted one group to investigate, so that various organisms of the same group obtained from different localities could be compared by parallel cultures, the results when compared and published would soon form a recognized standard of comparison. This would not only help beginners, but would obviate to some extent the causes which tend to confuse the work."

Not feeling myself competent to take the lead in a project of this sort, I refrained from taking any steps in the matter, but it may be mentioned that with the co-operation of Professor Adami, of Montreal, an attempt is now being made to organize somewhat upon the lines just laid down a scheme for the co-operative study of the water bacteria.

Bacteria Found in Montreal Water.—The forms which occurred were almost exclusively bacilli, only two species of micrococci being met with.

During the pollution due to heavy rains and melting snow a considerable number of molds, were present. A form of *Fusarium* was once detected in tap water.

The relative proportions of the species present often gave valuable indication of slight degrees of pollution when the total

number of colonies was not sufficient to attract attention * and certain species, notably B. mycoides, appeared when the water was exposed to the washings of cultivated land.

As a rule 5 to 7 forms were detected in each sample when the water was pure, while in impure samples from the Montreal harbour, I have isolated as many as 16 species from the sample. On the other hand when a large number of bacteria developed in stored waters which were pure nearly 90 per cent of the colonies would belong to one species, usually *B. Fluorescens liquefaciens*, and if during the summer the proportion of this organism (which was normally from 30 to 40 per cent of the total colonies) fell to below 12 per cent, other proofs of pollution were usually forthcoming.

A singular circumstance was that in winter this ratio fell to 5 or 10 per cent., although the water was pure, the proportion suddenly rising again when the warm weather returned, while *B. Aquatilis* and other members of the yellow pigment-forming group formed the leading flora during winter. This transition is shown in the following table of analyses :

No.	Sample.	Date.	Temp. of water.	Bacteria per c.c.	B. Fluresc. liq. %	B. Aquatilis. %
128	Tap	May 6, 1891.	6°	646	4	30
129	Tap	May 7	6°	860	8	35
146	Tap	May 14	10°	106	15	20
143	Reservoir	May 14	13°	106	15	20
140	Basin	May 13	11·5	146	12	20
138	St. Cunegonde	May 13	10°	189	15	15
131	St. Lawrence	May 13	9°	245	10	25
155	Tap	June 4	11·5	140	40	2
156	St. Lawrence	June 5	14°	123	30	4

The following were among the common forms met with in Montreal water :

B. arborescens, *B. aquatilis*, *B. flourescens*, *B. fluorescens liquefaciens*, *B. janthenus*, *B. glaucus*, *B. megatherium*,

* See page 21.

B. multipediculus, *B. mycoides*, *B. nacreosus*, *B. aurantiacus* *B. ramosus*, *B. aquatilis sulcatus*, *B. mesentricus vulgatus*, *B. mesentericus fuscus*, *B. proteus*, *B. fulvus*, *B. fuscus*, *B. ochreceus*, *B. plicatus*, *B. implexus*, *B. ruber*.

Among the rare forms may be mentioned B. Berolinensis, of which one single colony was met with.

Spirilla were not detected, but it must be mentioned that the plan of cultivating in weak peptone solutions was not known at the time.

I was able by means of the Parietti and Péré methods to isolate forms apparently belonging to colon group, but never succeeded with Montreal water in finding a perfectly typical distinctive culture of *B. Coli* or *B. Typhi*, whereas I found these present in some spring water at a village where typhoid was epidemic.

CONCLUSIONS.

From the result of this analysis it appears :

1. That the Montreal water is of good quality, as compared with other surface water, and does not appear to be at present a source of danger to public health, though its future purity is not altogether assured.

2. That from a biological point of view the St. Lawrence water is not superior to that of the Ottawa.

3. That the St. Cunegonde site offers no advantages.

4. That the reservoir water is superior to that of the settling basin, and that the Ottawa water is well adapted for storage in open reservoirs.

5. That better facilities for settling water should be provided, and no water pumped into the mains without previous sedimentation.

Inasmuch as the extreme severity of the winter makes the employment of filtration impracticable, it is necessary to watch very carefully any minor sources of possible pollution, especially those lying between the settling basin and St. Anne.

It would be advisable to make experimental studies upon the dimensions and capacity of a suitable settling basin, and also to make arrangements by which the water could be

examined regularly at weekly or fortnightly intervals in order that any variations from the usual standard of purity established by these analyses may be promptly investigated. It would also be well to see if a better quality of water could not be obtained from the Lake of Two Mountains, and to investigate the amount and quality of water available in the lakes in the Laurentian Mountains, lying to the north, in case of a change of supply becoming necessary in the future.

I have to record my thanks to Mr. A. Davis, Superintendent of the Montreal Water Works, for having obtained permission to publish the foregoing report, and also for kindly loaning the cuts which illustrate it.

[Reprinted from THE MEDICAL NEWS, September 28, 1895.]

THE USE OF THE AUTOCLAVE FOR STERILIZING NUTRIENT GELATIN.

BY WYATT JOHNSON, M.D., AND J. E. LABERGE, M.D.,
OF MONTREAL, CAN.

[*From the Laboratory of the Board of Health of the Province of Quebec.*]

AN impression seems to exist in many bacteriologic laboratories that the sterilization of nutrient gelatin by means of the autoclave is impracticable, owing to the tendency of this medium to lose its power of setting firmly when exposed to a temperature above the boiling-point of water. Our experience has shown the contrary to be the case, and the results obtained by a single sterilization in the autoclave have been fully as satisfactory as those obtained by fractional sterilization at 100° C. on successive days, so that we now employ the autoclave by preference to avoid delay and uncertainty. The steam-pressure employed is 3/4 of one atmosphere (equal to 115° C.), saturated steam for fifteen minutes, after the gelatin has been filtered and filled into tubes.

In a series of comparative tests in which half of the nutrient medium was prepared by fractional sterilization in an Arnold sterilizer, that prepared in the autoclave was equally transparent, retained the desired degree of alkalinity (usually 2 per cent. acid to phenolphthalein), and remained firm in plates or roll-tubes at a temperature of from 24° to 25° C. In no case have we had any spontaneous growth in the tubes after this sterilization.

We claim no priority for these observations, as the autoclave at 105° C. seems to have been employed for some years in several French laboratories for sterilizing gelatin,[1] though this fact does not appear to have become as generally known as it might be. In the laboratory of the Board of Health of the Province of Quebec attention was first attracted by the fact that some gelatin, sterilized in the autoclave through a mistake, retained its power of setting. In our experience a temperature of 105° C. has not always been sufficient to insure perfect sterilization.

With reference to the temperature we employ, it may be well to mention that the test-tubes and small bottles which we use are made of very thick glass, so that the heat penetrates slowly. In working with thinner ones a lower pressure or a shorter time may be found to give the best result. We have employed gelatin giving a firm jelly, as considerable difference exists between the different brands in respect to their melting-points.

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Das „Centralblatt für Bakteriologie, Parasitenkunde und Infektionskrankheiten", welches seit dem Jahre 1887 erscheint, und an welchem die hervorragendsten Forscher des In- und Auslandes ihre Mitwirkung bethätigt haben, will den augenblicklichen Stand der theoretischen und praktischen Forschungen auf dem Gesamtgebiete der Bakteriologie und Parasitenkunde,

Fortsetzung auf Seite 3 des Umschlags.

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Ueber den Gebrauch von im Wasser aufgelösten trockenen Blute für die Serumdiagnose des Typhus.

Von

Wyatt Johnston, M. D., Montreal,
Bakteriologist des „Board of Health der Provinz Quebec".

Ein Artikel von Pfuhl in einer der letzten Nummern des „Centralblatts für Bakteriologie (20. Januar 1897) bezeichnet den Gebrauch einer wässerigen Lösung frischen oder trockenen Blutes als eine neue und brauchbare Methode, die Serumprobe beim Typhus anzuwenden. Es werden fünf Fälle erwähnt, bei denen diese Probe erfolgreich von ihm angewendet wurde.

Die Angaben Herrn Professor Pfuhls, die guten Resultate dieser Methode betreffend, kann ich bestätigen, da ich seit September 1896 mich beständig mit derselben beschäftigt habe, in welchem Monat ich als der Erste diese Modifikation der Widal'schen Probe beschrieb. Auf meine Empfehlung hin wurde damals die Methode vom „Board of

Health of the Province of Quebec" als Basis einer kostenlosen öffentlichen Anwendung der Typhusdiagnose eingeführt. Dieser „Board of Health" ist das erste Gesundheitsamt, welches eine öffentliche Diagnose dieser Art für Typhus anwandte. Widal und Sicard hatten schon vor meiner ersten Beobachtung gezeigt, daß Lösungen von zwei Tage lang getrocknetem Blut eine Reaktion ergeben und daher das Prinzip aufgestellt, welches von mir benutzt wurde; praktisch angewandt wurde dieses Prinzip von ihnen nicht. Es ist mir seitdem ebenso wie Widal gelungen, ohne jede Schwierigkeit die Typhusreaktion mit Blutproben zu erzeugen, welche seit 6 Monaten getrocknet waren. In der Zeit zwischen dem 17. September 1896, dem Datum meiner ersten Mitteilung, und dem Erscheinen von Professor Pfuhl's Artikel hat die Methode, Lösungen von getrocknetem Blut anzuwenden, vielseitige Prüfungen bestanden, nämlich von dem „Medical Department of the United States Army und dem United States Marine Hospital Service," dem „Ontario Board of Health", den öffentlichen Gesundheitsämtern von New York, Chicago, St. Louis, Minneapolis, New Orleans, Philadelphia und anderen Städten. Soviel ich erfahren konnte, sind die Erfolge dieser Proben im ganzen zufriedenstel'end ausgefallen, indem man mit trockenem Blute ungefähr dieselben Resultate erzielte, wie mit flüssigem Serum und außerdem fand, daß ersteres leichter zu beschaffen und zu versenden ist, als letzteres. Natürlich gestattet seine Anwendung nicht, den Grad der Verdünnung mit derselben Genauigkeit festzustellen, als dies beim Gebrauch der Serummethode möglich ist, wenn man auch annähernde Resultate mit dem Hämometer erhalten kann.

Ich möchte hinzufügen, daß die Methode bisher noch nicht in der jüngst erschienenen deutschen medizinischen Litteratur erwähnt wurde, und es ist daher ganz verständlich, daß die praktische Anwendung derselben Prof. Pfuhl unbekannt war. Das, was er in betreff der Vorzüge, wässerige Lösungen als Mittel zu benutzen, die roten Blutkörper zu entfernen und so die Reaktion deutlicher zu machen, sagt, kann ich vollkommen bestätigen. Offenbar hat er die Frage ganz unabhängig und in einer von der meinen verschiedenen Weise aufgenommen, indem er als ursprünglichen Zweck die Entfernung der roten Blutkörperchen betrachtete, während ich in meinem Falle mehr Gewicht darauf legte, die Beschaffung und Versendung der Musterproben soviel als möglich zu vereinfachen.

Die Prüfungsmethode, die ich bei einer Zusammenkunft der „American Public Health Association" am 17. Sept. 1896 in Buffalo vorführte (New York Medical Journal. 31. Oct. 1896), war einfach die, daß ich einen getrockneten Blutstropfen in Wasser auflöste, und einen Tropfen der so erhaltenen Lösung mit einem einer Bouillonkultur von Typhusbacillen vermischte. Die durch diese Methode bei mehr als 500 Untersuchungen von mir und Dr. D.D. McTaggart erzielten Resultate waren sehr zufriedenstellende. Diese Ergebnisse erschienen teilweise in dem „British Medical Journal" vom 5. Dez. 1896. Längere Auszüge unserer Arbeiten brachten auch „La Semaine médicale" und „La Presse médicale". In einem am 7. Jan. 1897 vom „Board of Health for the Province of Quebec" herausgegebenen

Rundschreiben und in der Märznummer des „Montreal Medical Journal“ haben wir einige weitere Einzelheiten, die Wirkung von Blutlösungen auf Typhuskulturen betreffend, veröffentlicht, wobei wir feststellten, daß man am besten abgeschwächte und bei Zimmertemperatur erhaltene Stammkulturen benutzte, um die besten Ergebnisse mit Blutlösung zu erhalten. Man gebraucht die aus diesen Stämmen nach einem Wachstum von 24 Stunden bei 37° C bereitete Kultur zur Probe. Dies thut man deswegen, weil Blutlösung eine größere Neigung zeigt als Blutserum bei gesunden Personen, Agglutination mit virulenten Kulturen hervorzubringen. Diese Neigung fällt bei Anwendung abgeschwächter Kulturen fort. Wir halten eine genaue Feststellung des Verdünnungsgrades bei Diagnosen nicht für nötig. Sie stört die Einfachheit der Technik und hat nicht viel zu bedeuten, wofern nicht der Virulenzgrad der Kultur ebenfalls konstant bleibt.

Wir finden, daß abgeschwächte Kulturen dem Typhusblute gegenüber weniger empfindlich sind, als virulente. Wir haben ferner gefunden, daß die körnige Zerstörung, welche bei Pfeiffer's Tierexperiment vorkommt, weit eher bei Blutlösung, als bei Blutserum von derselben Stärke auftritt. So zeigte sich in gewisser Hinsicht die Blutlösung wider Erwarten augenscheinlich wirksamer, als das Serum.

Wir haben die Reaktion auch als Mittel gebraucht, um Typhusbacillen auf mechanischem Wege von unreinen flüssigen Kulturen, welche von Faeces u. s. w. stammten, abzusondern, indem wir die Kulturen durch ein Sieb gehen lassen und die Klümpchen zum Gebrauch zurückbehalten, welche auch mit dem Mikroskop herausgesucht werden können. Um gute Resultate zu erhalten, dürfen die Typhusbacillen nicht gar zu spärlich vorhanden sein. Es wird später über diese Isolierungsversuche ausführlich berichtet werden.

Getrocknetes Blut von Impfcholera ergab gute Cholerareaktionen, wenn wir es mit Wasser befeuchteten (New York Medical Journal. 30. Nov. 1896).

Eine Immersionslinse ist nicht nötig, um die Reaktion zu beobachten. Wir finden, daß man mit auf Papier getrocknetem Blute bessere Resultate erzielt, als mit auf Glas getrocknetem, da das Gerinsel am Papier klebt und die Lösung klar bleibt. Das am besten einzuschlagende Verfahren ist, daß man einen von einer Haarröhre aufgesogenen Wassertropfen eine oder zwei Minuten lang auf der Blutkruste unbeweglich stehen läßt. Eine Oese von der so erhaltenen Lösung wird dann von der Spitze des Tropfens genommen und mit einer Oese der Kultur vermischt. Dr. McTaggart hat gefunden, daß man, wenn die Reaktion sich langsam oder unvollständig entwickelt, dieselbe dadurch beschleunigen kann, daß man das Deckgläschen von dem hohlgeschliffenen Objektträger abhebt und einen weiteren Tropfen von der Blutlösung hinzufügt. Wir benutzen gewöhnlich Lösungen, die soweit verdünnt sind, daß sie dem Tropfen nur einen schwachen Farbenton geben. Wo die Reaktion stark ausgebildet ist, ist sie selbst bei Lösungen von 1:100 noch nachweisbar.

Bei einer Anzahl von Fällen, die klinisch dem Typhus ähneln, aber unter negativen Blutreaktionen verlaufen, haben wir das Auf-

treten einer sehr ausgeprägten Reaktion mit dem Colibacillus bemerkt.

Die Vorzüge, welche der Gebrauch von getrocknetem Blut gewährt, sind sehr groß für einen Distrikt, wie die Provinz Quebec, welche sich über 200 000 englische Quadratmeilen erstreckt und nur teilweise bewohnt ist.

Montreal, 3. März 1897.

sowie der damit in Beziehung stehenden Wissensfächer wiedergeben, sowohl durch Originalaufsätze und durch ein wöchentliches systematisches Verzeichnis der neuesten einschlagenden Litteratur, als auch durch Referate, welche in gedrängter Kürze regelmässig jede Woche eine Uebersicht über die neuesten einschlagenden Publikationen aller Länder zu geben bestimmt sind. Die hohe Bedeutung der oben genannten Fächer für die Wissenschaft und Praxis des Mediziners, Zoologen, Botanikers ist heute allgemein anerkannt.

Weit über die engen Räume des Laboratoriums hinaus, in denen sie entstanden und herangewachsen ist, hat die bakteriologische Forschung einen stetig sich erweiternden Wirkungskreis gewonnen, die höchsten Probleme der Medizin, die Verhütung und Heilung der Krankheiten, sind von ihr erfolgreich in Angriff genommen worden. Diese stehen jetzt im Vordergrund des Interesses. *Dementsprechend soll vom Jahre 1896 an neben der Morphologie und Biologie der Bakterien und Parasiten mehr als bisher auch die Epidemiologie und Pathologie der Infektionskrankheiten* in dem Centralblatt Berücksichtigung finden.

Es ist deswegen seit dem Januar 1896 Herr Professor R. Pfeiffer, Vorsteher der wissenschaftlichen Abteilung im Institut für Infektionskrankheiten zu Berlin, in die Redaktion eingetreten.

Um die angedeuteten Ziele zu erreichen, zerfällt der Inhalt des **Centralblatts für Bakteriologie, Parasitenkunde und Infektionskrankheiten** in folgende Abteilungen:

1) **Referate.** Diese bilden einen Hauptteil des Blattes und es ist die Aufgabe derselben, den Inhalt aller diesbezüglichen im In- und Auslande selbständig oder in periodischen Schriften erscheinenden Arbeiten über Bakteriologie und Parasitologie, Infektionskrankheiten des Menschen und über die durch tierische und pflanzliche Feinde verursachten Krankheiten, die gegen dieselben anempfohlenen Vorbeugungs- und Bekämpfungsmittel, sowie über alles, was dazu beitragen kann, unsere Kenntnisse von dem Leben der Pilze und anderer Schmarotzer zu erweitern, in knapper, streng wissenschaftlicher Form wiederzugeben. Objectivität der Darstellung soll möglichst streng gewahrt werden, sachliche Kritik doch nicht ausgeschlossen sein, sofern sie sich von allen Persönlichkeiten freihält. Durch Namensunterschrift der Referenten soll die Gediegenheit der Besprechungen möglichst gesichert werden.

2) **Zusammenfassende Uebersichten.** Diese Uebersichten haben den Zweck, den nicht auf diesen Gebieten selbstthätigen Lesern ein möglichst getreues Bild der historischen Entwickelung unserer gegenwärtigen Kenntnis über bestimmte einschlagende wichtige Fragen, z. B. über die Cholera, Tuberkulose, Milzbrand etc. zu geben; dieselben sollen in längeren, also nicht jährlichen, Zwischenräumen wiederholt werden.

3) **Systematisch geordnete wöchentliche Uebersichten über die neueste bakteriologische und parasitologische Litteratur aller Länder;** dieselben geben ein möglichst vollständiges Bild aller Leistungen der letzten Wochen.

4) **Originalarbeiten.** Das Centralblatt für Bakteriologie, Parasitenkunde und Infektionskrankheiten veröffentlicht, entsprechend seinem Charakter als zusammenfassendes Organ, zugehende nicht zu umfangreiche Mitteilungen event. Abbildungen. Die Beigabe von Tafeln kann in Ausnahmefällen zugestanden werden. Als Originalarbeiten sollen auch Original-Referate über Arbeiten bakteriologischen oder parasitologischen Inhalts veröffentlicht werden, welche in bakteriologischen etc. Instituten gearbeitet wurden, aber anderweitig erscheinen. Es wird das Bestreben der Redaktion sein, solche Originalreferate möglichst gleichzeitig mit dem Erscheinen der betr. Arbeiten zum Abdruck zu bringen und sie erbittet für diesen Zweck die Mitarbeit der Vorstände bakteriologischer Institute.

6) **Berichte und Originalabhandlungen über Impfung und Schutzimpfung, sowie künstliche Infektionskrankheiten.**

7) **Berichte über alle die Entwickelungshemmung und Vernichtung der Bakterien und anderer Parasiten betreffenden Fragen.**

8) **Berichte über die in das Gebiet der Bakteriologie und Parasitologie einschlagenden Vorträge und Verhandlungen auf Naturforscherversammlungen, ärztlichen und sonstigen Kongressen.**

Das „Centralblatt für Bakteriologie, Parasitenkunde und Infektionskrankheiten" erscheint im Umfange von 2—3 Bogen wöchentlich. Jährlich erscheinen zwei Bände im Umfange von mindestens 60 Bogen. Der Preis eines Bandes beträgt 15 Mark.

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Frommannsche Buchdruckerei (Hermann Pohle) in Jena. — 7071

THREE CASES ILLUSTRATING THE VALUE OF THE BACTERIOLOGICAL DIAGNOSIS OF LEPROSY FOR PUBLIC HEALTH PURPOSES.

BY

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AND

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(*Reprinted from the Montreal Medical Journal, January*, 1897.)

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(From the Laboratories of the Board of Health of the Province of Quebec and the Montreal General Hospital.)

The case which we wish to bring before the Society to-night is that of a Chinaman, aged 27, who was brought to the Montreal General Hospital in a dying condition, and died a few hours after admission. The man had been a little over a year in the country and was not known to be suffering from any serious illness.

As some suspicions of violent death arose, a coroner's autopsy was performed.

External examination of the body showed a gangrenous, ulcerated area, 2 inches in diameter, involving the skin over the right elbow, surrounding which the tissues were thickened, firm and nodular Numerous firm nodules were seen on the skin over the face, extremities, back and genitals; over the surface of the glans these were especially well marked.

The nodules on excision showed no signs of necrosis or suppuration and appeared to be chronic. The cellular tissue of the right upper arm was œdematous, with ecchymosis in the course of the lymphatics, and during the autopsy numerous ecchymosed areas, varying from 2 to 6 inches in extent, were found in regions which excluded the idea of their being the result of contusions. The most marked ecchymosis was beneath the right iliac and psoas muscles.

Bacterial examination of these ecchymoses showed a few short, thick bacilli (Proteus) staining by Gram's method; no micrococci.

The nerves showed no changes and in particular no nodules in the nerve sheaths. The lungs were deeply congested, no consolidation, no tubercles, or other nodular deposits. In the left epididymis there were several firm, whitish nodules the size of peas. Cut surface firm and

[1] Read before the Montreal Medico-Chirurgical Society, Nov. 9, 1896.

smooth. The other organs showed nothing of special note. Nutrition was good. The gangrene of the skin over the elbow and the evident condition of septicæmia present appeared to explain death.

The general nodular skin eruption did not correspond with either syphilis, lupus, glanders, variola, nor with any of the forms of skin diseases ordinarily met with. It was only towards the close of the autopsy that the idea of the eruption being due to leprosy occurred to us. A cover slip was then prepared by smearing it with a drop of the juice obtained by scraping one of the nodules. On staining this with carbol fuchsin and decolourizing with sulphuric acid and methylene blue (Gabbett's fluid), innumerable, slender, red rods were seen, many of them arranged in the peculiar clumps designated as lepra cells.

Examination of the nodules in the testicle showed also the presence of numerous lepra bacilla, and sections of the skin showed large numbers in the deeper layers. Cultures from the smaller nodules of the skin on Loeffler's serum and glycerine remained sterile. Inoculation of a small nodule into the anterior chamber of a rabbit's eye, shows at the end of three months a nodular exudate over the anterior surface of the iris. No signs of tuberculosis appeared in the animal, the general condition being good and its weight not diminished.

The examination of cover slips thus established the nature of this case with certainty in a few minutes, whereas without this neither the post-mortem nor the clinical examination by the house physician or the physician in attendance had led to the discovery of its real nature.

The bacteriological method appears to be thus eminently adapted for the examination of any doubtful case of skin disease which may be leprosy, as the bacilli are certain to be found in large numbers in cover slips prepared from the material obtained on scraping the nodules of the skin.

The microscopic resemblance between the leprosy and tubercle bacilli should not form a serious source of error, as in tuberculous lesions of the skin the bacilli are always very scanty, and usually only a few are found in the entire cover slip, while in lepra each microscopic field shows enormous numbers of them.

The lepra bacilli also readily stain by the simple aniline dyes, while tubercle bacilli do not.

The ease and certainty by which the diagnosis of leprosy can be made was also shown in a case which one of us (Johnston) examined for Dr. Shepherd six years ago. This patient, a West Indian mulatto, had a good position as waiter in a Montreal hotel, and to avoid losing

his position, allowed himself to be treated for three years for syphilis without any improvement resulting. He then visited Dr. Shepherd's skin clinic, where a diagnosis was made at once from the appearance of the skin eruption and the presence of anæsthetic areas, this diagnosis being confirmed by the microscopic examination.

Recently a case has come under our notice where a Chinese laundryman in Three Rivers, supposed to be a leper, was kept under observation and isolated while a scraping from one of the skin lesions was forwarded for bacteriological examination to the Laboratory of the Board of Health of the Province of Quebec.

No leprosy bacilli were found. Subsequently our personal examination of the case showed it to be one of psoriasis, and a repetition of the bacterial tests again gave negative results.

While authorities differ as to whether leprosy under certain conditions is actually contagious, there can be no doubt as to the advantages of the bacteriological method of diagnosis for public health purposes, since by this means an obscure case can be made out in the course of a few minutes, and if the suspected person is situated at a distance from any laboratory, the samples scraped from a nodule of the skin may be sent by mail enclosed in an envelope addressed to the bacteriologist. In choosing a nodule from which to take the specimen, it is perhaps better to select one in an early stage, before much scarring has taken place, by pricking or scraping off the top of a nodule, and collecting the fluid which exudes on pressure. When dried on an ivory vaccine point or a glass slide samples may be more readily sent by post.

Manson has recommended rendering the nodule anæmic by clamping the base with forceps before taking the sample, but in our experience this is not necessary, as the blood does not interfere with the examination.

We have reported these cases because in spite of the dread with which leprosy is regarded by the public, this simple means af diagnosis does not seem to have been as frequently made use of as it should be by the sanitary authorities.

ON THE DIFFERENCE BETWEEN SERUM AND BLOOD SOLUTIONS, THE CONDITION OF THE TEST CULTURE AND THE SIGNIFICANCE OF BACTERIUM COLI INFECTION IN RELATION TO TYPHOID DIAGNOSIS.

BY

WYATT JOHNSTON, M.D., AND D. D. McTAGGART, M.D.,

(From the Laboratories of the Board of Health of the Province of Quebec and the Montreal General Hospital.

Reprinted from the Montreal Medical Journal, March, 1897.

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By

Wyatt Johnston, M.D., and D. D. MacTaggart, M.D.

Montreal.

(From the Laboratories of the Board of Health of the Province of Quebec and the Montreal General Hospital.)

We wish to report some details concerning technique which we find necessary in order to insure successful results in Serum Diagnosis by the dried blood method, with which we have now tested over 500 bloods.

We mention the facts only in so far as they have a direct practical bearing on diagnostic work.

Our results already published were as follows :

1. Out of 129 cases, which we had good reason to regard as true typhoid, if we exclude a few cases where the first samples were taken at a very early stage and no re-examinations could be obtained, and also a few cases first examined late in convalescence, we have met with but one apparently genuine case of severe typhoid, which, when re-examined under satisfactory conditions, did not give a decisive reaction by the dry blood method and this one also gave no reaction by the serum method. Occasionally the first appearance of the reaction is delayed beyond the end af the first week.

2. We have never met with a well marked reaction under conditions where there were not strong reasons for believing it to be due to typhoid.

3. In a few cases where the result of the blood examination remained in doubt the mild type of the fever made an accurate clinical diagnosis impossible. In such cases, we believe bacteriological examination to be the most exact method of procedure.

4. We have not yet met with a case of typhoid where a decisive reaction was obtainable by the serum method and not by the dried blood method.

5. We found that pseudo reactions may be avoided by attention to the character of the culture media. We have found that by using an attenuated or quiescent stock culture grown at room tempera-

ture, and transplanted at intervals of about one month, a suitable degree of sensitiveness was obtained. From such stock cultures a 24 hour bouillon at 37°C., with a moderately diluted blood solution, or serum would give prompt and decisive reaction within a few minutes in the case of typhoid patients, while concentrated solutions of non-typhoid blood or serum were found to give no reaction, even at the end of 24 or 48 hours, hence estimation of the amount of dilution is not necessary for ordinary diagnostic work. (See circular Board of Health, Prov. of Quebec, Jan. 7th, 1897.)

The reaction, although specific in degree, is now generally considered to be quantitative, and small amounts of the agglutinative substances are admitted to be present in varying amounts in non-typhoid bloods. The specific substances are, however, a hundredfold more abundant in typhoid blood.

With virulent cultures the presence of agglutinative substances in non-typhoid bloods may lead to pseudo reactions occurring which can usually be excluded by estimating quantitatively the intensity of the reaction. These pseudo reactions we have found to be characterized by a rapid clumping, without the corresponding loss of motion so characteristic of the true reaction. If watched for some hours these clumps tend to break up.

Quantitative estimation is now generally done by diluting the typhoid serum, but may also be done by modifying the virulence of the culture.

The degree of dilution which can be employed with a given blood solution or serum, while still producing a decided reaction, will depend entirely on the activity (virulence) of the culture employed. This factor has been too much left out of the reckoning in much of the work already published, and it probably affords a natural explanation of the widely different results obtained by competent observers.

Cultures which are made active and virulent by frequent (daily) transplantation and growth at body temperature, are much more sensative to the agglutinative substance than cultures which have become quiescent and attenuated by infrequent (monthly) transplantation and growth at room temperature.

This is apparently at variance with Pfeiffer's statement (*Cent f. Bakt* XIX, p. 594), that highly virulent cultures are less influenced by typhoid and cholera sera than less virulent ones. No details are given by Pfeiffer as to the conditions under which his non-virulent cultures were used. Pfeiffer's statements refer to serum and not to blood solution, he pays little attention to the agglutinative and much to the paralytic phenomena of the reaction, and attaches most

importance to certain disintegrative changes produced by his special method of testing *in vivo*. We have stated elsewhere that highly active cultures, if left for a few hours longer than usual between the times of transplantation, rapidly undergo involution changes, and while in this condition are far more liable to show agglutination than was the case with the same cultures tested a few hours earlier. We have found that for class purposes involution forms in cholera are as abundant and striking in a virulent culture left unchanged for three or four days as would be the case with a non-virulent culture, grown at room temperature, if left without transplanting for as many weeks or months. Bouillon cultures, which have stood long without transplanting, show a tendency to spontaneous partial clumping, which is quite absent during the first 24 hours. For this reason we prefer to use 24 hour bouillons, which are free from sediment, for the test.

The peculiar disintegration obtained by Pfeiffer in typhoid cultures placed directly in the peritoneum of a specially immunized animal, do not tend to occur where the serum is tested in vitro by the hanging-drop method. With blood solution, however, this peculiar phenomenon is frequently witnessed. The clumped bacteria, if watched, for an hour or so, may be seen to break up in granules, which gradually become indistinct and vanish whilst under observation until practically no trace remains of the clumps which shortly before studded the entire field of the microscope. The change is more liable to occur in cultures some days old than in young cultures, and more, perhaps, with attenuated than virulent cultures. It does not occur with all samples of typhoid blood, and is not well marked in very dilute blood solutions.

This greater tendency to bacteriolytic action in blood solutions often makes the reactions obtained with them look at first sight less striking and intense than that obtained with serum where the clumps usually remain intact. Apparently, however, the difference indicates that a large amount of the bactericidal substances originally found in the plasma do not permanently remain as constituents of the serum. This not only has an obvious bearing on serum therapeutics, but explains how the action of serum may be modified by mechanical mixture with the fibrin elements of the blood.

Quantitative estimation of the degree of dilution in the case of blood solutions is possible by hæmometry as well as by making direct measurement. With samples of freshly dried blood, sufficiently accurate observations can be made to express the degree of dilution in multiples of 10—($\frac{1}{10}$ $\frac{1}{20}$ $\frac{1}{30}$, etc.)

We have employed a cell having a depth of 0·85 mm. and giving

with a Fleischl's hæmometer a tint reading 100 p.c., with $\frac{1}{10}$ dilution of normal blood. In anæmic cases the dilution will vary with the degree of anæmia, which can readily be determined. Blood dried for some time gradually yields less and less hæmoglobin, owing to the change of this substance into the hæmatin compounds. This change goes on rapidly in air where gas is being burned and slowly in pure air. In any case, the error is in the direction of a less dilution than that shown by the hæmometer. As a matter of experience, we find exact estimation of the dilution, while interesting for scientific purposes is not necessary for the practical purposes of the test if attenuated cultures are used and the establishment of fixed arbitrarytime limits, as recommended by Grünbaum seem only of use in avoiding pseudo results, due to the use of highly virulent cultures.

Grünbaum, being enthusiastic for exact estimation of dilution in all cases, claims (*Lancet*, Sept. 19, 1896), that though most sera will in time produce clumping, that typhoid serum can still be specifically identified by its being the only serum, which, with free dilution in a ratio of 16 to 1, will produce a complete clumping and arrest motion in 30 min. A fixed dilution ratio, with an arbitrary time limit, appears to us quite uncalled for as a routine diagnostic practice, and has no standard value unless a culture of fixed virulence is used.

Since writing the above we find that Grünbaum has now stated on theoretical grounds "that possibly the use of attenuated cultures would enable us to dispense with the dilution" (*Lancet*, Dec. 19, 1896.)

We had anticipated *a priori* that the solution obtained from the dried blood would be less sensitive as a reagent than the fresh liquid serum. We find the blood solution on the contrary to be apparently more potent than the serum, in causing the agglutination though not as to the paralytic effect, and perhaps to give the reaction at a somewhat earlier stage of the disease. This view agrees with the researches of Widal, who found that the agglutinating substance was contained in the globulins and fibrinogen, and that the serum albumin and corpuscles contained none. Thus the blood serum contains only a part of the agglutinative substance. Dr. A. H. Appel of the U. S. Army has also recently made studies and observations showing the greater agglutinative properties of solutions of the whole blood as compared with that of the serum. A decided agglutination can be obtained from weak solutions of the entire blood when none is produced by stronger solutions of the serum. While Widal places the limits of dilution with serum below 1 to 200, R. Stern who employed solutions

of the entire blood in bouillon reports reactions with dilutions of 1 to 2000.[1]

Owing to the greater sensitiveness of blood solutions as compared with typhoid serum, there is a greater tendency to pseudo-reactions if active virulent cultures are used, than is the case in working with serum. This difficulty is, however, completely obviated by employing attenuated cultures for testing. Cultures which exhibit darting movements in hanging drops are too sensitive for the dry blood test. Those cultures having a quiet but rapid gliding motion in hanging drops have given us uniformly good results. If the movements of the culture become sluggish, one or two daily transplantations at body temperature will make it more active and sensitive. One or two cc., of the living bouillon cultures injected into the peritoneum of a guinea pig produce immunity and a marked blood reaction without injuriously affecting its health.

Clean preparations containing very little fibrin can readily be obtained if care is taken not to stir up the film of blood clot and to use plenty of water for dissolving.

We find that the blood dries in a few minutes sufficiently to be enclosed in an ordinary letter.

Our routine method of testing is to place a large drop of water from capilliary pipette, on the film of dried blood and let it stand for a minute or two. A loop full of the solution so obtained is taken *from the top of the drop* and mixed with a loop full of the bouillon culture, or may, if desired, be diluted further.

For the re-examination of cases giving a negative reaction, a somewhat more virulent culture can be used or a quantitative estimation also made by the serum method. We have not succeeded however in obtaining a decided reaction by the serum when the result with the dried blood was inconclusive and now attach equal importance to a negative result by the dried blood test.

Our published observations (*N. Y. Med. Journal*, Oct. 31, 1896, *British Medical Journal*, Dec. 5, 1896), on the dry blood method

[1] We observe that Widal, who was the first to show that dried blood could produce the reactions, and already, in June, 1896, obtained reactions from serum after four months drying, has recently (*Semaine Med.* Jan. 13, 1897), reported that he has been able to obtain successful results by the dried blood method in the earliest stages of the disease and that the blood after six months drying retained the power of producing the reaction. The dried blood also gave him positive reactions late in convalescence in cases where agglutination had become very feeble. We are glad to find our published results on these points agree with those of so high an authority. We have found that with those who have had difficulties with the dried method, these have been due to their having acted upon the erroneous idea that the blood solution was much weaker than the serum whereas, even with attenuated cultures, we have got a reaction readily with it in dilutions as high as 1 to 125.

were made with attenuated cultures, and pseudo reactions were practically never encountered.

Later on, for a few weeks we tried active virulent culture transplanted daily at 37°c., but these gave us with the dried blood solution numerous and very peculiar pseudo-reactions, i.e., reactions not due to existing typhoid. For instance, the blood of one of us (W. J.) when dissolved gave prompt and abundant agglutination with a virulent culture, while we habitually use it as a suitable negative control blood with attenuated cultures. A solution of the blood of the other (D. D. McT.) gave no reaction. (W. J. had typhoid fever 16 years ago; D. D. McT. has never had it). W. J's blood serum gave no pseudo-reaction with the virulent culture.

On resuming the use of the attenuated cultures described above, the pseudo-reactions disappeared. On re-examining, the blood drops which had given them with the virulent cultures, no longer did so when tested with attenuated cultures, although dry blood from genuine cases taken at the same time still reacted typically.

For practical diagnostic work it may be stated that when a blood does not show a decisive reaction in a serious case of fever which has lasted over a week, the fever is almost certainly not typhoid. In very mild febricular cases the result may remain doubtful, unless investigated by an early bacteriological examination of the spleen pulp or stools.

In this connection we may state that we find that Elsner medium containing 25 per cent. gelatine instead of 10 per cent. will remain solid at a temperature about 30 C., and give visible typhoid colonies within 24 hours.

Reaction with the Colon Bacillus.

Very little attention has as yet been paid to the clinical significance of serum reactions with colon bacillus. Courmont and Rodet have stated that typhoid blood serum reacts with colon cultures, while Achard and Chantemesse state that it does not. Widal states that he has studied quantitatively the intensity of reaction of typhoid sera with Coli, but has been unable to draw any important diagnostic conclusions from the results.

Various observers have reported colon reaction as being present occasionally in different chronic and acute diseases. This can readily be understood in the light of our present knowledge of terminal infections. One case which at first strongly resembled typhoid but gave no serum reaction, has been recorded by Vedel who found a marked colon reaction and looked upon it as only colon infection, this opinion being confirmed by the subsequent events. Personally we have found

reactions with the colon bacillus to be rare with typhoid blood or serum (even in cases when perforative peritonitis had occurred) provided the typhoid reaction was well marked. On the other hand we have been struck by the large proportion of positive colon reactions obtained in cases having step-ladder temperature and other symptoms strongly resembling typhoid but without the typhoid serum reaction. We think that under these circumstances the colon reaction may have a real diagnostic importance, and indicates that the colon infection whether occurring alone or as a secondary complication of typhoid may be playing an important part in the production of the patient's condition. The whole question of associated colon infection deserves further study.

The reaction can be tested with ease by placing a duplicate drop of blood solution or serum on the cover slip with the drop to be tested by typhoid culture and mixing it with a drop of colon bacillus culture. Pseudo-reactions can be avoided by using stock cultures kept at room temperature, and transplanted infrequently. Test cultures grown in bouillon from the stock at room temperature for 24 hours are free from scum or sediment, and give reliable results. The conflicting results just mentioned may have been due to pseudo-reactions having been taken seriously.

In our case of apparently genuine typhoid without serum reaction, (on which, by the way the test was first applied during the third week) the blood reacted very decidedly to B. Coli, producing typical clumping. The same held good of four other blood samples referred to us for examination as having a clinical course like typhoid, but with negative serum reaction. A complete colon reaction we have found to be exceptional in ordinary typhoid and its presence would indicate a condition of Coli intoxication sufficient to explain the existence of many symptoms giving to typhoid its ordinary clinical features. Whether this excludes typhoid, is another question. W. H. Park has observed a case of fever with no typhoid serum reaction, where he was able to cultivate the typhoid bacillus by spleen puncture. Later on in the case however a relapse occurred and the reaction appeared. The possibility of a latent typhoid infection overshadowed by toxic phenomena, due to concurrent action of the colon bacillus is quite consistent with the generally accepted opinion that many of the symptoms in typhoid and especially the intestinal ones are due to secondary infection by B. Coli. It follows that in severe cases of typhoid type, with no typhoid reaction, the blood should be tested with a culture of B. Coli and a bacteriological study made by examination of the stools or by spleen puncture.

In a few cases we have met with a partial typhoid reaction only, in mild cases clinically fibricular, where the fever subsided by lysis in within two weeks of the onset. Here, the possible presence of typhoid appeared to indicate the prudence of keeping the patients in bed and avoiding articles of diet which are contra-indicated in typhoid. Our experience has been that fibriculæ, with completely negative blood reaction, get suddenly well after a few days of fever. Here, also, spleen puncture, as in Dr. W. H. Park's case, might enable a decided diagnosis to be made earlier than by the blood test alone. Wesbrook recommends spleen puncture under the circumstances. The possibility of infection by organisms resembling the typhoid bacilli must naturally be borne in mind.

Diabetic blood has been found by Block and by W. H. Park, to give a decided agglutination. We have examined two cases of diabetes which both gave perfectly negative results.

Conclusions.

The difference in reaction observed between typhoid blood solution and blood serum is not simply due to varying intensity, but to an alteration in the relative prominence of the agglutinative, paralytic and disintegrative phenomena which constitute the reaction. The extent of this difference ~~also~~ varies with the virulence of the culture, but the difference probably depends also on the presence of part of the specific substances elsewhere than in the blood serum.

Blood solution has a greater capacity than blood serum for producing the disintegrative (bacteriolytic) changes described by Pfeiffer Descriptions of this phenomena are conspicuously absent from the many recent accounts of the reactions with typhoid serum as observed in hanging drops.

The paralytic effect is relatively more marked with serum than with blood solutions.

Agglutination without stoppage of motion is more readily occasioned in virulent cultures by blood solution than by serum, and does not indicate existing typhoid.

It appears preferable that for the dry blood method only attenuated cultures should be used. These have the advantage of being more easily kept in readiness than virulent cultures, and are less sensitive to changes of temperature. With the serum method virulent cultures give prompt results. Dried blood serum can be readily obtained and transmitted to the laboratory by pushing aside the edge of a blood drop which has clotted for a few minutes but has not dried

and collecting the serum beneath it on the tip of an ivory vaccine point, etc. This does not, however, give a quantitative result.

For ordinary diagnostic purposes, the simplicity of the method as originally described does not require modification, provided attenuated cultures are used.

A drop of the solution obtained from a dried typhoid blood drop, mixed with a drop of the culture, will give the reaction promptly, without any special attention to the degree of dilution. In order, however, to obtain the best results, it is well to dilute freely and especially to avoid having a sticky solution of syrup-like consistency.

In cases where the clinical type strongly resembles typhoid and where the serum does not give the typhoid reaction, a decided reaction with cultures of the colon bacillus may explain the symptoms.

Our results with the dried blood test have been very satisfactory, giving uniformly positive results with genuine and well marked typhoid cases, and not reacting with non-typhoid bloods when attenuated cultures were employed.

Although the use of serum undoubtedly enables the results to be recorded and compared with greater scientific precision, we find that dried blood answers just as well for routine diagnostic work.

The alterations in reaction, induced by very slight modifications of the manner of testing, help to explain differences in the results reported by experienced and careful observers. With the same blood and culture, the amount of dilution possible largely depends on whether plain bouillon, bouillon culture or water is used for diluting. Opinions also vary as to what should be regarded as constituting a reaction. Personally, we do not think that anything less than complete clumping and total arrest of motion obtainable by the dry as well as the moist test in a young attenuated culture, should be regarded as typical.

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ON THE IODINE TEST FOR SEMEN.[1]

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If one were asked to instance a couple of medico-legal tests generally conceded to be sufficiently reliable to be accepted without hesitation as a positive proof of the condition they were presumed to indicate, one would not be unlikely to select as examples the hemin test in the case of blood stains and the recognition of spermatozoa in stains due to semen as fulfilling these requirements, when properly performed by competent persons.

While this is no doubt true, it has long been recognized that the technique at our disposal for the examination of spots supposed to consist of semen leaves very much to be desired.

It is by no means certain that all the stains sworn to from time to time as being seminal have really been such, so much does the testimony concerning these objects depend on the degree to which caution and experience have tempered the personal skill and acuteness of the expert. The identification of spermatozoa with absolute certainty, while easy enough in a fresh stain, becomes increasingly difficult with the lapse of time; and the incidental handling, washing or wearing of the articles of clothing, which commonly require this examination, makes it more and more problematical whether the spermatozoa can be demonstrated entire and intact.

There are numerous extraneous objects which are so like the detached heads and tails of spermatozoa as to mislead even those who are thoroughly experienced in the work. The generally accepted rule is that no body which simply resembles the head or tail of a spermatozoon should be considered as serious proof, and the search must be continued until perfectly formed and entire spermatozoa are recognized. The uncertainty produced by finding substances resembling these heads and tails in the specimens examined, may, however, lead the expert to prolong needlessly the examination of stains which are not seminal at all. Delays from this cause may have serious results in judicial procedure, simply by retarding or preventing the public exoneration of innocent persons wrongly suspected. We have also, on the other hand, the possible miscarriage of justice owing to the experts or juries attaching importance to incomplete proof, where the other circumstances of the case are such as to arouse strong suspicions, and must not lose sight of the consequences of abandoning a search for spermatozoa because they could not be promptly found.

[1] Read before the Society, October 3, 1896.

Thus, in addition to a possibility which cannot be altogether ignored that the bodies affirmed to be spermatozoa were not so in reality, we have the serious drawback that the expert can only feel perfectly certain that the stain is non-seminal after making protracted and laborious observations; these, if omitted, may leave a crime unpunished in the case of a genuine stain not received in good condition.

The difficulties mentioned do not imply by any means that the spermatozoa test is not a good one, but simply that it is not adequate for the task it has had to fulfil of being the sole and only recognized test for semen. What is evidently wanted is some simple, prompt, preliminary test, by which one could decide in a few minutes whether a given stain is likely to repay a search microscopically for spermatozoa, or not. At the same time such a test would evidently be still more useful if it was sufficiently accurate and distinctive to afford good corroborative evidence of the presence of semen, and so to guard against the possibility of the expert being led to regard as spermatozoa some entirely extraneous substances.

A test which appears in great part to fill those conditions has recently been announced by A. Florence, of Lyons, whose monograph entitled "Du Sperme et des Taches de Sperme en Médecine Legale,"[2] contains much besides that is of interest.[3]

Florence recommends the use of a reagent not infrequently employed for testing alkaloids, known as the ter-iodide or tri-iodide, of potassium. This reagent is a solution of iodine and potassium iodide, in proportions which correspond to the formula $K I_3$ but which is not a definite chemical combination. The formula recommended is as follows:

Iodide of potassium C. P. . . .	1.65 grammes
Iodine	2.54 grammes
Distilled water	30 c. c.

Good results can also be obtained by one-half the amount of iodine (1.27 grammes equivalent to $K I_2$), but the iodine must be in excess. The well-known Gram's solution is not suitable for the reaction. The solution keeps perfectly well in glass-stoppered bottles. It should be used cold, as warmth interferes with the reaction.

When a drop of the liquid obtained by moistening a seminal stain is placed alongside a drop of the above solution on a glass slide or watchglass, so that the edges of the drops come in contact, there appear almost immediately large numbers of peculiar, brownish-red, pointed crystals. These are rhomboidal, and resemble so closely in form, size and color the hemin crystals that they could readily be mistaken for them, though a careful examination shows points of difference. This comparison to hemin crystals will convey a better idea of their appearance than could be given by the most elaborate description, and will enable them to be distinguished from the crystals which are met with in seminal fluids. They also form small turnstile groups or crosses, in the manner which characterizes the hemin crystals.

[2] Archives d'Anthropologie Criminelle, January, February and March, 1896.
[3] It has also been published separately by Storck, Lyons.

According to Florence (and my own observations confirm his statement), the crystals are sparingly soluble in cold water and very soluble in warm water, reappearing again on cooling. On exposure to the air, they gradually disappear, but reappear on adding a fresh quantity of the reagent. They are readily soluble in ether, alcohol, acids, fixed alkalies and iodide of potassium; they resist solution in very weak ammonia solutions. For their formation to be typical and abundant considerable dilution is necessary, and I have found the degree of this solution a most important detail in making the test.

Dried stains usually give the reaction in a manner fully as prompt and typical as fresh semen, and I have been using as a demonstration specimen for class use a stain on cotton over twelve months old, obtained from a homicide case. Recently, the crystals have been more difficult to obtain in abundance, the reason being apparently the increased difficulty of obtaining a solution of the semen rather than an impairment of the reaction.

The exact nature of the crystals has not been shown, as far as I am aware; I have regarded them as some special crystalline form of iodine.

The nature of the substance which occasions the reaction has not yet been thoroughly established. Florence claims to have isolated from seminal stains a substance which he terms *viriespermine*, and which he regards as a distinctive body producing this phenomenon. He states that it does not correspond in reactions with the substance isolated and called *spermine* by Poehl. It is said to be very soluble and to resist completely the effects of ammoniacal decomposition.

By using a larger quantity of material, the reaction can be obtained in the test-tube, an abundant red or chocolate-brown deposit of crystals being thrown down. In this way the demonstration of the seminal character of a stain might even be made without the use of a microscope.

A single fibre teased out of a thread in a piece of cotton stained by semen is sufficient to give a profuse crop of crystals under the microscope.

As to the degree to which this reaction is characteristic of semen: Florence claims that it will not react with any of the other secretions of the body, such as blood, urine, sweat, saliva, tears, bile or milk, nor with pus or nasal or vaginal mucus. The secretion of the Cowper's glands does not give it. I have made a number of tests with the various substances mentioned above, and have always obtained negative results, or at all events have never obtained a characteristic reaction.

The sperm of animals is stated by Florence not to give the reaction, as far as his observations went, though these were not extensive. Personally, I have not tested this point with sufficient thoroughness to give an opinion; but as far as my observations go a pseudo-reaction of doubtful nature can be obtained with at least some forms of animal semen. This point is still, as far as I know, unsettled.

In respect to Florence's claim to priority, I think it can hardly be disputed. I can find no recent work on legal medicine in which any chemical test is given for semen. Wood[4] says, "There are no chemical tests by which seminal stains

[4] Vol. ii of Witthaus and Becker's Handbook, 1891, p. 79.

can be recognized." Dixon Mann (1893), Strassman (1895), Vibert (1896, 4th Edition), Hoffman, Taylor, Liman and our other standard authors do not refer to it. Roussin indeed recommended for examining seminal stains the use of solution of iodine and iodide of potassium, but only for the purpose of staining the spermatozoa, and the fluid which he recommended (iodine 1, iodide of potassium 4, water 100) does not give the Florence reaction. Apart from Florence's work there is practically no literature on the subject except the older work of Orfila as to the odors obtained upon heating the stains or treating them with nitric acid.

Florence states that he started with the firm conviction that so unusual a fluid as semen, which had such well-marked physical peculiarities, must contain some characteristic chemical substance. Acting with this hypothesis in view he proceeded ***seriatim*** to test seminal stains with all the ordinary reagents used in obtaining chemical reactions, especially those found of value for recognizing alkaloids and those generally employed in physiological chemistry. By trying these one after the other he discovered several which gave him positive results, and among these he selected the ter-iodide of potassium as the one best adapted for medico-legal requirements.

During the past three months I have been making some observations on cadavers (22 cases) upon the occurrence of this reaction in connection with the secretions from the prostate, seminal vesicles, testicle substance and the post-mortem ejaculations from the meatus, with a view of determining whether the prostatic ingredient of the semen or the semen proper was chiefly concerned in giving the reaction. The material was obtained for the most part by allowing it to dry on cotton-wool swabs, so as to obtain a condition comparable with those under which seminal stains ordinarily come under medico-legal examination.

Pressure of other work has prevented me making these examinations with sufficient thoroughness to make their publication in detail seem advisable until I have gone over the material again more carefully, but the general results are as follows: Drying does not appear to interfere with the reaction materially within the time limits I have mentioned; and, in fact, I have often obtained the reaction more satisfactorily from moistening the dried secretion than from the original fluid. Decomposition, such as is met with in drowned bodies and bodies long exposed to the air, appeared to interfere with it to some extent, contrary to what Florence's observations would lead us to expect. The semen from the meatus or from seminal stains gave a better reaction than that substance obtained from the regions where the prostatic and testicular components of the semen had not yet mingled. Semen expressed from the prostatic duct into the urethra gave prompt and characteristic results, while these were much harder to obtain from the testes or the contents of the vesicles. So much was this the case that at first I thought the reaction might be due to the prostatic element of the secretion and not to the strictly seminal part. In some cases, however, typical results were obtained from the contents of the seminal vesicles and from the substance of the testicles. In two cases the reaction was imper-

fect or almost absent, certainly not sufficiently typical to justify medico-legal deductions, one being a case of cancer of the seminal vesicles and prostate, and the other double chronic vesiculitis with corpora amylacea very abundant in the prostate. In both of these, spermatozoa were present in the semen.

Hypertrophy of the prostate did not appear to affect the reaction. In one case the reaction appeared to be present before the period of puberty (seven and one-half years), though here it did not appear to be perfectly typical and satisfactory. I could not find it in the secretions of infants and very young children.

These few observations are not recorded here so much with a view of passing judgment upon the percentage reliability of the test as to indicate the direction in which observations might be made, as the total number of observations I have been able to make so far is not sufficient to justify general conclusions.[5]

With regard to the practical merits of the test, I think that those who use it for testing seminal stains will agree as to its decided value as a preliminary test, and accord it a position analogous to the guiacum test for the blood, by which we can promptly find out whether there is a probability of positive results being obtained upon further examination. Personally, if it was obtained in a typical manner, I should regard it as very strong corroborative evidence in a case where spermatozoa were claimed to be present.

With regard to its independent value as a test, Florence formulates his conclusions as follows:

(1) From the occurrence of the reaction alone, without discovering even fragments of spermatozoa, he would conclude that it was probably a seminal stain.

(2) From the coincidence of well-formed and typical but detached heads of the spermatozoa, along with the reaction, he would affirm positively the presence of semen.

(3) With *débris* of spermatozoa or even with perfectly-formed heads, but without the reaction, he would not feel certain that he was dealing with a stain produced by human semen.

It will be noticed that Florence attaches to the detached head of spermatozoa more significance than is accorded to them by our standard authorities. It must be mentioned that Florence has added materially to the data which may be used in identifying the heads of human spermatozoa, as the result of his careful study by means of oil immersion lenses and staining methods. By these more refined methods, highly characteristic details were brought out which would escape notice if ordinary dry lenses of moderate magnification were the only ones employed. The illustrations of spermatozoa given in his monograph are far superior to the illustrations which adorn the average medico-legal text-book, which are in many cases little more than caricatures. It must be remembered that the drying and subsequent moistening of the spermatozoa have a tendency to deform them somewhat, so that the appearance

[5] Two instances of advanced and well-marked disease of the seminal receptacles is obviously a very high proportion for only 22 cases. In both these two instances of organic disease the fact of iodine reaction being interfered with while the spermatozoa were still present indicates the greater certainty of the spermatozoon test.

obtained may not always be perfectly uniform. This tendency to deviation would, however, be more likely to cause human semen to be confounded with that of animals, than the reverse. Personally, my experience does not entitle me to express an opinion as to the extent to which detached heads and tails of spermatozoa should be allowed to constitute evidence of semen, but I think it will be a long time before anything less than the entire spermatozoon will be accepted as legal tender by either judges or juries.

I think it can be said with confidence that the iodine reaction of semen, as described by Florence, is a decided step in advance, and that in it we have a new and very promising preliminary and confirmatory test for normal semen, and one which gives relatively good results in the case of stains which from a prolonged drying, react with difficulty to the microscopic test.

The sources of fallacy which attend the iodine test are, however, as yet practically unknown.

To determine what inference may be drawn from the absence of this reaction in a suspected stain, we need much fuller information and experiments on the effects of external conditions, etc., upon substances known to be stained by semen; we also need information as to the extent to which pathological conditions may exert a modifying influence, how the age limit affects the reaction, and on many other points.

Before we can assign accurately the significance of a positive result, we require much further testing and corroboration regarding the behavior of the reagent with substances other than semen, and also with the semen of animals.

After making all due allowance for this we must rejoice at the success of M. Florence in discovering a crystalline reaction for semen. As a rule, a good crystalline is better than a color reaction and we may expect that further researches in this direction will lead to the discovery of new and valuable micro-chemical tests.

ON THE MEDICO-LEGAL APPLICATION OF ENTOMOLOGY.

BY

WYATT JOHNSTON, M.D., AND GEO. VILLENEUVE, M.D.

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ON THE MEDICO-LEGAL APPLICATION OF ENTOMOLOGY.[1]

BY

WYATT JOHNSTON, M.D., and GEO. VILLENEUVE, M.D., Montreal.

The remarkable results of Mégnin's[2] studies on cadaveric fauna have made medico-legal entomology part of the regular stock-in-trade of the medical expert. One may now judge from the animal fauna met with in a dead body how long it has been exposed. The creation of this special department of legal evidence is an honour which belongs to Mégnin alone, although, prior to him, Bergeret[3] had already made some practical applications based on the erroneous views current at the time.

Through the publishing of his Faune des Cadavres, the popularisation of the subject has been greatly furthered, though the subdivision of the stages in the process of destruction into 8 successive stages is perhaps less easy to comprehend than the simpler classification previously adopted with 4 stages.

The result of over 15 years experience at the Paris morgue, during which period M. Mégnin had been entrusted with all the expertises requiring a knowledge of entomology, has been, according to Professor Brouardel[4], entirely satisfactory. Often the conclusions which he had arrived at from the examination of a few debris and insects were borne out in the most striking manner by the subsequent course of the case. Sometimes the mystery was never cleared up. But in no single instance did the results of the inquiry go to show that M. Mégnin's deductions were erroneous. There is something almost uncanny in the way which M. Mégnin could state for instance after examining a few bones and some dust that a murder had been committed during the latter part of February of the year before last —and then be absolutely justified by the dying confession of the suspected party. The chief danger to be feared from Mégnin's imitators

[1] Read before the Canadian Medical Association, Montreal, August, 1896.

[2] P. Mégnin Faune des Cadavres, Paris, 1894, Gazette Hebdom. de Méd. et de Chirurg., July 20, 1883; La Faune des Tombeaux, 1887.

Annales d'Hygiene, 1855, Tome IV., p. 404.

[4] La Mort Subite, 1894, p. 99; L'Infanticide, 1896, p. 141.

is that they might tend to indulge in guesses having no very solid basis and to apply rules to countries and climates where they were inapplicable. We considered that before any safe deductions can be made in the case of Canada a number of comparative observations must be made to show how far the data obtained from French fauna hold good here.

It is very much to be regretted that in addition to giving the generalisations and medico-legal applications made by him, M. Mégnin has not also given us the assistance of his numerous observations which underlie these deductions, that we may know accurately the degree of variation of dates actually met with under definite conditions of experiment.

For convenience we have arranged in tabular form the dates which Mégnin assigns for different fauna to attack the body, as far as these can be determined from the literature above mentioned, though, of course, considerable latitude must continually be allowed for variations, and for this tabulation M. Mégnin is not personably responsible.

The principle is that the products formed at different epochs in the progress of decomposition attract certain forms and repel others.

The typical course of events shown by the table may be summarised as follows: While the body is still fresh it attracts the diptera *Musca*, *Curtonevra* and *Calliphora*. After decomposition has set in, the flesh flies *Lucilia* and *Sarcophaga* are attracted. Later, when fatty acids are formed, the body is invaded by the beetle *Dermestes* and by the moth *Aglossa* (this latter we have never met with in our Canadian observations, but it is apparently very common in France). Later *Pyophila* of the diptera and *Necrobia* of the coleoptra appear, as the condition becomes caseous. After this comes a period of ammoniacal decomposition marked by liquefaction of the tissues into a blackish pulp, during which stage a group of coleoptera, *Necrophorus, Silpha, Hister* and *Saprinus* are met with, as well as the diptera *Ophyra, Thyreophora, Phora* and *Lonchea*. In the next stage the tissues dry up and are invaded by acari, the debris and excrement of which form a powdery deposit. Subsequently, with the progressive drying of the tissues, *Aglossa* (2nd generation) reappears, together with the moth *Tineola* and the coleoptera *Attagenus* and *Anthrenus*. Finally, when nothing but the dried ligments remain on the bones, two forms of coleoptera, *Tenebrio* and *Ptinus*, appear and devour these.

In the case of buried bodies, the fauna are said to be much less varied and to consist mainly of *Phora* and *Ophyra* in the diptera, and *Philonthus* and *Rhizophagus* of the coleoptera, together with any diptera which have gained access to the body before burial.

FAUNA OF DEAD BODIES EXPOSED TO THE AIR[1]—(COMPILED FROM MÉGNIN).

	Physical Condition.	Minimum time.	Forms met with.
First Period.....	Bodies fresh.....	First three months.	(D) *Musca.** *Cyrtoneura.** *Calliphora.**
Second Period...	Decomposition commenced.....		(D) *Lucilia.** *Sarcophaga.**
Third Period.....	Fatty acids.....	3 months to 6 months.	(L) *Dermestes.** *Aglossa.* (D) *Pyophila.** *Anthomyia.* (C) *Necrobia* (*Corynetes*).
Fourth Period...	Caseous products.....		
Fifth Period.....	Ammoniacal fermentations, black liquefaction.....	4 months to 8 months.	(D) *Thyreophora.* *Ophira.** *Lonchea, Phora.* (C) *Necrophorus.* *Silpha.** *Hister.** *Saprinus.**
Sixth Period.....	Dessication.....	6 months to 12 months.	(A) *Uropoda.* *Trachynotus.* *Tyroglyphus.** *Glyciphagus.* *Serator.*
Seventh Period..	" extreme...	1 year to 3 years.	(L) *Aglossa.* *Tinéola.* (C) *Attagenus.* *Anthrenus.*
Eighth Period...	Debris.....	Over 3 years.	(C) *Tenebrio.* *Ptinus.*

FAUNA OF BURIED BODIES.

Before Burial	(D) *Calliphora**, *Cyrtoneura.*
After Burial.....	(D) *Ophira**, *Phora.*
	(C) *Philonthes**, *Rhizophagus.*
(The genera marked * in the table are those met with by ourselves.)	(T) *Acharates, Templetonia.*

[1] (D) Diptera, (C) Coleoptera, (L) Lepidoptera, (A) Acari, (T) Thysanura.

The following list of all the species mentioned by Mégnin has been furtished us by Mr. F. A. Chittendale, of the Washington Bureau of Entomology, and contains corrections of the list as first published in the MONTREAL MEDICAL JOURNAL. Those found by us and not mentioned by Mégnin are placed in square brackets. Those marked **A** are NorthAmerican; marked **E** European, and those marked **C** Cosmopolitan, and common in North America.

DIPTERA.—**C** *Musca domestica, Musca carnaria*—**E** *Sarcophaga carnaria*, **C** *Cyrtoneura stabulans*, **C** *Calliphora vomitoria*, **C** [*C. erythrocephala,*] **C** *Lucilia cæsar*, **E** *Sarcophaga carnaria*, **E** *S. arcensis*, **E** *S. laticrus*, **C** *Piophila casei*, **C** *P. petasionis*, **E** *Anthomyia vicina* (*species unknown*), **E** *Thyreophora cynophila*, **E** *T. furcata*, **E** *T. anthropophaga*, **C** *Ophyra leucostoma*, **E** *O. cadaverina*, = *Pyrellia cadaverina*, **E** *Lonchea nigrimana*, **C** *Phora aterrima.*

COLEOPTERA.—**C** *Dermestes lardarius*, **C** *D. frischii*, **C** *D. undulatus*, **C** *Necrobia* = *Ernatus ruficollis*, **C** *". cæruleus*,—*violaceus*. **C** *Necrophora* (*fossor*), *interruptus*, **C** *N. humator*. **C** (*Silpha littoralis*) = *Asbolus littoralis*, **C** *S. obscura*, **A** [*S. noviboracensis*], **C** *Hister cadaverinus*, **A** *H. fœdatus*, **C** *Saprinus rotundatus*, **A** [*S. assimilis*], **C** *Attagenus pellio*, **C** *Anthrenus museorum*, **C** *Tenebrio molitor*, **C** *T. obscurus*, **C** *Ptinus brunneus*, **C** *Philonthus cheninus*, **C** [*P. politus*], **C** *Rhizophagus parallelocollis*, **C** [*Omosita colon*, **A** *Trox unistriatus*].

LEPIDOPTERA—**E** *Aglossa pinguinalis*, **C** *A. cuprealis*, **C** *Tinéola bisselliella*, **C** *T. pellionella.*

ACARI—**E** *Uropoda nummularia*, **E** *Trachynotus cadaverinus*, **C** *Tyroglyphus spinipes, T. siro*, **C** *T. longior*, **E** *Glyciphagus spinipes*, **E**. *G. cursor*, **E** *Serrator amphibius*, **E** *S. necrophagus.*

THYSANURA.—**E** *Achorutes armatus*, **E** *Templetonia nitida.*

Looking at the table, one is at once struck by the small number of genera represented out of the total diptera, coleoptera, acari and lepidoptera occurring in France, so that in practice their recognition becomes a relatively easy matter.

It must be kept in mind that Mégnin's observations apply to human bodies. One is also struck by the absence of several forms well known to attack the flesh of dead animals, birds or reptiles; either they do not appear in the above list, or only appear at a much later date. The burying beetles, for instance, which attack game left exposed for a few days, does not attack human bodies under several months. Hence observations conflicting with Mégnin's work which rest upon observations on other animals, horseflesh for example, have, to our mind, very little practical value, what is wanted being rather multiplication of observations upon human remains, when all the conditions as to dates and meteorological conditions are accurately known. In this direction we have made some studies, which will be mentioned later, and others are still in progress. The possibility that some of the fauna may be attracted by the clothes and not by the bodies we have not found to be a serious obstacle.

Our observations were begun over two years. As far as we are aware, no American or Canadian observations on this subject have been published, so that we had no direct information as to how far the dates and successions, laid down by Mégnin, might hold true of the climate of Canada. As to the comparative frequency of occurrence of various European, American and cosmopolitan genera and species there is considerably more information, though this is for the most part recorded in transactions and books not generally accessible.

To compare the different climates we have tabulated the mean monthly temperatures of the air and soil for Montreal, Greenwich and Paris (the depth of soil in the case of Paris being slightly different).

The differences in summer temperature are very much less than one might expect, and this may explain the general correspondence of our results with those of Mégnin. The climate of Canada is peculiar in having a long, cold winter, during which the ground is deeply covered with snow, which prevents the frost from penetrating deeply followed by a hot summer. The interval between winter and summer is short. Everything is in full leaf within about a month after the disappearance of the snow, and during the warm weather temperatures from 80° to 95° Fahr. (27° to 32° C.) are not uncommon. Thus the temperature of the surface soil in summer is rather higher at Montreal than at Greenwich, and apparently very near that of Paris, The percentage of bright sunshine between April 1st and September

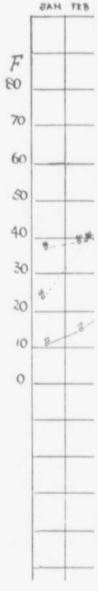


Fig. 1. M
Paris——
N.B.

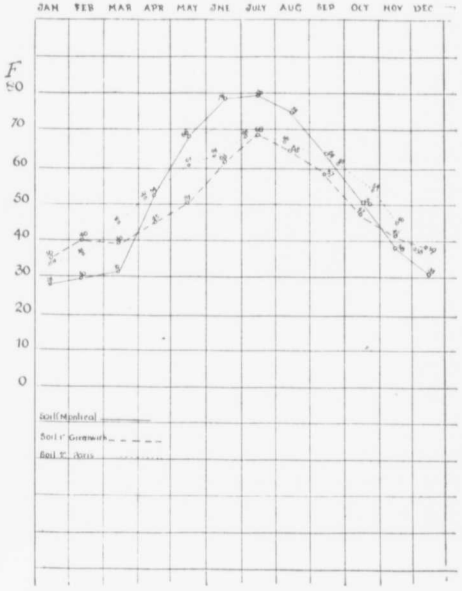


Fig. 3. Monthly means of soil temperature at Montreal 1 in. ——— Paris 2 in. — — — — and Greenwich 1 in. .....

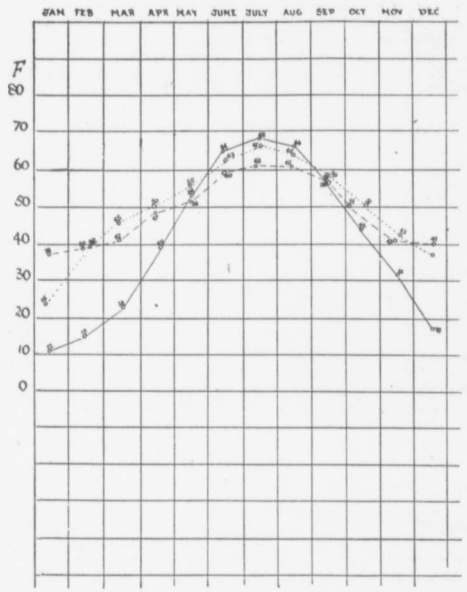
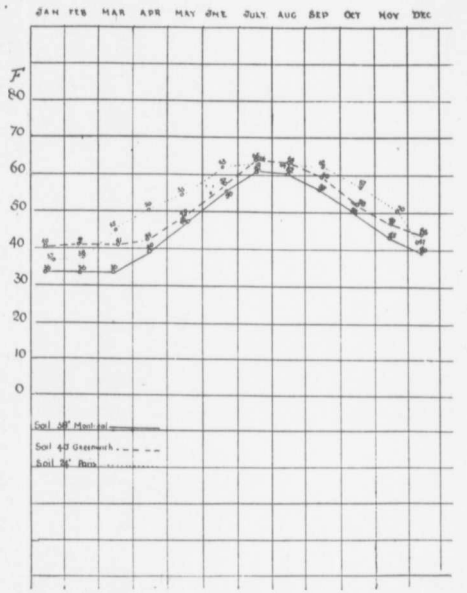


Fig. 1. Monthly means of air temperature at Montreal——
Paris— — — — and Greenwich.....

N.B.—The charts are not drawn quite accurately as to scale.



Fig, 2. Monthly means of soil temperature at Montreal 40 in.—— Paris 24 in.— — — — and Greenwich 38 in.....

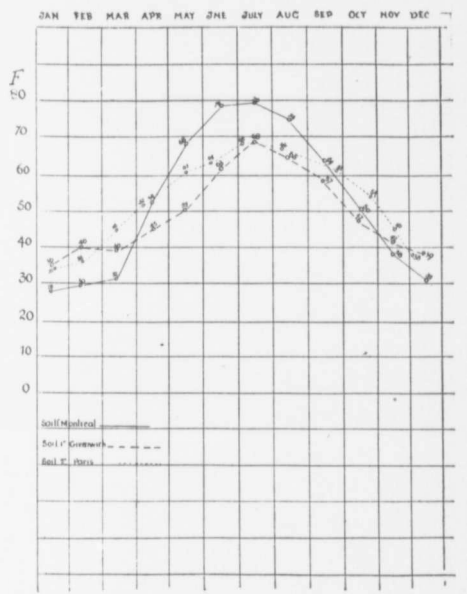


Fig. 3. Monthly means of soil temperature at Montreal 1 in.
—— Paris 2 in.— — — — and Greenwich 1 in.....

30th, a good index of soil heat, is 65 at Montreal and less than 53 at Paris. From the tracings it will be seen that the mean air temperature at Montreal is slightly higher than that at Paris during the summer. The relative humidity in summer is 72 at Montreal and 71 at Paris.

Our fauna were all obtained from the vicinity of Montreal.

	Air Temperature °Fahr. Monthly Means.			* Soil Temperature °Fahr. Monthly Means.					
	Montreal.	Paris.	Greenwich.	Montreal at 40 in.	Paris at 24 in.	Greenwich. at 38 in.	Montreal at 1 in.	Paris at 2 in.	Greenwich at 1 in.
January ...	12	26	38	36	37	40	28	35	36
February ..	15	40	39	36	38	41	30	36	40
March	24	45	42	36	45	41	32	46	39
April	40	50	47	40	50	44	54	52	45
May	54	55	53	48	55	49	68	61	51
June	64	63	60	56	63	57	78	64	63
July	67	67	63	62	64	65	79	68	68
August	66	65	62	62	64	64	74	66	65
September.	58	59	58	57	62	60	64	61	57
October....	46	51	51	52	57	53	50	54	47
November .	33	43	43	45	50	47	38	46	42
December .	18	38	41	40	41	46	31	38	39
Annual Mean...	41·8	52·0	50·0	47·6	52·0	50·3	51·8	51·8	49·5

It was suggested to us that the relatively rapid growth and development of the Canadian flora might be associated with a corresponding rapid evolution of the fauna as compared with those of Europe. This we did not find to be the case with the individual insect forms studied. We did find, however, that the successions of insect forms occurred in a shorter time on exposed bodies than we had expected from Mégnin's statements.

On the other hand we found that the order of the successions followed the rules laid down by Mégnin.

In our comparatively small number of observations, out of the 23

* The soil temperatures for Montreal are from observations by H. L. Callender and C. H. McLeod, Pro. Roy. Soc. Canada, 1895 and 1696. The soil temperatures for Paris are from observation by E. and H. Bequerel, Comptes Rendues, 1883, Tome 96, p. 1109. All temperatures were taken beneath turf. For simplicity they are expressed by the nearest whole number of degrees Fahr. The variations from the monthly means average 1° to 4° Fahr.

genera indicated by him, as characteristic of exposures up to one year, we have met with 11, and 5 of the 12 remaining are rare on this continent. We have only encountered two which he does not mention. These were *Trox* and *Omosita*, both rare in France, although necrophagous.

The diptera characteristic of the first and second periods were found by us regularly on bodies exposed during the warm months, and were not found during the cold weather, unless the body had been previously kept for some time indoors. An exception worth noting was met with in the case of a patient who escaped from an asylum on February 22, 1896, while the fields were covered with snow. The body was found on April 20, 1896, in a snowy spot in a field, and showed about the eyes and nostrils numerous small white larvæ, which on hatching out, proved to be those of *Calliphora Erythrocephala*. It is true that the development and hatching of the pupæ had not taken place. A knowledge of the period of the year at which breeding of the different insect forms takes place is of the utmost importance in determining the dates.

A deduction not mentioned by Mégnin, which we have repeatedly found of practical use, is that if empty dipterous puparia are present, the date of exposure may be reckoned as not less than one month, while the absence of empty puparia indicates an exposure of not over one month in warm weather.

Our observations conform with Mégnin's as to the order of the successions. Thus whenever acari were found, it was evident that *Silpha* and *Hister* had preceded them. *Pyophila* was only seen when saponification of the fat was well marked. *Dermestes* was seen earlier in the saponification period, but never in the preliminary decomposition. *Calliphora* and *Lucilia* were the forms met with up to the end of the first month in cases where the dates were accurately established. Where the exposure lasted a few days only, *Lucilia* was absent.

Our failure to find such forms as *Attegenus, Anthrenus, Tenebrio* and *Ptinus*, taken in connection with the fact that none of the observations reached the time limit at which they were found by Mégnin, is also confirmatory of his statements. In one case where the date of exposure was definitely fixed at five weeks, these forms were absent, although the bones of the skull were laid bare and the cervical vertebrae were stripped of their flesh and disarticulated. The inequality of the destruction in different parts of the body was striking in this case, for the state of the abdominal organs was so entirely free from decomposition that an analysis for arsenic was made, with negative results.

Whenever possible we bred the larvæ in order to determine the time necessary for the complete cycle. This was not found in any case to be lower than that given by Mégnin. It was found to be extremely difficult to obtain more than two generations. Unfortunately the pamphlet of instructions for practical entomological work, published by the Washington Bureau of Entomology was not obtained in time to adopt its valuable suggestions in our earlier observations.

In a case in which one of us (V.) was summoned as an expert, the nature of the fauna present afforded much valuable information. Early in May, 1895, the body of an unknown man was found dead in a lonely spot, with a bullet hole in the skull. There existed an advanced state of adipocere transformation, and in places the bones were partly bare. The body and clothes were swarming with small white larvæ which, from their characteristic skipping action, were thought to be those of *Pyophila casei*, and which on being hatched out subsequently proved to be so. In addition, the body and the clothing were literary covered with large dipterous larvæ and empty pupa cases. These we were not able to identify satisfactorily. No acari or coleoptera were found. The assumption of the police that the man had been murdered *during the winter* in a house near by was disproved by the evidence of abundant diptera, placing the date of exposure back to some time during the warm weather of the preceding summer or autumn. Following this clue, information was obtained which resulted in the body being identified as that of an individual who had been seen in the vicinity during the harvest season of the previous year and who was known to have a revolver in his possession. The subsequent finding of a revolver near where the body lay strengthened the original opinion of the medical examiner that the case was one of suicide.

In another case the body of a new-born male infant, found under some loose planks in the floor of a bath-room and directly over the kitchen ceiling in April, 1895, was sent to one of us (J) for examination with the statement that if the testimony of the witnesses was correct it must have been placed there on a certain night, 5 weeks previous. The supposed mother, a servant in the family had been noticed by strangers to have a suspicious degree of abdominal enlargement, though her mistress stated that she had not noticed this. After the night in question this enlargement suddenly vanished and the girl was noticed to be out of sorts for a few days. Her fellow servant who occupied the same room said that the accused had gone to the bath-room in the night "to change her socks" and had come back covered with blood. Marks of a copious bloody discharge were found

on her bedding and on her under-garments when seized by the police a month later. Unfortunately a proper vaginal examination was not consented to at the time of the inquest, though we learned subsequently that she allowed herself at the request of Coroner McMahon to be examined by a midwife, who claimed to have found a condition indicating recent parturition. There appeared to be no moral doubt that she had been pregnant and confined in reality at the time alleged, the only question being as to the identity of the child.

The body when received for examination was in an advanced state of decomposition and was swarming with *Dermestes lardarius* in both adult and larval stages, as well as large numbers of *Calliphora erythrocephala* larvæ and puparia of which a number were empty and some of the adult flies were found inside the coffin on opening it after it had contained the body for a few hours. The body was found on examination to be in an advanced state of decomposition. It exhaled a strong odour like that of old cheese, and the surface showed extensive pitting from the attacks of the insects. No microscopic or other evidence of acari could be found. The advanced state of decomposition made any decided opinion as to the cause of death impossible. The decomposition of the lungs was relatively far advanced, pointing to the probability of live-birth, but too far to let the question be decided positively. No marks of violence of the severer forms, alone recognisable under the circumstances, existed. The infant was between the 8th month and full term. There were no signs of its having been cared for.

Here we had the anomaly of very positive evidence of witnesses pointing to less than 5 weeks as the time elapsed since death, while on the other hand the state of the body, which according to Mégnin, the only authority on the subject, would require at least 3 months under favourable conditions for its production. The abundance of *Calliphora* did not correspond either with what is met with in early spring and in no other cases of one month's exposure have we ever personally met with the conditions found in this case. On the other hand the position of the body between the floors and over the kitchen was one likely to favour the drying which is so favourable to the *Dermestes*. Some experiments we made with the bodies of new-born infants showed that for the first month the *Dermestes* could not be induced to attack a body, but at the end of the second month they would do so. This latter period was the minimum and was only obtained in a dry atmosphere. The presence of the fatty acids in the vernix caseosa was thought of as a possible factor tending to hasten matters, but under experimental conditions it did not appear to make much difference.

The contradiction between the medical evidence and the theory of the prosecution was pointed out at the preliminary examination, but the case came to trial with the result that the prisoner was acquitted. In this case the circumstances were apparently contradictory of Mégnin's views but the material facts were not established so as to exclude doubt.

In another case where the body of an old woman was found lying in a field in August, 1895, there was extensive decomposition and the fatty tissues showed the adipocere change. The integument, where exposed, was parchmented and the bones of the upper extremity were exposed in places. As far as could be ascertained the body had been exposed since the middle of April making an interval of a little over four months. The insects present in this case were the diptera *Calliphora erythrocephala, Lucilia cæsar* and *Pyophila casei;* with the coleoptera *Silpha noviboracensis, Omosita colon, Hister fœdatus, Trox unistriatus* and *Saprinus assimilis.* In addition there were in parts of the body large numbers of acari not fully determined by the experts to whom they were referred but which belonged to the genus *Tyroglyphus.*

The finding in this case is decidedly what one might expect according to Mégnin as regards the forms present, though they appeared as already stated at an earlier date than he would assign. The presence of two forms not mentioned by him, Trox and Omosita is not evidence against the correctness of his statements as these forms are said to be rare in Europe.

Mégnin's method of computing the time interval by the *number of individuals* found and the proportion of males and females, though one of the earliest means employed by him appears to have a less solid foundation, as the number of individuals first having access cannot be positively known.

In the case of buried bodies our examinations have been confined to a few examinations for medico-legal purposes and are not numerous enough to be of any statistical value. We found that in these cases *Philonthus politus* was invariably present. *Rhizophagus* was not met with. Other forms were extremely scanty except in the cases where the bodies had been kept for a few days in the warm weather before burial and as might be expected showed abundance of diptera, mostly *Calliphora.*

Some very interesting observations made by Dr. Murray Motter on buried bodies in Washington, D.C., which will be published shortly, have been in part privately communicated to us. They show the fauna of buried bodies at Washington to be much more varied and numer-

ous than would be anticipated from Mégnin's statements as to France. The importance of comparing the results in different localities is of course very great, and we venture to think that the soil temperature may prove a better index of what may be anticipated than can be had from the atmospheric conditions of climate and temperature, both as to exposed and buried bodies.

Conclusions.—It appears certain that observations and experiments upon exposed human bodies should be made in the particular locality before the present entomological data can be directly applied to legal medicine, In the vicinity of Montreal it seems probable that the deviations will be in degree rather than in kind, and concern species rather than genera. As a whole, the statements as to the fact of successions occurring and as to the general order of these successions are likely to be strengthened by further observations. Experiments with animal bodies, other than human, are apt to misleading, and adverse results under such circumstances have not very much significance. The time limits, however, apparently require modifications for the particular locality. We know very little at present as to the difference in habit of different species of the same genera.

We have to thank Messrs. Schwartz, Coquilette and Banks, of the Bureau of Entomology, Washington, as well as Professor Fletcher, of Ottawa, for having kindly determined species of diptera and coleoptera for us, Mr. A. F. Winn, of Montreal, for information as to the occurrence of North American species and Prof. C. H. McLeod, for information as to meteorology. Especially we have to thank Dr. M. G. Motter, of Washington, for very valuable assistance, as well as for information as to his personal results with the fauna of buried bodies.

Reprinted for the Author from the British Medical Journal,
December 25th, 1897.

NOTES ON HOUSEHOLD DISINFECTION BY FORMALDEHYDE.

Paper read in the Section of Pathology and Bacteriology, at the Annual Meeting of the British Medical Association held in Montreal, September, 1897.

By Wyatt Johnston, M.D.

Bacteriologist to the Board of Health of the Province of Quebec (in collaboration with D. D. McTaggart, M.D.)

During the past year the use of formaldehyde as a disinfectant has been greatly increased in America, its efficacy having largely become known through the work of Dr. J. J. Kinyoun and Professor F. C. Robinson. It is astonishing how little we actually know of what actually happens in the ordinary routine disinfection of rooms by different methods in comparison with our knowledge of what happens under more rigid experimental conditions. It seemed that devising simple and fairly uniform methods of testing room disinfection which would enable it to be done by the ordinary sanitary officials in the regular course of their duties, important information could be obtained which would be of service in making a choice of methods.

I have designed a little outfit for use by the Board of Health of the Province of Quebec for this purpose, in which by having the infected test objects (small bits of rubber bands dipped in bouillon culture) placed in muslin packets of distinctive colours, the degree of penetration of steam or disinfectant vapours can be tested by an unskilled person (a red packet is exposed near source of disinfectant, a yellow packet is exposed far from source of disinfectant, a green packet is covered lightly or placed in a pocket, a blue packet is covered deeply in a blanket roll or in a mattress). The packets to go in an envelope marked with blank spaces for details as to cubic space, amount of disinfectant used, etc., and can be sent by post. The use of colours and a small metal fastening for the packets also enables any bleaching or tarnishing effects to be detected. These were invariably present with sulphur fumigation, never with formaldehyde. During the past year, a good deal of disinfection of elegant private houses has been done by us. No injury whatever has been reported from formalin. In planning the test outfit, valuable advice was received from Dr. W. H. Park (New York). Practically these test outfits were found to

answer well. Staphylococcus aureus was the test organism chiefly used. Spore cultures were only employed in case of steam. We found that, owing to the high inhibitory powers of even minute traces of formalin, it was best to neutralise the test objects in excess of ammonia before making cultures, or to allow several hours to elapse.

The desiderata in room disinfection we would rank as follows:

1. Efficiency especially as to certainty of result under known conditions. The uncertainty of action with sulphur gas is a great drawback.

2. Freedom from injury to the goods treated.

3. Cost, in which there must be included not only the direct outlay for the disinfection itself, but the cost of apparatus, staff, means of transportation, etc. The methods in general use are disinfection by solutions and fumigation by sulphur or formalin, with steam for articles which require penetration. Preliminary fumigation, so as to disinfect the surfaces of articles to be removed for steam treatment is a safeguard, but causes delay.

What I have to report at present concerns the use of formaldehyde. As is well known, solutions of this substance are powerful and rapid disinfectants, and the vapour has shown considerable powers of penetration. If not so rapid in its action as steam, it is far more certain than sulphur gas, and has no tendency whatever to destroy goods.

In room disinfection I have chiefly employed regenerators, by which the gas is liberated under pressure from a mixture of equal parts of formalin and 20 per cent. calcium chloride solution. The small apparatus made by the Sanitary Construction Company (New York) has been found on the whole most convenient, though I have tried a number of others. My experience with formaldehyde lamps has been less satisfactory. Their effects seemed uncertain, and a quantity of methyl alcohol vapour passes off unconverted as the draft of the lamp increases. The results as to penetration were relatively more satisfactory when the lamps were used in a small enclosed space than would be expected from the results obtained in ordinary rooms.

We found that it was advisable to use larger quantities of formaldehyde than are generally advised, and our results, at first disappointing, became very satisfactory upon using 1 lb. of formaldehyde per 1,000 cubic feet or 1 quart wood alcohol for the same space, and prolonging the actual time of generating the vapour to from one to three hours. We notice that others who, at first, recommended smaller amounts, now advise larger ones. Most of those who report poor results with formaldehyde use small amounts of the agent. Those whose results are surprisingly good with minute quantities of disinfectant we find usually employ culture methods, which do not exclude all inhibitory effects.

The cost for private disinfection is not considerable, at the rate of 25 to 30 cents per lb. per 1,000 cubic feet, and though this would be relatively high in municipal work, it is not at all prohibitory. With these quantities, we succeeded in most instances in securing sterilisation of the exposed objects, and of a large proportion of these lightly covered, that is, placed in pockets or beneath the bed clothes or pillows, but in room fumigation, test objects placed inside blankets or inside matresses were not sterilised. We have to deal in the con-

tents of an ordinary sick room with several different sets of articles. First, surfaces curtains which are relatively accessible to vapours. Secondly, carpets and hangings, which are less accessible. Thirdly, blankets, mattresses, and pillows, which are difficult to penetrate. In order to disinfect articles of the second and third classes in an ordinary room, a great excess of vapour and consequent waste is necessary. By the use of closed chambers, a greater penetration of the vapour can be secured, and a smaller amount is required. The vacuum method of securing penetration does not appear to give the same rapidity of effect that it does in the case of steam. We have been able to secure complete sterilisation in two hours of test objects placed in a closely rolled blanket, by using a vacuum of 15 inches followed by an air pressure of 10 lbs. In this apparatus, however, the steam jacket had to be incidentally heated in obtaining the vacuum, and the action of formaldehyde is known to be increased in proportion to the temperature. In any case, the use of a vacuum does not make penetration a matter of a few minutes as in the case of steam.

Placing the articles in a cupboard or trunk, and blowing in the vapour usually gave fair penetration, if excess of vapour was used. Pasting up minute cracks does not appear to make much difference. Though no large crevices or draughts should be allowed in the room we found it quicker and less troublesome to generate an excess of the vapour than to paste up cracks. I have devoted some attention to constructing a portable chamber or receptacle suitable for room disinfection. One such chamber, 40 inches by 30 inches by 60 inches, made of galvanised sheet iron was light enough to be carried (with some difficulty) on a stretcher by two men, and could be closed so as to give a vacuum of 2 to 3 inches. This was found clumsy in actual practice, and the saving in time did not permit of its being used in two places on the same day. Out of door disinfection is out of question in a Canadian winter. Recently I have tried the plan of using a tent or canopy with those articles requiring most penetration placed beneath it, spread out on convenient articles of furniture. The gas being conducted under this by a rubber tube gave some increase of penetration, while enough escaped from beneath it to sterilise the exposed surfaces in the room.

My latest attempt in this direction has been the construction of bags which are as nearly as possible air tight or gas tight. The cost does not exceed 5 to 8 dollars for one as large as an ordinary disinfectant chamber, 5 ft. by 3 ft. by 4 ft. By using "enamelled duck" and having a projecting flap round the open end so as to be rolled up with a corresponding flap of the cover, the sealing up is fairly good, though capable of improvement in construction. Owing to an unexpected interruption to work, I have not yet had an opportunity of testing this apparatus thoroughly at the time of the meeting, but it appears worthy of trial to see whether the best results are got by blowing in the vapour and by placing in articles which have been sprayed with formalin solutions. Carpets, rugs, mattresses, and pillows can be well sprinkled without being drenched, and placed in the bag, or when the articles cannot stand direct wetting, clothes drenched with the solution can be placed inside. In this way the effects of disinfectant spraying are greatly enhanced, though to what precise extent this may prove useful in room disinfection I am not

at present able to say. From the first, I have been strongly of opinion that methods which permitted of all the articles in the sick room being disinfected without the necessity of removal was very desirable for localities unprovided with larger disinfecting plants and staffs, and that even in cities, economy might be effected by thus doing away with transportation.

With regard to disinfecting solutions, my favourite one is formalin used by means of a pump or spray, as suggested by A. C. Abbott. A ½ to 1 per cent. solution seems sufficient, and the cost of this, though more than than that of 1-1000 sublimate, is only about two cents per gallon. The pump used costs a few shillings, and weighs but a few ounces, but apparently answers just as well as the heavy and expensive Equifex sprays sold for the purpose. The public need clearer ideas of the "effective cost" of different disinfectant solutions—that is to say the cost of a given quantity of solution of effective strength. I think that the adoption of a uniform standard, say, that sufficient to destroy staphylococcus aureus in five minutes would be a convenient strength for house disinfection. The effective cost of several disinfectants is about as follows, when used in amounts generally required for private disinfection:

Carbolic acid, 1-30, costs 7 cents per litre.

Sublimate, 1-1000, costs ¼ cent per litre.

Formaldehyde, 40 per cent. strength, 1-200, costs ½ cent per litre.[1]

Few realise the waste of money involved in the use of carbolic acid. The cheaper grades sold as carbolic acid contain practically no phenol at all. Other things being equal, I think that preference should be given to substances which are not poisonous. I need not here go into the matter of formalin vapour for treating goods liable to be injured by steam or disinfecting solutions further than to say that it is a most satisfactory agent for this work for which we previously had no reliable method. Whether the dust in the walls and cracks are absolutely sterilised or not by fumigation, most sanitarians will agree that the average fumigation suffices to remove the danger of infection from exposed surfaces, as far as can be judged from epidemiological evidence.

NOTE.

[1] Recent reports seem to show that the very conservative estimate of the effective strength assigned to formaldehyde is too low. Thus Le Dentu (*Sem. Méd.*, 1897, p. 315) stated that instead of the 40 per cent. commercial solution being one-fifth as effective as the same weight of sublimate it is really twice as effective in equal quantities. This, if true, would raise the effective strength of formalin and formol to tenfold what is represented in the table.

An Experiment with the Serum Reaction as a Test for Typhoid Infection in Water, etc.

BY

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AN EXPERIMENT WITH THE SERUM REACTION AS A TEST FOR TYPHOID INFECTION IN WATER, ETC.*

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To say that the injection of a suitable quantity of living or dead typhoid bacilli into a suitable animal will produce a typhoid reaction in the blood of the animal is to state a well-known fact. So far as I am aware, however, no one has attempted to utilize this as a means of demonstrating typhoid infection of drinking water, milk, etc.

Wishing to test this point practically, I introduced one cubic centimetre of a typhoid bouillon culture into a flask containing two litres of tap-water from which eleven thousand colonies to the cubic centimetre grew on gelatin at room temperature. After shaking the flask, one cubic centimetre of the water thus infected was introduced into a second flask containing two litres of the same water. From this second flask, which thus represented a dilution

* Read before the Montreal Medico-chirurgical Society, May 4 1897.

of one to four millions of the original bouillon culture, one cubic centimetre was placed in five cubic centimetres of ordinary peptone bouillon and grown at 37° C. for twenty-four hours. The bouillon was then sterilized for one hour at 65° C., and injected into the peritoneal cavity of a rabbit.

The animal's health remained good, except for a slight loss in weight. Its blood, examined after an interval of eight days, gave a perfectly typical reaction when tested with a typhoid culture. The blood had been tested before inoculation with negative results. The blood of a control animal inoculated with five cubic centimetres of a bouillon culture made from the same water without adding typhoid gave no reaction, nor did that of another control animal kept with the others and not inoculated.

It had occurred to me some months previously that by testing in this manner samples of suspected water and milk, typhoid infection might be demonstrated more readily than by making cultures. I tried it in the case of two samples of suspected milk in December, 1896, with negative results, but in both of these the circumstances of the case made typhoid infection seem improbable, and I thought it better to apply the test under more definite conditions.

It will be remembered that Vaughan * inoculated white rats with mixed cultures from water sediments for the purpose of demonstrating in a general way whether infective or toxic substances were present. Now that we have a definite means of recognizing the effects of the typhoid bacillus this method of investigation offers more prospect of being of permanent utility.

* *Transactions of the Society of American Physicians*, 1892.

I am now, with the aid of Dr. D. D. McTaggart, making studies as to the conditions under which a positive result may be looked for. The above experiment is cited only as an illustration of the method, possibly an exceptional one. Whether it will prove of practical use in laboratory work I am not at present able to say.

Concentration of the suspected substance by collecting the bacteria in a porcelain filter naturally renders the test more delicate, as does also the employment of specialized media for the cultures. I have found that rabbits show the reaction at an earlier stage than guinea-pigs, in some cases in two or three days after inoculation. They also have the advantage of being less susceptible than guinea-pigs to septic influences. The preliminary sterilization of the culture is not essential. It lessens to some extent the chances of obtaining a reaction from typhoid infection, but, on the other hand, it permits a larger dose to be given. By averting the danger of concurrent septic infection by other bacteria it increases the animal's chance of surviving long enough to give the reaction time to develop. Small repeated doses we know to be safer than large initial ones. With proper care a typhoid reaction can be induced without the animal's health being seriously impaired.

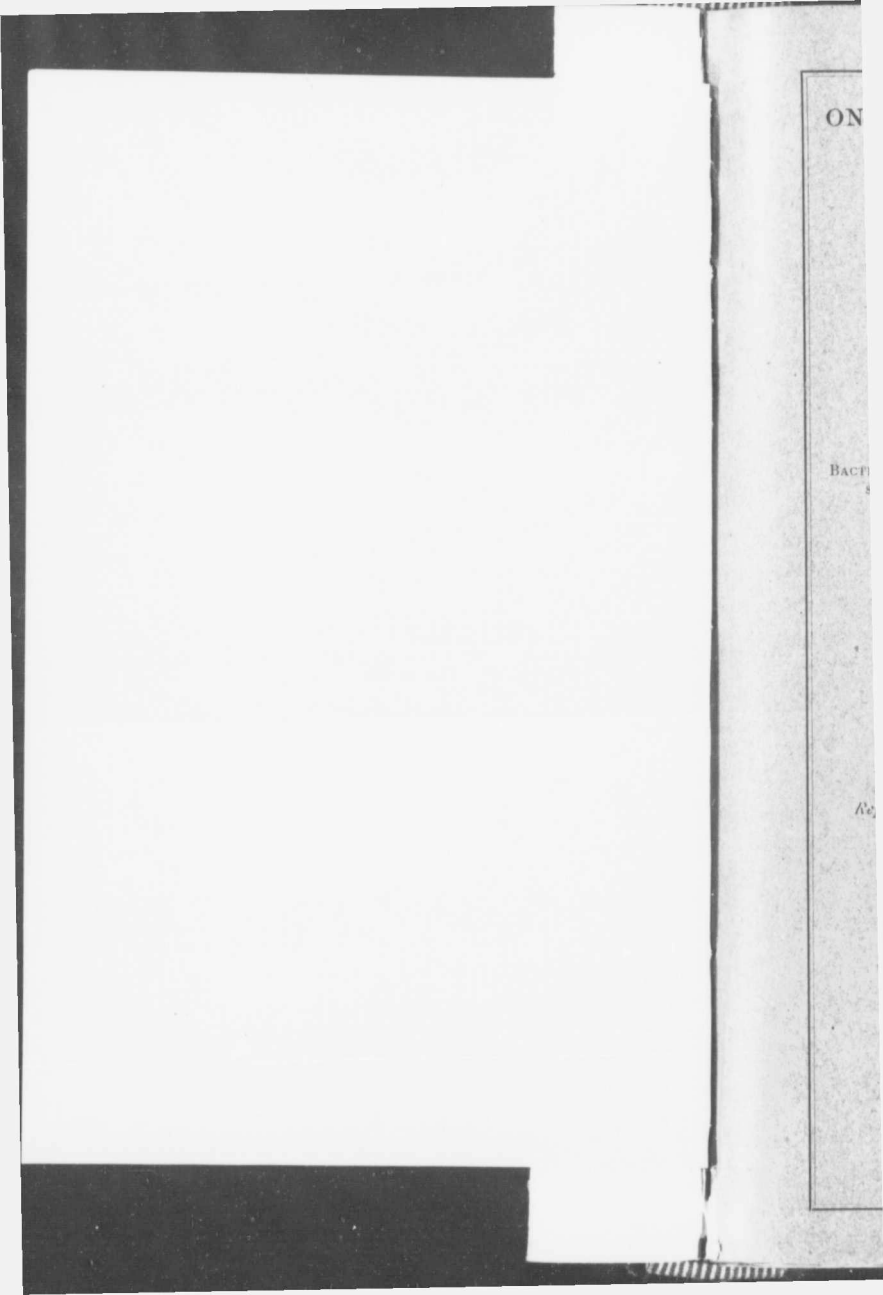
Capacity to produce a blood condition which will react with a genuine typhoid culture is stronger proof of a suspected organism being the genuine typhoid bacillus than capacity of a doubtful culture to react with typhoid blood, as clumping has been shown to occur with other organisms. Hence the production of the blood reaction experimentally with an organism isolated from a suspected water should not be omitted when it is necessary to operate under very rigid conditions of experiment.

The only use to which typhoid serum reaction appears to have been applied so far by others in connection with suspected water is in the testing of organisms isolated by the usual means to see whether they react. I have already published elsewhere * short accounts of some experiments where impure twenty-four hours' bouillon cultures containing typhoid and colon bacilli were treated by adding sufficient typhoid serum to produce clumping, and then in one to two hours, when this was complete, were filtered through an inch of sand, as done in the Sedgwick-Rafter method for the quantitative microscopical analysis of water sediments. It was found that the filtrate yielded almost exclusively red colonies when grown on lactose litmus agar, whereas those obtained from the sediment were nearly all blue ones, showing that the separation of typhoid and coli by this means is rapid and complete. Care must be taken to decant or filter the culture before adding the coagulant (typhoid serum), as there is always some sediment with *Bacillus coli* at the end of the twenty-four hours' incubation. Introducing a thread or cotton filament, on which typhoid blood or serum has been dried, into the culture leads to localized clumping of the typhoid bacilli about and upon it. The paralytic effect of the typhoid serum, however, prevents this method of separation from being entirely satisfactory. I have found that for the mechanical separation to take place the typhoid bacillus must be present in considerable amount, and I have not yet worked out a satisfactory routine method of applying it to the examination of fæces or water.

* *Centralblatt für Bakteriologie*, xxi, and *British Medical Journal*, December 5, 1896 (abstract in *American Medico-surgical Bulletin*, January 10, 1897.

In the phenolized and acid bouillons recommended for typhoid isolation the typhoid clumping, as has been correctly stated by Alpers and Murray,* does not take place, but by neutralizing with soda solution I have been able to obtain it after slight delay. Alpers and Murray are not quite correct in stating that the typhoid serum reaction has only been applied to blood examinations. Elsner, Gruber, and in this country W. L. Russell have used the method in a similar manner to that mentioned by Alpers and Murray for the purpose of identifying suspected organisms isolated by culture from water or fæces. In fact, this was the chief use to which the typhoid serum reaction was applied prior to the announcement of Widal's discovery.

* *American Medico surgical Bulletin*, March 25, 1897.



ON THE APPLICATION OF THE SERUM DIAGNOSIS OF TYPHOID FEVER TO THE REQUIREMENTS OF PUBLIC HEALTH LABORATORIES.

By WYATT JOHNSTON, M. D., Montreal,

Bacteriologist to the Board of Health for the Province of Quebec; Assistant Professor of Hygiene, McGill University; Pathologist to the Montreal General Hospital.

Reprinted from Transactions of the American Public Health Association.

CONCORD, N. H.:
PRINTED BY THE REPUBLICAN PRESS ASSOCIATION.
1897.

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ON THE APPLICATION OF THE SERUM DIAGNOSIS OF TYPHOID FEVER TO THE REQUIREMENTS OF PUBLIC HEALTH LABORATORIES.[1]

Great interest attaches to Widal's important communication to the effect that the serum of persons suffering from typhoid fever, even in the early stages, is capable, when mixed with a pure culture of the typhoid bacillus in bouillon, of arresting the active movement so characteristic of this organism and causing the bacilli to agglutinate into clumps resembling zoogloœa. The serum of typhoid convalescents and immunized animals had been shown by Pfeiffer, Durham, and Gruber to possess this property. But Widal has certainly been the one to demonstrate its great clinical value. With the serum of an undoubted case of typhoid fever we are able to apply what appears to be the most conclusive of the tests at our disposal in deciding whether a given organism is really the typhoid bacillus or not. On the other hand, with a culture of the genuine typhoid bacillus, we are able to decide whether a doubtful case is or is not typhoid fever.

Although the test is so recent in origin, those who have tried it appear practically unanimous as to its being of great delicacy, and, in particular, the negative results which it furnishes are of nearly as much practical value, something which can scarcely be said of the routine bacterial tests for tuberculosis.

Widal's original method was to obtain the serum from the vein of a patient's arm by means of a sterilized syringe, descanting the serum after it had separated and adding it to bouillon culture of typhoid bacilli. This was then placed in the incubator, and showed, after several hours, a flocculent precipitate composed of the immobilized and agglutinated bacilli and a clearing of the upper part of the fluid. This was found by Widal to be characteristic of typhoid blood. The blood in other febrile disorders, such as malaria, typhus, tuberculosis, pyæmia, etc., as well as the serum of healthy persons, was found to have no power of producing this phenomena when mixed with typhoid cultures. Those who have repeated Widal's experiments have also been able to confirm his statements that the colon bacillus does not give this reaction with typhoid blood.

Widal was fortunately led to simplify the method materially by taking

[1] Read before the American Public Health Association, at Buffalo, N. Y., September 17, 1896.

a few drops of blood from the finger tip, and as soon as the serum was separated from the edge of this, mixing it with a drop of actively mobile typhoid culture, whereupon the reaction could be satisfactorily observed under the microscope and was usually complete in a few minutes.[1] Dieulafoy testifies to the remarkable accuracy of the test and its value in diagnosticating obscure cases.

My attention was first directed to the test through having been consulted by physicians as to the nature of suspected cases of typhoid, and my experience has been thoroughly in accord with that of Widal and others as to its great value as an aid to clinical diagnosis.

As the reaction appeared to depend probably upon the presence of some substance analogous to the ordinary toxines, and as many of these preserve their characteristics in a dry state, it naturally occurred to me that this might be true of the substance producing the serum reaction. The advantage of being able to operate with a dried substance was obvious, especially with reference to the possible application of the method to the rapid bacteriological diagnosis of typhoid fever in municipal laboratories, just as is now done in the case of diphtheria, and my observations have been made with this end in view.

Instead of taking the serum as soon as it exuded, I allowed the drop to dry, and found that upon moistening it subsequently the solution obtained was just as efficacious as the pure serum for the diagnostic purposes of the test.[2]

This power appears to remain practically unimpaired even after the blood has been allowed to dry for many days. My experiments upon how long the blood will continue to react when in this dry state are not yet finished, but blood drops dried for from two to four weeks still give the reaction.

In this manner I have tested the blood of ten patients suffering from undoubted and typical attacks of typhoid. The reaction was obtained conclusively in every instance. In eight cases the loss of mobility and the agglutination was complete in from two to fifteen minutes. Of the two others, one, in a very early stage of the disease, required thirty minutes for the completion of the reaction, while the other in a very late stage, following a relapse, required one hour.

The blood of ten other hospital patients, as well as a number of healthy individuals, was next tested, and in no single instance was the reaction obtained. Occasionally a pseudo-reaction with some agglutination was observed within a few minutes of the mixture of blood solution and culture, but some movements of translation (wandering through the field)

[1] This plan of observing the reaction directly under the microscope had been published by Gruber and Durham some months previously.

[2] Since writing the foregoing, I have been able to obtain fuller accounts of Widal's work than were at first available, and find it stated by him that dried serum, and to a lesser extent dried blood, are capable of furnishing the reaction. This circumstance does not appear to have been hitherto utilized practically.

always persisted on the part of isolated bacilli, and these gradually increased in number and activity till, in an hour or two, lively motion was resumed, and was found to be still present on the following day and, in some instances, where it was followed up, at the end of a week. With the typhoid bloods nothing but the oscillating or "Brownian" movements were seen, as a rule, though where the proportion of serum added was very small peculiar revolving and tugging movements, apparently due to the action of the flagella, could be made out, movements from one part to another of the microscopic field being, however, completely abolished.

In two doubtful cases examined for diagnosis the results were negative. In one of these the malaria plasmodium was subsequently detected. The other left the hospital before the diagnosis was cleared up, but her temperature had remained normal for two weeks, and her only symptoms were persistent headache and giddiness. One of the control cases, examined with negative results, had a history of typhoid two years previously.

In making a communication upon this subject before the American Public Health Association, at Buffalo, N. Y., on September 17, 1896, I subjected the method to what I considered to be a fair practical test as to its applicability to public health purposes. I left instructions for Dr. D. D. McTaggart, resident pathologist, to forward by post to my destination, after I had left Montreal, a letter containing dried blood drops from several cases of undoubted typhoid fever and also dried blood drops for control from other hospital cases, preferably patients suffering from febrile conditions, but making sure that they had not had typhoid recently. All these blood drops were to be numbered and a key giving the clinical diagnosis in each case placed within a separate sealed envelope.

I left Montreal September 13th. Samples of blood from six patients were collected and forwarded as directed, on September 14th. On September 16th, the letter was delivered unopened at Buffalo, N. Y., to Dr. Bissell, the city bacteriologist for Buffalo, who kindly took charge of the key. At the end of an hour spent in examining the specimens, I wrote my diagnosis upon the outside of the sealed envelope. It will be seen from the subjoined signed statement, which Dr. Bissell kindly made at my request, that the results were perfectly in accord with the clinical diagnosis in each case, while the specimens, which were then examined by a number of competent bacteriologists, showed that good objective grounds existed for arriving at the conclusions given.

STATEMENT BY DR. MCTAGGART, RESIDENT PATHOLOGIST, MONTREAL GENERAL HOSPITAL.

The samples of blood were mailed to Dr. Johnston one day after he had left Montreal. Dr. Johnston had no knowledge of the contents of the "key," and no private means of knowing which of the numbers referred to typhoid and which to non-typhoid blood.

(Signed) D. D. McTaggart.

STATEMENT BY DR. BISSELL, CITY BACTERIOLOGIST, BUFFALO, N. Y.

Buffalo, September 16, 1896.

Received to-day from Dr. Wyatt Johnston a sealed letter, mailed in Canada, with postmark, "Montreal, September 14, 1896." This was opened by me and found to contain (*a*) six glass cover slips, numbered from 1 to 6, with a drop of dried blood on each, and (*b*) also a sealed envelope marked "key." Received from Dr. Johnston, after examining the blood by the (Widal) serum diagnostic test, the following report: No. 1, typhoid; No. 2, typhoid; No. 3, typhoid; No. 4, not typhoid; No. 5, not typhoid; No. 6, doubtful,

probably not typhoid. The key was then opened by me, and the clinical diagnosis from all cases found as follows: No. 1, typhoid; No. 2, typhoid; No. 3, typhoid; No. 4, malaria; No. 5, enlarged glands of neck; No. 6, heart disease.

(Signed) WILLIAM E. BISSELL.

It will be noticed that a qualified though correct opinion was given at the time of my making the report in one of the negative cases (No. 6). This doubt was owing to the fact that it was the last specimen examined, and that a partial agglutination appeared to take place at first, though motion was not abolished. Subsequent examination some hours later showed such lively motion that I should have had no hesitation in declaring it not to be typhoid, had the circumstances permitted that much delay before an opinion was given.

A ready means of diagnosis in typhoid fever is something which has long been desired by sanitary officials. The medical profession is proverbially lax with regard to the notification of typhoid cases, and we may assume that this neglect is in part due to the want of any adequate *quid pro quo* in return for such notification. Probably the assistance derived from a prompt bacteriological diagnosis, or even corroboration of diagnosis in the early stages of typhoid, will lead to the more uniform reporting of cases. Besides distinguishing typhoid from such well-characterized diseases as tuberculosis and malarial disease, this test may also be expected to clear up the mystery which surrounds those doubtful cases of so-called bilious fever, remittent fever, gastric fever, typho-malarial fever, etc., which are so common in times and places where typhoid is prevalent, and rare in the absence of typhoid, at least in temperate regions which are free from malaria.

Those who are called upon to investigate epidemics of typhoid are much perplexed by the large number of cases of ill-defined and transitory fever occurring among those personally exposed to the infection, and the impossibility of coming to anything like a definite conclusion upon the evidence hitherto obtainable as to whether these are to be regarded as cases of abortive typhoid or not. In my own experience, such cases have usually equaled or outnumbered the cases where the symptoms justified a definite diagnosis.

I may add a few words with regard to technic. I use a dry lens of about one-fourth inch focal distance. The dry blood drop is partly dissolved with germ-free water, and a drop of the solution obtained is placed upon a cover glass which has just been passed through a flame and mixed with a drop of a typhoid bouillon (a watery suspension of an agar culture also answers very well). This is placed over a hollow cell sealed by vaseline. I control the examination by comparing it with a blood drop from an undoubtedly typhoid case, and also with normal blood. It is also advantageous to place a minute drop of the blood solution upon the cover slip alongside the mixture of culture and serum, so as to satisfy one's self in negative cases that the blood contains no motile bacteria. Uniformity of temperature is the chief detail to be attended to, as the agglutination does not take place so well if the movements are sluggish.

A hot-water dish filled with warm water forms a cheap and convenient substitute for an incubator, and a simple warm stage made of a sheet of copper is also useful. In a well-warmed laboratory, however, the use of these adjuncts is unnecessary. Hollow cells are convenient, but not indispensable. For collecting the blood drop, any smooth surface suffices; cover glasses or slides have the advantage of being clean and sterile, but I have found ordinary writing paper or smooth cardboard most convenient, as it could be more easily labeled or forwarded. The swabs used for diphtheria outfits will answer, but the presence of extraneous substances, such as fibres, was found annoying. The presence of blood pigment is rather an advantage, as it enables the drop to be more easily focused. The small fibrin particles of clot sometimes bear a superficial resemblance to the islets of agglutinated typhoid bacilli, but are readily distinguished from them by the presence of leucocytes in their meshes.

One advantage of having the blood dried is that it insures it against contaminating growth occurring during shipment. In case any doubt as to the reaction exists at first, it will usually be dispelled by watching the preparations for some hours, or, if necessary, for a day or two. This permits a decided and progressive increase of motion in non-typhoid cases and allows the more perfect agglutination in the genuine ones.

The one indispensable factor is perfect purity of the culture. The one which I use was kindly forwarded me by Mr. J. J. Mackenzie, bacteriologist to the Ontario Provincial Board of Health, and was stated to have come originally from the Berlin Hygienic Institute. It grows typically on gelatin, potato, bouillon, agar, and milk; reacts typically with litmus agar, produces no indol or gas, and shows the motility and staining reactions characteristic of the Eberth bacillus.

I have made this communication because the method here suggested seems better adapted than those hitherto employed for bringing this test within the range of ordinary public-health laboratory work, and enabling it to be dealt with, if I may so express it, in a wholesale manner.[1]

This article was published in the "New York Medical Journal" of October 31, 1896. Further articles on the same subject were published by me in the "New York Medical Journal," November 28, 1896 (with Dr. D. D. McTaggart) in the "British Medical Journal," December 5, 1896; Circular of the Board of Health of the Province of Quebec, January 7, 1897 (with Dr. D. D. McTaggart) in the "Montreal Medical Journal," March, 1897; "Centralblatt für Bakteriologie," Baud XXI, 1897.

[1] Drying the blood as a preliminary step has enabled the Board of Health of the Province of Quebec to offer to the medical profession here a free public service of typhoid diagnosis by the serum method similar to that which is followed in diphtheria. Outfits consisting of a folded and sterilized piece of paper, in which the blood drop is sent inclosed in a suitable envelope, are placed in convenient depots. In case of negative results, an additional sample, taken by collecting a few drops of blood in a small glass tube, is examined, but this extra precaution is seldom necessary. As to the degree of accuracy which this application of the test may afford, it is too early to speak positively. From my experience hitherto, I am inclined to believe that it will compare not unfavorably with those obtained in the cases of diphtheria and tuberculosis. In one case the reaction was present on the third day.

LABORATORY OF THE BOARD OF HEALTH OF THE PROVINCE OF QUEBEC.

CIRCULAR ON ATTENUATED TEST CULTURES AS A SAFEGUARD AGAINST PSEUDO-REACTIONS IN SERUM DIAGNOSIS OF TYPHOID BY THE DRIED BLOOD METHOD.

MONTREAL, 7th January, 1897.

To the President of the Board of Health of the Province of Quebec:

SIR: In my work in serum diagnosis done jointly with Dr. D. D. McTaggart, we recently met with a series of peculiar partial reactions in which the dried blood solution from many perfectly healthy persons gave a very decided agglutination. The blood serum from the same persons was found much less liable to give these pseudo-reactions. This made it less easy to exclude other febrile diseases, and as with this test accuracy in the negative diagnosis is of great practical importance, others who may meet with similar pseudo-reactions will be interested in learning how they may be avoided.

These pseudo-reactions were not encountered in our earlier cases when attenuated cultures were used. They began to appear when we employed a short time virulent cultures, and disappeared again on resuming the use of attenuated ones. Active, virulent cultures, intensified by daily transplantation and growth, at the body temperature, are therefore not suitable for the dried blood test. Where only active cultures are employed, we do not think that the dried blood method can be considered to have had a fair trial.

The explanation of this difference appears to be that the serum contains relatively less of the substances causing agglutination than solution of the entire blood. Hence solutions of the entire blood react more intensely to the test than solutions of the blood serum alone. This was the reverse of what we had anticipated.

It is found that old laboratory stock cultures kept at room temperature, and transplanted at intervals of about one month, give us the best results. Bouillon test cultures grown from this stock for twelve to twenty-four hours at body temperature are found to react decisively with solutions of typhoid blood or typhoid serum, the reaction being, as a rule, well marked within fifteen minutes. With non-typhoid blood or serum solutions, the same test cultures give no reaction even after twenty-four or forty-eight hours' contact. Intraperitoneal injection of one c. c. of such living bouillon culture produces in guinea pigs a marked blood reaction and immunity without much disturbance of health. We find that the best results in cases of dried blood are obtained with cultures where the motion, as seen under the microscope, is of a rapid, gliding character, but free from darting movements. If the movement is sluggish, owing to too great attenuation of the culture, a few daily transplantations at body temperature will make it more active. Exact estimation of the degree of dilution has not been found necessary for ordinary diagnostic work when attenuated cultures are used. A very faint tint in the drop examined usually indicated sufficient strength. The solution should not be thick and viscid.

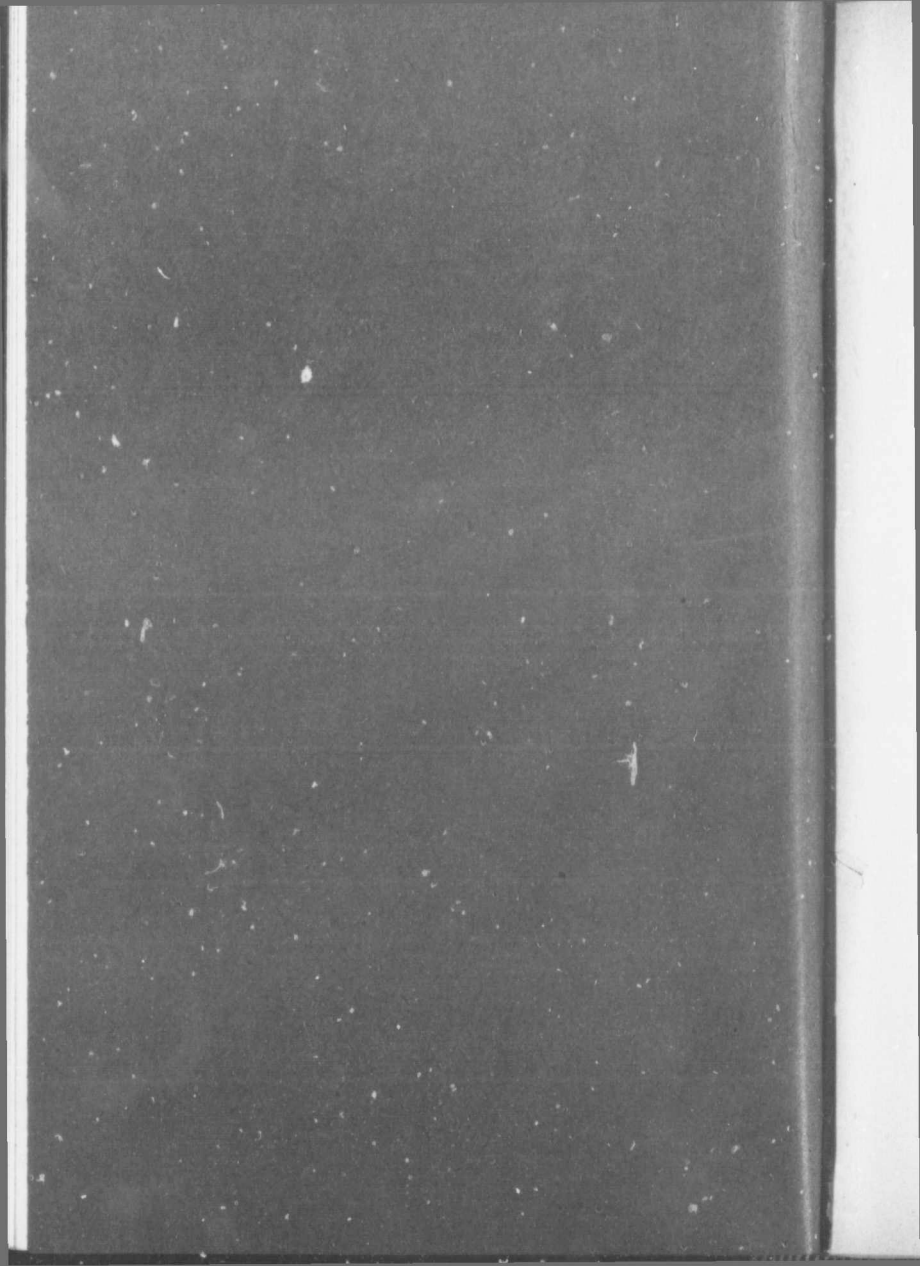
All the results which I have reported ("N. Y. Medical Journal," Oct. 3, 1896, and "British Medical Journal," Dec. 5, 1896,) were obtained with attenuated cultures. A report, giving some additional technical details, has been prepared, and can be sent to any who desire further information.

I remain, yours respectfully,

WYATT JOHNSTON,

Bacteriologist to the Board of Health, Province of Quebec.

NOTE.—Subsequent experience has confirmed the above statements as to the best method of technique with the cultures which I have used. From the recent literature of the subject it seems, however, established that the cultures used by others give the best results under different conditions, so that it is safer not to generalize.



PATHOLOGICAL REPORTS

MONTREAL GENERAL HOSPITAL.

No. III.

REFERENCE INDEX OF POST-MORTEMS

FROM

1883 TO 1895.

COMPILED BY

WYATT JOHNSTON, D. D. MACTAGGART

AND

F. L. JOHNSTON.

Gazette Printing Co., Montreal.

In trying to obtain an idea of the associations under which a particular disease or lesion has been met with in the experience of others, one becomes impressed with the difficulty of obtaining statistical information. In the casuistic literature only those aspects of the cases are treated fully which attract attention at the time. In the matter of pathological records it is difficult to find a sufficient number of cases showing all the chief complications of the principal disease.

The increased attention now being paid to the indexing of pathological reports and the publishing brief abstracts of the chief pathological changes found in the routine autopsy records promises to yield a mine of valuable information. The manner in which this can best be recorded is not an easy matter to decide, but, no abstract or synopsis of an autopsy is as compact and convenient as the simple publication of the anatomical diagnosis in all the cases coming to autopsy during the year as was done for the first time by Drs. Adami and Martin in the case of the Royal Victoria Hospital, Montreal.

In order to facilitate reference we have added a special index in which the morbid conditions are placed under the particular organ or system to which they belong and recorded by the reference number. We have to thank Dr. Martin for permitting us to consult the very full (unpublished) nosological list compiled by him.

It was thought best not to depend upon an alphabetical arrangement but to classify as far as possible in a nosological manner. The perfect type of such a classification would be one where the conditions were "double-indexed" and recorded, first under their relation to general pathology and then under the organ affected. In classifying the diseases of the different organs the natural order to follow is that used by most text-books on pathology, viz. : 1. anomalies ; 2. circulatory disturbances ; 3. inflammations and infections (it becomes harder and harder for us to separate the two in view of recent investigations) ; 4. progressive changes ; 5. retrogressive changes, lastly, 6. injuries and foreign bodies.

A complete system of double entry was found in practice to make the index too bulky and we have only duplicated in certain special instances. In deciding when double indexing was necessary we were largely guided by whether the clinical importance of the particular lesion in question was much influenced by the organ affected. Thus amyloid degeneration being

capable of very wide distribution, as a rule, without much clinical interest attaching to its particular location, was not double-indexed; whereas tumours, suppurations and any other conditions in which widely different clinical results depend upon the situation of the lesions, were entered both under the general and special pathological headings. In the case of the lesions which only affect one kind of tissue, e.g. fractures, all that appeared necessary was to enter them in one place, under bones. Owing to the diversity and clinical importance of the localisation in tuberculous lesions they were double indexed. Where the index is not duplicated a cross reference is given.

The fact that the autopsies were performed by a number of different persons and that during the period covered there were no less then 15 complete changes of management of the laboratory, makes it difficult to classify uniformly. Where doubt exists as to the opinion held we have employed the phraseology used by the person who made the autopsy. In the anatomical diagnoses the principal disease is given first rather than the immediate cause of death.

Common conditions of little clinical significance are mentioned as a rule when they had a special interest in that particular case or were strikingly well marked. As to how far these records have an exact statistical value as showing the relative frequency of different morbid conditions, this is in our opinion much less than in the case of a small series covering a short time only, where either a positive or negative result is recorded in each case. The observations of a dozen men extending over as many years are bound to be diverse and unequal as to the frequency with which particular lesions are looked for. When however, persistent frequency is noted of any particular condition there is all the more reason to rely on the result of the observations. We notice that the proportion which pulmonary tuberculosis bears to the total deaths from all causes is practically the same as that determined by mortality statistics for this city and province. The frequency with which interstitial changes are noted in the kidneys also accords with the recent literature of the subject. The cases may be as a whole taken as typical of the ordinary run of hospital cases though as the autopsy is not a matter of hospital routine but requires the consent of the relatives there is a relatively large proportion of rare and exceptional cases, because in these more trouble is naturally taken to obtain this consent.

One thing noticed was a decided falling off in the percentage of cases of amyloid degeneration during the last 10 years, doubtless owing to improved methods of treating suppurative conditions and to the better facilities for making early diagnosis and averting the graver forms of many chronic diseases.

The rarity of certain diseases, notably malaria, is peculiar to the locality. In regard to rare conditions, however, these come in a very irregular manner. Thus trichinosis was only met with in two cases during the 20 years covered by the hospital post-mortem records, and both of these occurred within a few months.

I may instance a remark of Dr. Osler in his text-book to the effect that in his Montreal cases internal hæmorrhagic pachymeningitis had been absent while it was of frequent occurrence in the service at the Blockley Alms House in Philadelphia. Personally I have found this condition to be of frequent occurrence in Montreal, but only in autopsies in coroners' cases, this special class of material not having been available in Dr. Osler's time.

It is to be regretted that the means at our disposal do not permit of the publication at present of a full analysis of the pathological material in the autopsy records. It is hoped to do so later in a series of shorter reprinted articles. In addition to the present series of 934 post-mortems, we also hope shortly to index a series of 700 post-mortems, which were made by Dr. Osler during the period from 1876, when the pathological department of the hospital was first established, to 1882, as well as a series of 400 medico-legal autopsies made since 1893 by myself.

It must be mentioned that the series now indexed is composed of autopsies made before any properly equipped pathological laboratory existed in connection with the hospital. In consequence the bacteriological and microscopical study is much more scanty than would otherwise have been the case. At present, thanks to a better laboratory and a larger staff we are able to follow the current methods of the modern laboratories, in which a microscopical and bacteriological examination of all the principal organs is made in every case as a matter of routine. On comparing the present and past pathological work of the General Hospital, what impresses me most is the high standard of work done by my predecessors, Drs. Osler and Adami, under very unfavourable circumstances as to laboratory equipment and assistance.

I have great pleasure in recording my appreciation of the substantial encouragement which the Hospital Committee of Management have given to the pathological laboratory at a time when very heavy calls were being made upon the Hospital funds.

We hope in the future to be able to keep our pathological indexes published up to date and would be glad to see an inter-laboratory arrangement whereby copies of autopsy reports could be furnished to persons interested in studying any special class of cases and willing to defray the expense of the clerical work involved.

W. JOHNSTON.

MONTREAL, August, 1897.

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GENERAL AND SPECIAL INDEX OF POST MORTEMS PERFORMED AT THE MONTREAL GENERAL HOSPITAL DURING THE PERIOD FROM 1883 TO 1895.

AUTOPSIES MADE BY: WM. OSLER, J. G. ADAMI, F. G. FINLEY, R. J. B. HOWARD, W. H. JAMIESON, WYATT JOHNSTON, H. A. LAFLEUR, C. F. MARTIN, W. P. SUTHERLAND AND E. P. WILLIAMS.

INDEX PREPARED BY WYATT JOHNSTON, D. D MACTAGGART AND W. F. L. JOHNSTON.

GENERAL INDEX.

1883.

701 (Osler)—Female, 40. Jan. 10, 1883.

CHRONIC ULCERATIVE PHTHISIS. Cavity in right lung; fibroid and fatty liver, with Liebermeister's furrows.

702 (Osler)—Female, 40. Jan. 10, 1883.

PURULENT BRONCHITIS. Cystic bronchocele; cyst at apex of left pleura, continuous with bronchocele. Sub-peritoneal fibroid of uterus.

703 (Osler)—Female, 57. Jan. 14, 1883.

FRACTURE OF BASE OF SKULL. Slight atheroma of mitral and aortic valves; chronic adhesive pleurisy; calcareous plate in right pleura; cartilaginous looking fibroid areas of left pleura; meningeal hæmorrhage and multiple ecchymoses of pia; hæmorrhage of pons; fatty liver; granular kidneys, with cysts.

704 (Osler)—Male, 25, private case. Jan. 24, 1883.

SPLENIC MYELOGENOUS LEUCHÆMIA. Hyperplasia of spleen, 7½ lbs.; hyperplasia of mesenteric and retro-peritoneal glands, and bone marrow; dilatation (and thrombosis?) of portal and mesenteric veins; catharrhal enterocolitis, hyperplasia of solitary follicles to double normal size; leuchæmic liver and kidneys; dilatation and fatty degeneration of heart; pyoid marrow; ulcerative cystitis.

705 (Osler)—Child, 5 months, private case. Jan. 27, 1883.

ŒDEMA OF GLOTTIS. Wrinkled condition of aryepiglottic folds; true and false vocal cords covered with catharral exudation; spleen large.

706 (Osler)—Female, 26. Jan, 27, 1883.

PHTHISIS. PYOPNEUNOTHORAX. Perforation near anterior border of upper lobe of left lung; empyema; extensive tuberculosis with cavities; mitral and aortic valves thickened; a few spots of atheroma in aorta at lower part of arch; uterine mucosa congested; varicosity of ovarian veins.

707 (Osler)—Female, 4. Feb. 2, 1883.

DIPHTHERIA. Tracheotomy; diphtheritic membrane over epiglottis, larynx and trachea, forming a complete cast above tracheotomy incision, below orifice mucosa deeply congested; left lung collapsed, commening pneumonia in right lung.

708 (Osler)—Male, 26. Feb. 6, 1883.

ULCERATIVE ENDOCARDITIS. Fusion of aortic segments. Thick, fleshy vegetations on valve; perforation, 3 mm from edge of left half of fused valve; vegetations extend below base of single segment towards mitral valve. Hypertrophy and dilatation of heart. Lungs hyperæmic; bases oedematons. Kidneys large (cyanotic induration); show old and recent infarcts, and numerous hæmorrhagic infarcts in cortices. Spleen acute enlargement; intestines show infarctions beneath mucosa; fluke deformity of left liver lobe; catarrhal gastritis and ecchymosis. Acute hæmorrhagic embolic lepto-meningitis.

709 (Osler)—Female, 16. Feb. 18th, 1883.

PHTHISIS. Numerous cavities; fibroid change in both lungs; acute tuberculous pleuritis with subpleural hæmorrhage; miliary tubercles of spleen; tuberculous ulceration of intestine, most marked in jejmum and cæcum; tuberculosis of vagina. Liver fatty.

710 (Osler)—Male, 23. Feb. 19th, 1883.

PULMONARY PHTHISIS. Emphysema; rupture of aneurism of pulmonary artery; mesenteric glands enlarged.

711 (Osler)—Male, 6. Feb. 20, 1883.

DIPHTHERIA of pharynx and posterior nares; enlargement of glands of neck.

712 (Osler)—Private case. March 3, 1883.

FIBRO MYOMA OF UTERUS.

713 (Osler)—Male, 27. March 4, 1883.

ACUTE GENERAL MILIARY TUBERCULOSIS. Lungs stuffed with miliary tubercles, caseous nodules at apices; hyperplasia of spleen (3 times, normal size); a few miliary tubercles in calices of kidneys.

714 (Osler)—Male, 12. March 10, 1883.

CARIES OF CERVICAL VERLEBRÆ. Tuberculosis; Cavity in lung, apex (size of walnut); amyloid spleen; nutmeg liver.

715 (Osler)—Female, 5. March 9, 1883.

DIPHTHERIA. Membranous laryngitis and tracheitis; atelectasis of left lung, inflation of right lung.

716 (Osler)—Male, 19. March 8, 1883.

CHRONIC PARENCHYMATOUS NEPHRITIS. Anasarca; chylous ascites and hydrothorax; (3 to 4 pints milky fluid in peritoneum); mesenteric and retro-peritoneal lymph glands enlarged; thoracic duct normal.

717 (Osler)—Male, 32. March 15, 1883.

SUPPURATIVE ARTHRITIS OF HIP AND KNEE JOINTS. Nutmeg liver; pyæmia; icterus; amyloid kidney.

718 (Osler)—Child, 5 months. Private case.

ROUND CELLED SARCOMA OF KIDNEY. Sarcomatous thrombosis with obliteration of right iliac artery and vein and vena cava.

719 (Osler)—Female, 53. Private case. March 23, 1883.

CANCER OF CERVIX UTERI. Left hydronephrosis; nutmeg liver; Cancerous infiltration of rectum and bladder.

720 (Osler)—Female, 65. March 26, 1883.

GLIOSARCOMA OF BRAIN in region of 3rd ventricle and infundibulum; Brown athrophy of heart; adhesive peritonitis and perihepatitis. Dilatation of bile-ducts.

721 (Osler)—Male, 76. March 26, 1883.

FRACTURE OF BASE OF SKULL (Notes not entered).

722 (Osler)—Male, 80.

SOFTENING OF BRAIN. Multiple atheroma of arteries: miliary aneurisms; atheroma of coronaries; hypertrophy of left verticle; atheroma of heart valves. Slight chronic interstitial myocarditis; emphysema of margins of lungs; œdema of lungs; mucopurulent bronchitis; thickening of capsule of spleen; extreme senile athrophy of kidneys with cysts.

723 (Osler)—Private Case, Male. March 27, 1883.

CHRONIC INTERSTITIAL NEPHRITIS. Hyperæmia and œdema of lungs; fatty liver; hypertrophy of middle lobe of prostate; diverticulum of bladder; œdema of pia mater. Uræmia (?)

724 (Osler)—Female, 28. March 28, 1883.

PHTHISIS; ULCERATIVE ENDOCARDITIS.

725 (Osler)—Private case, 73. March 22nd, 1883.

ANEURISM OF AORTA, ascending arch. Rupture into pericardium.

726 (Osler) Male, 55. March 29, 1883.

PHTHISIS. Emphysema of anterior margins of lungs; large cavity in left lung.

727 (Osler)—Female, 39. April 7, 1883.

CHRONIC INTERSTITIAL NEPHRITIS. Pregnancy. Uræmia. Congestion and cloudy swelling of kidneys; hyperaemia and oedema of lungs; punctate ecchymosis of liver. Hæmorrhage of left corpus striatum and left ventricle; gestation nearly at term; fœtus in utero.

728 (Osler)—Male, 65. April 10, 1883.

CEREBRAL HÆMORRHAGE.

729 (Osler)—Female, 50. April 11, 1883.

ACUTE BRONCHO-PNEUMONIA. Atheroma of aortic and mitral valves; cloudy swelling of kidney; slight fatty liver; atheroma of aorta; chronic bronchitis and emphysema in both lungs.

730 (Osler)—Male, 48. April 12, 1883.

CHRONIC TUBERCULOUS PERITONITIS WITH EFFUSION. PERFORATION OF TUBERCULOUS ULCER OF INTESTINE. Tuberculous entero-colitis; extensive tuberculosis with cavities in both lungs. Tubercles in capsule of spleen. Tuberculous perihepatitis.

731 (Osler)—Male, 28. April 14, 1883.

CHRONIC ULCERATIVE PHTHISIS of both lungs. Fluke shape deformity of left lobe of liver; fatty liver; one small tuberculous ulcer in ileum.

732 (Osler)—Male, 63. April 11, 1883.

CHRONIC INTERSTITIAL NEPHRITIS. Dilatation and fatty degeneration of heart; emphysema; adhesions at apices.

733 (Osler)—Male, 8. April 15, 1883.

DIPHTHERIA. Apical tuberculosis of lung (both); cloudy swelling of kidneys; pseudo-membranous pharyngitis.

734 (Osler)—Male, 53. April 15, 1883.

CHRONIC ULCERATIVE PHTHISIS of both lungs; slight insterstitial nephritis; several small tuberculous; ulcers in ileum and colon.

735 (Osler)—Private.

UMBILICAL HERINA.

736 (Osler)—Not entered.

737 (Osler)—Female, 52. April 24, 1883.

COLLOID CANCER OF PANCREAS. Secondary in lung; no cancer in liver.

738 (Osler)—Male, 35. May 1, 1883.

PHTHISIS. Cavity of left upper lobe; acute pleurisy over right lower lobe; tuberculous enteritis; Chyle extravasations in mesentery; enlarged lymph glands.

739 (Osler)—Female, 60. May 9, 1883.

PERNICIOUS ANÆMIA. Brown atrophy of heart; chronic interstitial nephritis; stomach mucosa, pale thin and tough; liver elongated left lobe; aorta atheromatous; iliacs rigid; submucous fibromyoma uteri.

740 (Osler)—Male, 8. May 9, 1883.

ACUTE NEPHRITIS FOLLOWING SCARLATINA. Anasarca; ascites; hydrothorax; atelectasis.

741 (Osler)—Female, 18. May 14, 1883.

CHRONIC ULCERATIVE PHTHISIS of both lungs. Tuberculous pleurisy adhesive perihepatitis; eight to ten tuberculous ulcers in ileum; a single ulcer in colon; adhesive pelvic peritonitis; hydrosalpynx.

742 (Osler)—Male, 38. May 30, 1883.

ACUTE SPINAL MYELITIS.

743 (Osler)—Male, 60. May 29, 1883.

HYPERTROPHY AND DILATATION OF HEART on both sides; Chronic mitral endocarditis with stenosis; presistent foramen ovale; emphysema; chronic interstitial nephritis; cirrhosis of liver; ascites; hydrothorax.

744 (Osler)—Female, 23. June 3rd, 1883.

GENERAL MILIARY TUBERCULOSIS. Adhesive pericarditis; miliary tuberculosis of lungs and kidneys; extensive adhesive pleurisy; caseous bronchial glands.

745 (Osler)—Female, 85. June 11, 1883.

CANCER OF STOMACH. Secondary cancer (?) of retroperitoneal glands; fibroid change of myocardium; brown atrophy of heart; atheroma of aorta; pigmentation and pseudo tubercles of pleura; emphysema; chronic bronchitis; fibroid and fatty liver; thrombosis of left renal vein; papillomatous cyst of left ovary.

746 (Osler)—Male, 6. July 20, 1883.

ACUTE TUBERCULOUS MENINGITIS. (Notes not entered).

747 (Osler)—Male, 24. July 16, 1883.

PHTHISIS. Cavities in both lungs; dilatation of right side of heart; spleen enlarged, with two supplamentary spleens; amyloid liver; granular kidney with amyloid. Enlarged gland in portal fissure.

748 (Osler)—Male, 32. June 16, 1883.

PERNICIOUS ANÆMIA. Lemon yellow tint of skin; heart hypertrophied; (410 gm.) and dilated with fatty degeneration; œdema of lungs; spleen enlarged and adherent to diphragm; kidneys anæmic enlarged (200 gm.) capsule adherent; liver yellowish-brown; marrow of femur red; mesenteri glands deep red.

749 (Osler)—Male, 52. August 19, 1883.

TYPHOID FEVER. Typhoid ulceration of Peyer's patches; enlargement of mesenteric glands; enlargement of spleen; pneumonia of right lung, congestion and œdema at bases of both lungs.

750 (Osler)—Male. Aug. 1, 1883.

TYPHOID ULCERATION OF INTESTINES. Swelling of mesenteric glands; pneumonia at bases of lungs; cloudy swelling of kidneys and liver; ecchymosis over surface of liver; gritty particles in gall bladder.

751. (Osler)—Female, 4. August 4, 1883.

(EDEMA OF GLOTTIS. Tracheotomy; abnormal degree of mobility of cervical vertebræ.

752. (Osler)—Male, 39. August 2, 1883.

CONTUSION OF HEAD; HÆMORRAGE INTO RIGHT EXTERNAL CAPSULE. Red softening of brain. Concussion ecchymoses on island of Reil; commencing meningitis (?) Hyperæmia of lungs.

753 (Osler)—Male, 64. August 3, 1883.

ACUTE PLEURISY. Congestion of lungs; venesection; fatty and parenchymatous degeneration of heart; slightly granular kidneys; atheroma of coronary arteries; fatty and cirrhotic liver.

754 (Osler)—Male, 39. August 12, 1883.

DOUBLE TUBERCULOUS PYELITIS. Acute miliary tuberculosis of both lungs and pleura; at both apices small caseous areas; tuberculosis of bladder; fistula of urethra; miliary tubercules in liver; hyperplasia and tuberclosis of spleen.

755 (Osler)—Male, 42. August 9, 1883.

HYPERTROPHY AND DILATATION OF HEART. General arterio-sclerosis (venesection).

756 (Johnston)—Male, 36. August 13, 1883.

FRACTURE OF BASE OF SKULL. Laceration of scalp; œdema of lungs; Necrosing cystitis; menengeal hæmorrhage.

757 (Johnston)—Male, 19. (First Ambulance Case, M. G. H.) August 13, 1883.

RUPTURE OF LEFT KIDNEY AND SPLEEN. Fracture of ribs in railway accident.

758 (Johnston)—Male, 39. August 13, 1883.

TYPHOID FEVER; PERFORATION AND PERITONITIS. Typhoid ulceration of intestines; hyperplesia of spleen and mesenteric glands; cloudy swelling of kidneys.

759 (Osler)—Private case. August 17, 1883.

PRIMARY CANCER OF LIVER. Secondary in lungs.

760 (Osler)—Male, 21. August 17, 1883.

CHRONIC SUPPURATIVE CALCULOUS PYELITIS OF BOTH KIDNEYS. Amyloid kidney, liver and spleen. Ulcerative colitis.

761 September 25, 1883.

CHRONIC ENDOCARDITIS with hypertrophy and dilatation. Chronic bronchitis; hyperplesia of spleen; arteriosclerosis; Liebermeister's furrows of liver; catarrhal gastritis.

762 (Osler)—Male, 29. September 23, 1883.

EPITHELIOMA OF PENIS. Secondary of inguinal glands; infarction (cancerous (?)) and abscess at base of right lung.

763 (Osler)—Female, 43. October 1, 1883.

TYPHOID FEVER; DIPHTHERIA OF TRACHEA AND BRONCHI. Slight chronic interstitial nephritis; hyperplasia of spleen; typhoid ulceration of intestines.

764 (Osler)—Male, 23, October 3, 1883.

FRACTURE OF SKULL. Intermeningeal hæmorrhage and concussion ecchymoses of brain; collapse and hyperæmia with œdema of lungs.

765 (Osler)—Male. October, 26, 1883.

ANEURYSM OF ABDOMINAL AORTA. Rupture into peritoneum.

766. (Osler)—Male, 35. October 9, 1883.

PHTHISIS. Tuberculous peritonitis with effusion; cavity in left lung; gray hepatization of right lung; hyperplasia of spleen; catarrhal gastritis; cirrhosis of liver.

767 (Osler)—Female. October 14, 1883.

RENAL CIRRHOSIS. Hypertrophy of left ventricle; hyperæmia and œdema of lungs; cheesy and calcified nodule at apex; kidneys 36 grm. each; extreme chronic insterstial nephritis with arterio-sclerosis; dysenteric ulceration of intestine; pyosalpynx; cyst in right broad ligament; atheroma of aorta; œdema of pia.

768 (Osler)—Male, 27. November 1st, 1883.

PHTHISIS. Large cavities in both lungs; acute verrucose endocarditis.

769 (Osler)—Female. 18. November 7, 1883.

TYPHOID FEVER. Ulceration of small intestine only; hyperplasia of mesenteric glands and spleen; cloudy swelling of liver; Membranous vaginitis.

770 (Osler)—Male, 31. November 13, 1883.

AMPUTATION OF LEFT LEG; ERYSIPELAS. Warty endocarditis of mitral; spleen slightly enlarged; chronic interstitial nephritis.

771 (Osler)—Male, 25. November 25, 1883.

CHRONIC AORTIC ENDOCARDITIS: ULCERATIVE ENDOCARDITIS OF MITRAL AND AORTIC VALVES. Great hypertrophy and dilatation of heart; acute lobar pneumonia; infarcts of spleen with hyperplasis, 300 grm., spleen enlarged, 600 grm.; old infarcts. Brain, normal.

772 (Osler)—Male, 37. November 29, 1883.

ACUTE LOBAR PNEUMONIA. P. M. invaginations of intestines; acute pericarditis with effusion; seropurulent right pleurisy with effusion and slight area of pneumonia at anterior margin; recent lymph over upper surface of liver.

773 (Osler)—Male, 36. Dec. 5, 1883.

TERTIARY SYPHILIS. Sclerosing osteitis of right tibia and of skull cap, with suppurative periostitis and pachymeningitis. (Only brain entered.)

774 (Osler)—Female, 36. Dec. 5, 1883.

SYPHILITIC THROMBOSIS OF RIGHT MIDDLE CEREBRAL ARTERY.

775 (Osler)—Male, 45. Dec. 9, 1883.

VALVULAR HEART DISEASE.

776 (Osler)—Male, 40. Dec. 17, 1883.

VALVULAR HEART DISEASE. Anasarca Hypertrophy and dilatation of both ventricles; more marked on right side; mitral incompetency; slight fibroid myocarditis; emphysema; brown induration and hypostatic pneumonia; spleen enlarged and hard; granular kidneys with infarcts; chronic catarrhal gastritis.

777 (Osler)—Female. December 17, 1883.

PURULENT ARTHRITIS; PYAEMIA.

778 (Osler)—Female, 26. Dec. 19, 1883.

CHRONIC MITRAL AND AORTIC ENDOCARDITIS. Oedema of lungs with induration; chronic bronchitis and infarcts in lung; cyanotic induration of kidneys; erosions of os uteri; calcification of fimbriae of fallopian tubes; cirrhosis of liver.

779 (Osler)—Male, 25. Dec. 20, 1883.

TYPHOID FEVER. Ulceration of intestines; hyperplasia and infarct of spleen; chronic interstitial nephritis, with cloudy swelling; encysted abscess of lungs, with scattered areas of consolidation.

780 (Osler)—Male, 23. Dec. 26, 1883.

FRACTURE OF LEG, PYAEMIA. Suppurative osteitis; acute septic peritonitis; chronic adhesive pleuritis with interstitial pneumonia; suppurative perinephritis on R. side; (appendicitis (?)) Triangular shape of left kidney.

1884

781 (Osler)—Female, 43. Jan. 12, 1884.

ELEPHANTIASIS OF LABIA PUDENDI, REMOVAL, PYÆMIA. Suppurative arthritis of both wrists; psoriasis of both ankles; slight enlargement of spleen; cloudy swelling of liver; abscesses in left pelvic wall; extensive syphilitic ulcer of rectum with stricture. Recto-vesico-vaginal fistula.

782 (Osler)—5. Jan. 14, 1884.

TUBERCULOUS MENINGITIS. Dropsy of ventricles; lymph and miliary tubercles at the base of brain; miliary tuberculosis of lungs, pleurae, spleen, peritoneum, kidneys and liver, catarrhal gastritis.

783 (Osler)—Female, 3. Jan. 22nd, 1884.

DIPHTHERIA of tonsils and epiglottis; acute capillary bronchitis; recent pleuritic adhesions: uvula bifid; bronchial glands swollen and congested with caseous masses and a few miliary tubercles. Miliary tuberculosis of lungs; broncho-pneumonia; supplementary spleen; miliary tuberculosis of spleen, liver and kidneys; tuberculous enteritis; ecchymosis and hæmorrhagic erosions of stomach.

84 (Osler)—Female, 3. Jan. 30, 1884.

NOMA OF VULVA.

785 (Osler)—Male, 65. Feb. 1, 1884.

FATTY AND CIRRHOTIC liver. Cirrhotic kidneys.

786 (Osler)—Male, 24. Feb. 1st, 1884.

ULCERATIVE ENDOCARDITIS.

787 (Olser)—Male, 65. Feb. 1st, 1884.

MULTIPLE SOFTENING OF BRAIN in left frontal and parietal regions and angular gyrus; subarachnoid œdema and atheroma of arteries; miliary aneurysms; hypertrophy of left side of heart with dilatation and atheroma of valves; atheroma of coronaries; slight atheroma of aorta; dissecting aneurysm of right iliac artery; small granular kidneys; emphysema and œdema of lungs.

788 (Osler)—Male, 40. Feb. 7, 1884.

CANCER OF LIVER.

789 (Osler)—Male, 38. Feb. 7, 1884.

PEMPHIGUS. Sero-purulent pleurisy of left side; bronchitis; perihepatitis fibrosa.

790 (Osler)—Male, 45. Feb. 14, 1884.

ACUTE LOBAR PNEUMONIA, all lobes of right lung; spleen enlarged and soft; catarrhal obstruction of common bile duct; jaundice; cirrhosis of liver; cloudy swelling of kidneys.

791 (Sutherland)—Male, 33. April 14, 1884.

PNEUMOTHORAX. Pulmonary Tuberculosis.

792 (Sutherland)—Male, 1. May 10, 1884.

TUBERCULOUS MENINGITIS.

793 (Sutherland)—Male, 47. May 14, 1884.

PERNICIOUS ANÆMIA.

794 (Sutherland)—Female. May 16, 1884.

DIPTHERIA. Tracheotomy.

795 (Sutherland)—Male, 56. May 2, 1884.

THORACIC ANEURYSM.

796 (Sutherland)—Male, 31. May 16, 1884.

CIRRHOTIC AND FATTY LIVER. Jaundice; obstruction of cystic duct by fibroid thickening; obsolete tubercles in apices of lungs; œdema with partial pneumonia of bases of lungs; old ulceration of larynx; chronic parenchymatous nephritis; jaundice;

797 (Sutherland)—Male, 50. May 21, 1884.

CHRONIC INTERSTITIAL NEPHRITIS. Small granular kidneys; hypertrophy and dilatation of heart (valves normal); œdema of lungs; nutmeg liver.

798 (Sutherland)—Male, 17. May 22, 1884.

TUBERCULOUS PERITONITIS (grape tuberculosis). General tuberculosis; chronic adhesive peritonitis; tuberculous left pleurisy with effusion; atalectasis of left lung; aortic verrucose endocarditis.

799 (Sutherland)—Male, 6. May 21, 1884.

DIPHTHERITIC LARYNGITIS AND TRACHEITIS. Pharynx free; slight broncho pneumonia.

800 (Sutherland)—Male, 68. May 28, 1884.

CEREBRAL HEMORRHAGE.

801 (Sutherland)—Male, 65. May 30, 1884.

PERNICIOUS ANÆMIA. Dilation of heart; milkpatch; aortic endocarditis; hypertrophy of left heart; hæmorrhagic retinitis.

802 (Sutherland)—Male, 52. June 4, 1884.

CHRONIC INTERSTITIAL NEPHRITIS. Contracted liver; hypertrophy of heart with dilatation; slight cirrhosis of liver; catarrhal gastritis.

803 (Sutherland)—Male, 21. June 8, 1884.

ACTULE LOBAR PNEUMONIA OE RIGHT LUNG. Double acute fibrinous pleurisy; acute fibrinous pericarditis.

804 (Sutherland)—Male, 30. Aug. 14, 1884.

PHTHISIS, with cavities in both lungs; cirrhotic and fatty liver; horseshoe kidney; double hydronephrosis; left pyelonephrosis; calculus in calices and large calculus in pelvis: bladder and urethra healthy.

805 (Sutherland)—Female, 56. Sept. 13, 1884.

TETANUS. Abscess with particle of leather from wound in sole of left foot; acute neuritis of left sciatic nerve with great œdema of sheath.

806 (Sutherland)—Female, 18. Aug. 19, 1884.

TYPHOID FEVER.

807 (Sutherland)—Male 27. Sept. 18, 1884.

PYOPNEUMOTHORAX. Tuberculosis of lungs: seropurulent pleurisy; small perforation communicating with cavity in left apex.

808 (Sutherland)—Female, 42. Oct. 17, 1884.

PERITONITIS. Abscess of liver; chronic perityphlitis (appendicitis ?)

809 (Sutherland)—Male, 28. Oct. 27, 1884.

VALVULAR HEART DISEASE. Hydrothorax; anasarca; cyanotic induration of kidneys; fatty and cirrhotic liver; hyperplasia of spleen with infarcts; milk patch on heart; incompetency of aortic valves; hypertrophy and dilatation of heart; recurrent endocarditis of aortic valves.

810 (Sutherland)—Female, 24. Nov. 13, 1884.

CATARRHAL COLITIS. Scars (from burning?) of face and arms.

811 (Sutherland)—Male, 24. Dec. 5, 1884.

TYPHOID FEVER. Typhoid ulceration of ileum; large firm spleen; fatty liver.

812 (Sutherland)—Female, 19. Dec. 6, 1884.

CHRONIC PARENCHYMATOUS NEPHRITIS. Acute pleurisy; albuminuric retinitis.

815 (Sutherland)—Male, 57. Dec. 10, 1884.

ACUTE RIGHT LOBAR PNEUMONIA. Acute right pleurisy with effusion; cyanotic induration of kidneys; cancerous (?) ulceration of œsophagus below level of bifurcation of trachea.

816 (Sutherland)—Male, 30. Dec. 19, 1884.

HYPERTROPHY AND DILATATION OF HEART. Mitral endocarditis with aneuryism of valve; aortic endocarditis; acute pericarditis; acute localized left pleurisy.

817 (Sutherland)—Male, 65. Dec. 19, 1884.

STRICTURE OF URETHRA. (Pyaemia?) Abscess of penis and prostate; right hydronephrosis; dilatation of bladder.

818 (Sutherland)—Male, 50. Dec. 26, 1884.

MALIGNANT ENDOCARDITIS. Pneumonia; acute meningitis.

819 (Sutherland)—Female, 25. Dec. 23, 1884.

ACUTE LOBAR PNEUMONIA. Anchylosis right knee.

1885

820 (Sutherland)—Male, 31. Feb. 14, 1885.

TUBERCULOSIS. Chronic phthisis; caries of vertebral; tuberculosis and amyloid of liver, kidneys and spleen; thrombosis of left common and external iliac veins.

821 (Sutherland)—Male, 73. Feb. 22, 1885.

CHRONIC INTERSTITIAL NEPHRITIS. Arteriosclerosis; hypertrophy and dilatation of heart; cirrhosis of liver and spleen; anasarca; ascites; hydrothorax.

822 (Sutherland)—Male, 23. Feb. 20, 1885.

CHRONIC PHTHISIS, OF LUNGS. Tuberculous ulceration of larynx and intestine; Caries of body of 5th lumbar vertebra; psoas abscess; tuberculous orchitis.

823 (Sutherland)—Male, 43. Jan. 13, 1885.

CANCER OF STOMACH at pylorus with dilatation; cancerous infiltration of pancreas.

824 (Sutherland)

DYSENTERY. Abscess of liver.

825 (Howard)—Female, 21. March 31, 1885.

TUBERCULOUS PERITONITIS. Laparotomy; adhesions about pelvis and pelvic abscess; miliary tuberculosis of lungs, liver and kidneys.

826 (Howard)—Male, 20. April 11, 1885.

TUBERCULOUS PYOPNEUMOTHORAX. Collapse induration of left lung with adhesive pleuritis; haemothorax; nutmeg liver; fatty kidney; chronic parenchymatous nephritis.

827 (Howard)—Female, 13. April 12, 1885.

ACUTE SEPTIC PERITONITIS. Pyosalpinx. Acute fibrinous pleurisy.

828 (Howard)—Male, 30. May 2, 1885.

ERYSIPELAS. Broncho pneumonia; granular kidneys.

829 (Howard)—Female, 40. May 6, 1885.

CHRONIC PHTHISIS WITH CAVITY. Amyloid spleen; lacing lobe of liver; fatty kidney.

830 (Howard)—Male, 52. May 9, 1885.

BRONCHO PNEUMONIA. Slight interstitial nephritis.

831 (Howard)—Female, 42. May 16, 1885.

CROUPOUS PNEUMONIA right lung; parovarian cyst.

832 (Howard)—Male, 10. May 16, 1885.

DIPHTHERIA. Fatty degeneration of heart; catarrhal laryngitis; broncho pneumonia (convalescence from diphtheria.)

833 (Howard)—Male, 75. May 29, 1885.

CEREBRAL SOFTENING, size of marble, in pons; atheroma of aortic valves and aorta; infarction of spleen; granular kidneys.

834 (Howard)—Male, 63. June 10, 1885.

CANCER OF RECTUM. Secondary in peritoneum, mesenteric and retro-peritoneal glands; thrombosis of pelvic veins; broncho pneumonia.

835 (Howard)—Male, 16. June 18, 1885.

CEREBELLAR ABSCESS, (rupture into 4th ventricle); hyperaemia and acute inflation of lungs; granular condition of lateral ventricles and choroid plexus.

836 (Howard)—Female, 50. June 19, 1885.

CEREBRAL HÆMORRHAGE into pons. Large granular kidney (right); uterus bicornus; fibro myomata; almost complete atrophy of left kidney.

837 (Howard)—Male, 45. June 20, 1885.

TUBERCULOSIS PHTHISIS. Universal adhesion of both pleurae; tuberculous pleuritis; phthisis with calcareous change at both apices and miliary tubercles throughout both lungs: œdema of glottis with tuberculous ulceration of larynx; miliary tubercles of trachea; fatty liver.

838 (Howard)—Male, 52. June 23, 1886.

ACUTE LOBAR PNEUMONIA AND CASEOUS PNEUMONIA. Bifid rib left side; yellow caseous tubercles of lungs; small cavity at apex; gray hepatization and caseous pneumonia; fatty liver.

839 (Howard)—Child, 1.

CAPILLARY BRONCHITIS. Atelectasis; fatty liver; rachitis; increase of cerebro-spinal fluid; dilatation of ventricles.

840 (Howard)—Male, 20. July 1, 1885,

RETROPHARYNGEAL ABSCESS. Syphilis; suppurating sinuses of neck; amyloid liver and kidneys.

840 A (Howard)—Male, 29. July 7, 1885.

RECURRENT OSTEOSARCOMA of left leg amputated recently; enlargement of retroperitoneal glands on left side; secondary osteosarcoma of right pleura and lung in form of discs of part of ossified sarcomatous tissue; secondary sarcoma of left lung and pericardium; abdominal viscera free from secondary enlargement.

841 (Howard)—Male, 55. July 2, 1885.

CHRONIC INTERSTITIAL NEPHRITIS.

842 (Howard)—Male. July 9, 1885.

CEREBRAL SOFTENING.

843 (Howard)—Female, 52. July 9, 1885.

STRICTURE OF RECTUM. Peritonitis from perforation of rectum by a bougie; Fibrous cicatrix of rectum; microscopically only fibrous tissue in growth.

844 (Howard)—Male.

FRACTURE OF BASE OF SKULL. Slight intermeningeal hæmorrhage.

844 A (Howard)—Female.

CIRRHOTIC KIDNEY. Atheroma and calcification about heart valves; brown atrophy; fracture of left radius.

845 (Howard)—Male. Died after dilatation of urethra.

STRICTURE OF URETHRA with production passage, lined with smooth gray membrane.

846 (Finley)—Female, 46. July 20, 1885.

PYOSALPYNX. Acute purulent peritonitis; abscess of L. ovary; granular contracted kidney; ulceration and stenosis of rectum.

847 (Finley)—Male.

CIRRHOTIC KIDNEY and amyloid kidney; amyloid liver and spleen.

848 (Finley)—Male.

GLANDERS. Farcy buds of skin.

849 (Finley)—Male, 25. July 19, 1885.

SLOUGHING OF RECTUM.

850 (Finley)—Female, 16. July 16, 1885.

UTERINE SEPSIS. Septichaemia.

851 (Finley)

DIAPHRAGMIATIC HERNIA. From old wound by ribs.

851 A (Finley)—Male, 32. Sept. 29, 1885.

TYPHOID FEVER. Pustular eruption; interstitial nephritis; large spleen.

851 B (Johnston)—Male, 20.

GUNSHOT WOUNDS OF ABDOMEN. Perforation of bowels by Snider bullet at short range; fracture of pelvis; laceration of right iliac veins; broad flat end of second right rib, not bifid.

852 (Johnston)—Male, 46. Oct. 9, 1885.

RECURRENT AORTIC ENDOCARDITIS and incompetency. Atheroma and dilatation of aorta; infarcts in spleen and right lung; chronic interstitial nephritis; embolism of left anterior cerebral artery; ankylosis of right ankle; nutmeg liver.

853 (Johnston)—Male, 29. Oct. 9, 1885.

TYPHOID FEVER. Ulceration of intestines in state of healing; perforation; peritonitis; hypostatic pneumonia; spleen 120 grm.

854 (Johnston)—Private.

DOUBLE FIBRO-CYSTIC SARCOMA of ovary. Secondary sarcoma in peritoneum with adhesive peritonitis, and in glands of portal fissure; jaundice; icterus and cloudy swelling of kidneys with slight hydronephrosis.

855 (Johnston)—Male, 60.

FATTY AND CIRRHOTIC LIVER. Cholæmia (?) jaundice; cholelithiasis.

856 (Johnston)—Male, 21.

SEPTICÆMIA. Poisoning by chlorate of potassium? Suppuration of deep inguinal gland; methæmoglobinæmia; acute parenchymotons nephritis; infarct of spleen. examination of coverslips for bacteria negative; prolongation of left lobe of liver.

857 Details not entered.

858 (Johnston)—Male, 18. Oct. 17, 1885.

TYPHOID ULCERATION of small intestine; enlargement of spleen; croupous pneumonia of right upper lobe and broncho-pneumonia of both lower lobes.

859 (Johnston)—Male, 54. Oct. 17, 1885.

CHRONIC PHTHISIS WITH CAVITY. Tuberculous pleurisy and pericarditis; phthisis with cavity at left apex; fibroid bronchitis and peri-bronchitis; chronic diffuse tuberculous pneumonia of left lower lobe; infarcts of spleen and kidney; tuberculous pyelitis and cystitis; stricture of urethra.

860 (Johnston)—Female, 68. Private. Oct. 19, 1885.

INTESTINAL OBSTRUCTION. Gangrene of bowel, localized peritonitis; obstruction caused by a coil of intestines becoming adherent to a large myoma of the uterus, and twisting round it; slight interstitial nephritis.

861 (Johnston)—Male, 60. Oct. 23, 1885.

PERFORATIVE APPENDICITIS. Perityphlitis from perforation of appendix; local and general septic peritonitis; distension of intestines; hyperæmia of lungs; thickening of aortic and mitral valves.

862 (Johnston)—Male, 25. Oct. 23, 1885.

TYPHOID FEVER. Intestinal hæmorrhage; typhoid ulceration of intestines including the rectum.

863 (Johnston)—Male, 42. Oct. 24, 1885.

ANÆMIA. Bronzing of skin of abdomen; chronic enlargement of liver and spleen; hyperplasia of bone marrow; atheroma of aorta; slight fatty degeneration of heart; cicatrices of colon apparently from former dysentery.

864 (Johnston)—Female, 46. Oct. 29, 1885.

CHRONIC INTERSTITIAL NEPHRITIS. Chronic gastrioenteric catarrh; acute general peritonitis; polypus uteri; atrophy of suprarenals; fibroid pleurisy with calcification; hyperplasia of spleen; slight mitral and aortic endocarditis.

865 (Johnston)—Male, 26. Oct. 28, 1885.

ADDISON'S DISEASE. Bronzing of skin for some months; diarrhœa; caseous tubercles in both suprarenals; enlargement of Peyer's patches and solitary follicles; caseous bronchial glands; universal adhesive pericarditis; slight broncho-pneumonia; caseous puckerings at apices of both lungs.

866 (Johnston)—Female, 18. Nov. 1, 1885.

THROMBOSIS. Internal saphenous vein and iliac vein; pulmonary embolism (cause of death.) Apical miliary tuberculosis; anæmia.

868 (Johnston)—Child, 5 days old.

INTESTINAL HÆMORRHAGE. Malæna; source not discovered; icterus; urate infarcts in kidneys.

869 (Johnston)—Male. Private. Nov. 1, 1885.

SCIRRHUS CANCER OF RIGHT KIDNEY. Infiltrating retroperitoneal glands; secondary in omentum; obliterative adhesive appendicitis and perityphlitis.

870 (Johnston)—Female, 29. Nov. 4, 1885.

PULMONARY TUBERCULOSIS. Cavities at each apex; tuberculous peribronchitis and pleurisy; miliary tubercles in liver spleen and kidneys; tuberculous ulceration of intestines.

871 (Johnston)—Male, 38. Nov. 5, 1885.

LACERATION OF ABDOMEN involving spleen, pancreas and left kidney; Hæmorrhagic infarction of lower lobe of left lung; fracture of ribs.

872 (Johnston)—Male, 33. Nov. 6, 1885.

FRACTURE OF BASE OF SKULL through frontal and sphenoid; extra meningeal hæmorrhage; broncho-pneumonia; Meckel's dverticulum; fusion of lobes of right lung; hypertrophy of heart; thickening of mitral valve. Shipping accident.

874 (Johnston)—Male, 48. Nov, 9, 1885.

CHRONIC TUBERCULOSIS OF BOTH LUNGS. Chronic tuberculous pleuritis; total synechia of pericardium; caseous tubercle of liver; caseation and fibrosis of suprarenals; catarrhal enteritis; milky opacity of pia mater.

876 (Johnston)—Male, 44. Nov. 10, 1885.

CHRONIC PARENCHYMATOUS NEPHRITIS. Commencing interstitial nephritis; fibroid and suppurative orchitis; localized adhesive peritonitis; œdema; ascites; anasarca; deformity of liver; fibrinous pleurisy.

877 (Johnston)—Male, 44. Nov. 12, 1885.

FRACTURE OF SKULL through left temporal, sphenoid and frontal bones; intermeningeal hæmorrhage; fragilitas ossium.

878 (Johnston)—Female, 20. Nov. 23, 1885.

TYPHOID FEVER. Perforation of intestines; hyperæmia and œdema of lungs; acute septic peritonitis.

879 (Johnston)—Female, 37. Nov. 23, 1885.

PHTHISIS. Amputation of left foot for caries; general miliary tuberculosis.

880 (Johnston)—Male. Dec. 4. 1885.

PHTHISIS WITH CAVITIES in both lungs. Thrombus of internal saphena vein, old fracture of second lumbar vertebra.

881 (Johnston)—Female, 25. Dec. 1, 1885.

THROMBOSIS OF CORONARY ARTERY. Thrombosis of left saphena vein; mural thrombus of heart; tertiary syphilis.

882 (Johnston)—Male, 54. Dec. 8, 1885.

PHTHISIS. Large cavities of lungs; tuberculous ulceration of intestines; miliary tubercles of kidneys.

884 (Johnston)—Female, 71. December 9, 1885.

ŒDEMA OF PIA. Degeneration of 5th nerve.

885 (Sutherland)—Male, 35. Dec. 8, 1885.

SYPHILITIC ENDARTERITIS of cerebral arteries: localized meningitis; chronic interstitial nephritis; apical tubercles cicatrised: Meckel's diverticulum; hyperplasia of spleen.

886 (Sutherland)—Male, 45. Dec. 10, 1885.

GONORRHEAL ARTHRITIS. Excision of knee joint; (pyaemia?); chronic adhesive peritonitis; apical nodules in lungs.

887 (Sutherland)—Male, 28. Dec. 12, 1885.

TYPHOID FEVER. Perforation of typhoid ulcer of ileum; acute hyperplasia of spleen; cloudy swelling of kidneys; broncho-pneumonia.

1886

1 (Johnston)—Male, 36. Jan. 13, 1886.

RECURRENT ENDOCARDITIS. Hypertrophy and dilatation of heart; brown induration of lungs; induration of spleen and kidneys; mesenteric glands enlarged and firm; pericardial adhesions.

2 (Johnston)—Female, 10. Jan. 4, 1886.

TYPHOID FEVER. Ulceration of intestines; fatty degeneration of heart; thrombosis of left ventricle; infarct of R kidney; spleen normal; healing typhoid ulcers in intestines; enlarged mesentric glands.

3 (Johnston)—Male, 30. Jan. 6, 1886.

ACUTE MANIA. Hyperæmia and thickening of pia; subarachnoid œdema; other organs normal.

4 (Johnston)—Male, 17. Jan. 13, 1886.

CHRONIC PHTHISIS with cavity. Slight amyloid in kidneys and spleen.

5 (Johnston)—Male, 61. Jan. 22, 1886.

CHRONIC VALVULAR HEART DISEASE. Dilatation and fatty degeneration of heart; sclerotic endocarditis of mitral and aortic valves; hydrothorax; right pleuritis: brown induration of lungs; collapse right lung; indurative hyperplasia of spleen and kidneys; cirrhosis of liver: hypertrophy of prostate; anasarca; icterus.

6 (Johnston)—Male, 38. Jan. 26, 1886.

EPITHELIOMA OF ŒSOPHAGUS. Secondary in lungs; bougie passed into lung? gangrene of lungs; pleurisy; chronic fibrous; perihepatitis.

7 (Johnston)—Male, 80. Jan. 28, 1886.

CHRONIC INTERSTITIAL nephritis. Arteriosclerosis; hypertrophy and dilatation of left ventricle; fusion and sclerosis of aortic and mitral valves; hypertrophy of prostate; chronic catarrhal gastritis.

8 (Johnston)—Male, 43. Jan. 31, 1886.

ACUTE ALCOHOLISM? Cloudy swelling of heart, liver and kidneys; hyperæmia of brain and lungs.

9 (Johnston)—Male, 63. Feb. 5, 1886.

SUPPURATIVE PYONEPHROSIS. Ischiorectal abscess; hypertrophy of prostate; ulcerative cystitis; emphysema; septic embolic pneumonia; anomalies of ascending colon and liver; obliterative phlebitis of hepatic veins with atrophy of left lobe of liver; atheroma of aortic valves.

10 (Johnston)—Infant.

SUDDEN DETAH. Cause not made out; organs appear healthy.

11 (Johnston)—Female, 43. Feb. 13, 1886.

CEREBRAL THROMBOSIS right middle cerebral artery. Sclerotic endocarditis and stenosis of mitral, tricuspid and aortic valves, with fusion of coronary segments; hypertrophy and dilatation of heart; emphysema; infarcts of spleen; nutmeg liver; chronic adhesive pelvic cellulitis and peritonitis; right pyelonephritis from uric acid calculus.

12 (Johnston)—Male, 6. Feb. 13, 1886.

ACUTE TUBERCULOUS MENINGITIS. Meningeal hæmorrhage; acute miliary tuberculosis of both lungs, with caseous bronchial glands; miliary tuberculosis of liver, spleen and kidneys; localized tuberculous pleuritis and peritonitis; tuberculous follicular ulceration of intestines.

13 (Johnston)—Private, Male, 7 months. Feb. 22, 1886.

ASPHYXIA FROM FOOD in air passages.

14 (Johnston)—Male, 40. March 4, 1886.

SUICIDE BY REVOLVER WOUND OF SKULL. Laceration of frontal lobes; meningeal hæmorrhage; acute tuberculous pleurisy; phthisis of both lungs; hypostatic pneumonia; distended gall bladder; no obstruction.

15 (Johnston)—Female, 75. Feb. 28, 1886.

THROMBOSIS OF CORONARY ARTERY. Myomalacia cordis; acute aneurysm of left ventricle; rupture into pericardium; atheroma and aneurysm of abdominal aorta; lacing deformity of liver and intestines; chronic suppurative arthritis right elbow; cellulitis right arm; dextro position of rectum.

16 (Johnston)—Female, 40. Feb. 14, 1886.

CANCER OF STOMACH at pylorus, with stenosis and gastrectasia.

17 (Johnston)—Male, 53. March, 7, 1886.

CALCULI OF BLADDER. Lithotomy incision and sinus in perineum; suppurative pyelitis with calculus: hypertrophy of prostate; chronic cystitis with sacculation and calculi of bladder; emphysema; broncho-pneumonia; perisplenitis; stenosing phlebitis of R. renal vein.

18 (Johnston)—Female, 20. March 9, 1886.

PUERPERAL SEPTICÆMIA. Subinvolution; puerperal state; acute purulent peritonitis; hæmorrhagic diphtheritic endometritis; acute pleurisy, with effusion; cloudy swelling of liver and kidneys; hyperplasia of spleen.

19 (Johnston)—Male, 65. March 15, 1886.

FRACTURE OF SKULL. Meningeal hæmorrhage; lateral dislocation of axis; diverticulum of bladder; hyperæmia of lungs.

20 (Johnston)—Male, 34. March 19, 1886.

SUPPURATIVE APPENDICITIS with perforation; pylephlebitis and hepatic abscess.

21 (Johnston)—Male, 25. March 27, 1886.

CHRONIC VALVULAR HEART DISEASE. Sclerotic and verrucose mitral endocarditis; pericardial synechiæ; hypertrophy of heart. (Partial autopsy.)

22 (Johnston)—Male, 21. March 13, 1886.

STRANGULATED R. INGUINAL HERNIA (reduced). Localized septic peritonitis; necrosis of intestine and strangulation from constriction of loop of bowel by old adhesions, (loop 18″ long); left inguinal hernia.

23 (Johnston)—Male, 47. April 5, 1886.

ULCERATIVE ENDOCARDITIS of aortic valves; hypertrophy and dilatation of heart; acute pericarditis (local); acute pneumonia; infarcts of lung; acute fibrinopurulent (septic) meningitis.

24 (Johnston)—Male, 41. April 6, 1886.

HYPERTROPHY AND DILATATION OF HEART. Chronic sclerotic aortic endocarditis; dilatation and atheroma of aorta; obsolete caseous apical tubercles; scar of glans.

25 (Johnston)—Male, 33. April 12, 1886.

CRUSH OF CHEST. Dislocation of 1st costal, cartilage; ecchymosis of pericardium; double pneumonia; acute right pleurisy.

26 (Johnston)—Male, 28. April 14, 1886.

ACUTE LOBAR PNEUMONIA. Follicular ulcers of colon.

27 (Johnston)—Male, 54. April 17, 1886.

CARCINOMA OF PANCREAS, secondary in liver, lungs, kidneys and duodenum; gall-stones; intestinal hæmorrhage; cirrhotic kidneys; old ulcer of leg.

29 (Johnston)—Private. April 26, 1886.

GLIOSARCOMA OF PITUITARY BODY. Atrophy of optic nerves; chronic hydrocephalus.

28 (Johnston)—Private. April 25, 1886.

TUBERCULOUS PYONEPHROSIS of right kidney and ureter; tuberculous cystitis; tuberculous right suprarenal; tuberculous vesicles; pulmonary phthisis.

30 (Johnston)—Female. Private. April 26, 1886.

PULMONARY EMBOLISM FROM UTERINE THROMBOSIS. Subinvolution; puerperal state; thrombosis of veins of uterine plexus, pelvic and iliac veins and inferior vena cava; pulmonary embolism; septic endocarditis; local septic pelvic peritonitis; acute hyperplasia of spleen; cloudy swelling of heart, liver and kidneys.

31 (Johnston)—Female, 22. April 27, 1886.

PUERPERAL SEPTICÆMIA. Mycotic aortic endocarditis; streptococcus infection; infarcts of spleen and kidneys: cloudy swelling of liver and kidneys; acute hyperplasia of spleen.

33 (Johnston)—Male, 6. April 30, 1886.

ACUTE CEREBRO SPINAL MENINGITIS (non-tuberculous).

34 (Johnston)—Male, 32. May 1, 1886.

PULMONARY PHTHISIS. Chronic tuberculous pleuritis; tuberculous ulceration of intestine; chronic lepto-meningitis with granulomata of ependyma; chronic parenchymatous and interstitial nephritis.

35 (Johnston)—Female, 20. May 3, 1886.

TUBERCULOUS SALPINGITIS. General acute purulent tuberculous peritonitis and pleuritis; milk patch of pericardium; tuberculosis of lungs and kidneys.

36 (Johnston)—Female, 26. May 4, 1886.

ACUTE TUBERCULOSIS OF LUNGS. Hyaline degeneration of spleen, liver and kidneys; fistula in ano and hæmorrhoids; tuberculous ulcers of duodenum, jejunum and cæcum; chronic purulent otitis media; bedsores; dilatation of heart: sclerotic endocarditis of tricuspid valve and recurrent mitral endocarditis.

37 (Johnston)—Male, 28. May 5, 1886.

CHRONIC PARENCHYMATOUS NEPHRITIS. Fatty and cirrhotic liver; alcoholism; atheroma of aorta; hyperæmia of lungs; anasarca; ascites; jaundice.

38 (Johnston)—Male, 26. May 7, 1886.

PULMONARY PHTHISIS, large cavities in each lung; tuberculous ulceration through whole length of intestine; tuberculous kidney; polypoid adenoma of duodenum; tubercles in suprarenals; verrucose endocarditis; tuberculosis of mesenteric glands.

39 (Johnston)—Female, 19. May 7, 1886.

TUBERCULOUS PHTHISIS OF LUNGS, large cavities; amyloid spleen, liver and intestines; concretion in vermiform appendix; tuberculous ulcers of larynx and ascending colon.

40 (Johnston)—Private.

DELIRIUM TREMENS, ALCOHOLISM. Subarachnoid œdema of brain; no meningitis. (Other organs not examined).

41 (Johnston)—Male, 50. May 10, 1886.

TUBERCULOSIS OF LUNGS. Tuberculous pleurisy; tuberculous ulceration of intestines, with perforation; peritonitis; tubercle of R adrenal; leucoderma; inguinal hernia; tubercles of kidneys.

42 (Johnston)—Male, 40. May 14, 1886.

CHRONIC ULCERATIVE PHTHISIS of lungs, larynx and intestines; tuberculosis of suprarenals; amyloid liver, spleen, kidneys and intestines; sclerotic mitral and verrucose aortic endocarditis; dilatation of tricuspid.

43 (Johnston)—Female, 50. May 15, 1886.

EMBOLISM R. MIDDLE CEREBRAL artery and softening of right corpus striatum, central lobe and internal capsule; sclerotic mitral and verrucose aortic endocarditis; chronic parenchymatous nephritis; infarction of kidney; amyloid (bacon) spleen; subperitoneal fibromyoma of uterus; amputation of right breast (one year previous); secondary cancer of left lung.

44 (Kirkpatrick)—Male, 45. May 17, 1886.

ACUTE LOBAR PNEUMONIA both lower lobes; acute double pleurisy; atheroma of aortic valves, with fusion; jaundice.

45 (Johnston)—Female, 12. May 18, 1886.

DIPHTHERIA of larynx, pharynx and trachea; tracheotomy; cellulitis of neck.

46 (Howard)—Male, 65. May 20, 1886.

ACUTE PLEURISY; collapse and œdema of lungs; atheroma of mitral and aortic valves; calcified apical tuberculosis.

47 (Howard)—Female, 42. May 23, 1886.

GRANULAR KIDNEYS. Hæmorrhage right corpus striatum, hypertrophy of heart; arteriosclerosis; acute pleurisy and pneumonia; retroversion of uterus; rectum on right side.

48 (Howard)—Male, 60. May 24, 1886.

LYMPHOSARCOMA? of right lung and pleura; nodules in kidneys, suprarenals and left lung.

49 (Howard)—Male, 45. June 1, 1886.

CHRONIC INTERSTITIAL NEPHRITIS. Hypertrophy of heart, with dilatation.

50 (Howard)—Female, 32. June 1, 1886.

TUBERCULOSIS OF LUNGS, WITH CAVITIES. Tuberculous peritonitis; fatty liver.

51 (Howard)—Male, 57. June 20, 1886.

UNDILATED STRICTURE OF URETHRA. Recent false passage; intense hyperæmia of lungs; kidneys enlarged and congested.

52 (Howard)—Female, 31. June 20, 1886.

CHRONIC PHTHISIS. Tuberculous ulceration of larynx, lungs and intestines; periurethral abscess; adhesive peritonitis.

53 (Howard)—Male, 36. June 26, 1886.

INTESTINAL OBSTRUCTION. Alcoholism; jaundice.

54 (Howard)—Male, 34. Sept. 23, 1886.

HÆMORRHAGE RIGHT SIDE OF BRAIN. Hæmorrhagic infarcts in lungs; Subpericardial and subcutaneous ecchymosis.

55 (Johnston)—Male, 22. Oct. 21, 1886.

TYPHOID FEVER. Ulceration of intestines in late stage; hyperplasia of spleen; slight parenchymatous degeneration of liver and kidneys; hæmorrhagic pericarditis (with miliary tuberculosis).

56 (Johnston)—Male, 52. Oct. 26, 1886.

COLLOID CANCER OF RECTUM. Secondary in inguinal and retroperitoneal glands and in lung; chronic and acute tuberculosis of lungs.

57 (Johnston)—Female, 42. Oct. 27, 1886.

CRIMINAL ABORTION. Traumatic septic endometritis; subinvolution; status puerperalis; infarction and septic pneumonia of lungs; acute fibrinous pleurisy; hyperplasia of spleen; cloudy swelling of liver, kidneys and heart; suppurative parotitis.

58 (Johnston)—Male, 35. Oct. 30, 1886.

FRACTURE OF PELVIS. Derrick accident. Laceration of urethra; urinary infiltration and phlegmonous cystitis; fracture of ribs: acute pleurisy; septicæmia.

59 (Johnston)—Female, infant.

SEPTICHÆMIA? Petechial rash. Nothing special in appearance of organs.

60 (Johnston)—Male, 33. Nov. 1, 1886.

INTESTINAL OBSTRUCTION. Adhesive local peritonitis; appendix attached to kink of ileum; acute gastro-enteritis; gall-stones.

61 (Johnston)—Female, 8 days.

SEPTICÆMIA. Child of leukaemic mother; gastroenteritis; purpura.

62 (Johnston)—Male, 43. Nov. 1, 1886.

CEREBRO SPINAL MENINGITIS? Broncho pneumonia; chronic catarrhal gastritis; chronic interstitial nephritis; cirrhosis of liver.

63 (Johnston)—Female, 50. Nov. 10, 1886.

HYPERTROPHY AND DILATATION OF HEART, non-valvular; anasarca; hydrothorax; hydropericardium; ascites; chronic interstitial nephritis; cyst of kidney; lacing lobe of liver; chronic pelvic adhesive peritonitis; arteriosclerosis; Meckel's diverticulum.

64 (Johnston)—Private. Nov. 12, 1886.

CHOLELITHIASIS. Perforation of gall bladder; gallstone obstructing common bile duct; jaundice; chronic local peritonitis; acute general septic peritonitis.

65 (Johnston)—Male, 27. Nov. 12, 1886.

FRACTURE OF SKULL. Derrick accident. Laceration and contusion of scalp; depressed fracture of L. parietal bone; rupture of left middle meningeal artery; inter and extra meningeal hæmorrhage; orbital hæmorrhage; œdema of lungs.

66 (Johnston)—Private.

CANCER OF STOMACH, with ulceration; secondary cancer of pancreas and retroperitoneal glands; secondary cancer of right lung and liver; peritonitis; cancerous pleurisy.

67 (Johnston)—Male, 45. Nov. 16, 1886.

EPITHELIOMA OF ŒSOPHAGUS. Secondary in bronchial glands and left bronchus.

68 (Johnston)—Female, 74. Nov. 16, 1884.

BRONCHO-PNEUMONIA. Fibroid pleurisy; chronic endocarditis; gall stone; abscess of brain.

69 (Johnston)—Female, 38. Nov. 18, 1886.

ALCOHOLISM; PERIPHERAL NEURITIS.

70 (Johnston)—Male, 22. Nov. 20, 1886.

TUBERCULOUS PYELITIS. Old right lateral lithotomy and cystitis; perineal fistula; caseous prostatitis and epididymitis; acute tuberculosis of both lungs; chronic left pleurisy; amyloid spleen, liver, kidneys and intestines.

71 (Johnston)—Female, 29. Nov. 25, 1886.

CHRONIC MYELITIS. Slaty pigmentation of follicles in jejunum; cystic ovary.

72 (Johnston)—Male, 52. Nov. 29, 1886.

SCIRRHOUS CANCER OF PYLORUS. Dilatation of stomach.

73 (Johnston)—Male, 41. Nov. 30, 1886.

NON-VALVULAR DILATATION AND HYPERTROPHY of heart. Slight fatty change of heart muscle; acute miliary tuberculosis of lungs, liver and kidneys; tuberculous endocarditis; tuberculous ulceration of colon.

74 (Johnston)—Female, 20. Nov. 30, 1886.

EMBOLISM OF LEFT SYLVIAN ARTERY. Verrucose endocarditis of mitral; white softening of brain; acute parenchymatous nephritis.

75 (Johnston)—Male, 38. Nov. 30, 1886.

CHRONIC PULMONARY PHTHISIS, cavity left lung; chronic caseous peribronchitis; tuberculous right pleurisy; tuberculous ulcers of intestine.

76 (Johnston)—Private.

CANCER OF PYLORUS. Dilatation of stomach.

78 (Johnston)—Female, 40. Dec. 6, 1886.

CEREBRAL SOFTENING and calcareous change in brain; calcified nodule in pons; chronic myelitis.

79 (Johnston)—Male, 24. Dec. 6, 1886.

CHRONIC TUBERCULOSIS OF LUNGS and intestine. Local tuberculous peritonitis; amyloid spleen, liver and kidneys.

80 (Johnston)—Male, 26. Dec. 7, 1886.

ACUTE MENINGITIS and chronic lepto-meningitis; hyperæmia of lungs and kidneys.

81 (Johnston)—Male, 54. Dec. 10, 1886.

ACUTE LOBAR PNEUMONIA, with interstitial pneumonia. Subacute pericarditis and pleurisy; cysts in kidneys; hypertrophy of middle lobe of prostate.

82 (Johnston)—Male, 19. Dec. 12, 1886.

TYPHOID FEVER. DIPHTHERIA. Ulceration of intestines; acute hyperplasia of mesenteric glands and spleen; diphtheria of pharynx, larynx and trachea; broncho-pneumonia of upper lobe of left lung; atrophy of left inner temporal convolution.

83 (Johnston)—Male, 41.

DIFFUSE ANEURYSM OF INNOMINATE ARTERY. Erosion of sternum; obliteration of superior vena cava, with dilatation of veins; pressure on right vagus; broncho-pneumonia.

84 (Johnston)—Female, 32. Dec. 23, 1886.

PHTHISIS. Cavities in both lungs; amyloid spleen, liver, kidneys and intestines; acute parenchymatous nephritis.

85 (Johnston)—Male, 50. Dec. 18, 1886.

SUPPURATIVE NEPHRITIS. Enlarged prostate and abscess of prostate; hyperplasia of spleen; pyæmia.

86 (Johnston)—Male 45. Dec. 16, 1886.

ANEURYSM OF AORTA.

87 (Johnston)—Male, 42. Dec. 31, 1886.

FRACTURE AND DISLOCATION OF DORSAL VERTEBRA.

1887

1 (Johnston)—Femals, 50. Jan. 14, 1887.

RECURRENT CARCINOMA OF MAMMA. Cancerous thrombus in left subclavian vein; secondary cancer in lungs and liver; chronic hydrocephalus internus; chronic mitral endocarditis.

2 (Johnston)—Female, 19. Jan. 20, 1887. Maternity.

PYÆMIA. Acute peritonitis and pleurisy: puerperal state; subinvolution; diphtheritic endometritis; streptococcus infection; purulent arthritis of wrist; cloudy swelling of liver and spleen; acute hyperplasia of spleen; catarrhal enteritis; follicular tonsilitis.

3 (Johnston)—Female, 11. Jan. 26, 1887. Dr. Ross.

ACUTE TUBERCULOUS MENINGITIS. Solitary tubercle of cerebellum; sub-acute tuberculous peritonitis; slight miliary tuberculosis of lungs, liver and kidneys; double optic neuritis; anchylosis of ankle; ovarian cysts; miliary tubercles of intestines.

4 (Johnston)—Male, 22. Jan. 26, 1887. Dr. Roddick.

COMPOUND COMMINUTED FRACTURE THROUGH LEFT KNEE. Fracture of R. Femur, tibia and fibula, R. ulna and radius; prolongation left lobe of liver; lobulation of kidneys; atheroma of mitral valve; enlarged solitary follicles of ileum.

5 (Johnston)—Male, 28. Jan. 26, 1887. Drs. Ross and Bell.

MILIARY TUBERCULOSIS of lungs, liver, spleen and kidneys. Apical tubercle (caseous); right tuberculous pyelitis; tuberculous urethro perineal rectal fistula; R. tuberculous epididymitis; ablation left testicle; tuberclosis of suprarenals; bedsores; acute tuberculous meningitis.

6 (Johnston)—Female, 52. Jan. 27, 1887. Dr. Molson.

ACUTE PLEURISY, WITH BRONCHITIS AND CONGESTION OF BOTH LUNGS. Parenchymatous and chronic interstitial nephritis; fibromata of kidneys; atheroma of aorta; pulmonary apoplexy.

7 (Johnston)—Female, 22. Private.

PERIPHERAL NEURITIS. Caseous area in lower lobe of left lung.

8 (Johnston)—Male, 38. Feb. 11, 1887. Dr. Bell.

CARCINOMA OF STOMACH, secondary in omentum retroperitoneal glands and liver; cancerous degeneration of marrow of ribs and sternum, caries of 1st and 6th dorsal vertebræ.

9 (Johnston)—Female, 70. Feb. 14, 1887. Dr. Molson.

BRONCHO-PNEUMONIA. Atheroma of coronary arteries and general arteriosclerosis nodosa, hypertrophy of left ventricle; fatty degeneration of heart; chronic bronchitis; granular kidneys.

10 (Johnston)—Male. 26. Feb. 15, 1887.

DOUBLE CALCULOUS PYELITIS. Nephrolithotomy; obliteration of L. ureter; acute swelling of retro-abdominal lymph glands; icterus.

11 (Johnston)—Male, 20. Feb. 17, 1887. Dr. Ross.

PHTHISIS, WITH CAVITIES IN BOTH LUNGS. Chronic adhesive pleuritis; tuberculous ulceration of ileum; appendix dilated, 10 c.m. in circumference.

12 (Johnston)—Male, 65. Feb. 17, 1887.

CHRONIC TUBERCULOSIS OF LUNGS. Brown atrophy of heart; dilatation of aorta, with atheroma; brown atrophy of liver; softening of brain; tuberculosis of thoracic duct.

13 (Johnston)—Female, 25. Feb. 20, 1887. Maternity.

ACUTE SEPTIC ENDOMETRITIS AND PERIMETRITIS. Puerperal fever; laparotomy; acute septic pleurisy; cyst of broad ligament; cloudy swelling of heart, liver and kidneys; acute hyperplasia of spleen; streptococcus infection.

14 (Johnston)—Female, 65. Feb. 24, 1887. Private.

DISSEMINATED ATROPHY OF CORPORA STRATIA WITH CYSTS.

15 (Johnston)—Female, 40. Feb. 24, 1887. Maternity.

PUERPERAL SEPTICÆMIA. Streptococcus infection; diphtheritic endometritis; phlegmonous parametritis and perimetritis; acute sero-purulent peritonitis; cloudy swelling of liver and kidneys; hyperplasia of spleen.

16 (Johnston)—Male, 22. Feb. 27, 1887. Dr. Molson.

CHRONIC TUBERCULOSIS OF LUNGS WITH CAVITIES. Tuberculous ulceration of larynx and trachea, small intestine and rectum: enlargement of mesenteric glands: brown atrophy of liver; pediculi capitis and pubis.

17 (Johnston)—Child, 3 months. Feb. 28, 1887. Private.

ŒDEMA OF GLOTTIS. Sudden death. Hyperæmia of lungs; subacute laryngitis and bronchitis; rachitis; anæmia; anasarca; syphilis?

18 (Johnston)—Fœtus. Feb. 10, 1887. Private.

HYDRAMNIOS. Organs normal.

19 (Johnston)—Female, 20. Maternity.

PUERPERAL SEPTICÆMIA. Lactation; diphtheritic endometritis; acute septic peritonitis and pleurisy; chronic ovaritis fibrosa; acute hyperplasia of spleen; pharyngeal diphtheria; streptococcus infection.

20 (Johnston)—Female, 50. March 14, 1887. Dr. Bell.

WOUND OF NECK, PERFORATING LEFT PLEURA. Compound fracture of sternum and both first ribs; acute sero-fibrinous hæmorrhagic pleurisy; acute pericarditis; compression of R. sub-clavian artery; fatty liver.

21 (Johnston)—Male, 60.

FRACTURE OF RIGHT RIBS, 1ST TO 9TH, AND LEFT, 1ST TO 3RD; HÆMOTHORAX. Emphysema: chronic productive pachy-and lepto-meningitis; R. inguinial hernia; double hydrocele; Meckel's diverticulum. (Coroner's case. Verdict: Death from natural causes!)

22 (Johnston)—Male, 50. March 15, 1887. Private.

CEREBRAL HÆMORRHAGE RIGHT CORPUS STRIATUM; HÆMORRHAGE INTO VENTRICLES. Alcoholic. (Only brain examined).

23 (Johnston)—March 22, 1887. Private.

GLIO-SARCOMA OF R. FRONTAL LOBE OF BRAIN. Pericardial and pleuritic synechiæ; arterio-sclerosis; catarrhal enteritis and gastritis; emphysema; hypostatic broncho-pneumonia; granular kidneys; parenchymatous nephritis.

25 (Johnston)—Male, 30. April 22, 1887. Dr. McDonnell.

CHRONIC PARENCHYMATOUS AND INTERSTITIAL NEPHRITIS. Anæmia; anasarca; ascites; chronic hyperplasia of spleen; gastro-enteritis; fatty heart; slight acute pericarditis.

26 (Johnston)—Female, 17. April 26, 1887. Dr. Wilkins.

NON-TUBERCULOUS ACUTE BASILAR MENINGITIS.

27 (Johnston)—Male, 7. April 28, 1887.

FRACTURE OF SKULL, temporal and frontal bones; trephining; purulent otitis and pachymengitis; abscess of brain; purulent otitis media.

28 (Johnston)—Male, 30. April 30, 1887. Dr. MacDonnell.

RENAL CALCULUS. Double hydronephrosis.

29 (Johnston)—Male. May 10, 1887. Dr. Wilkins.

CHRONIC SCLEROTIC AND ACUTE VERRUCOSE ENDOCARDITIS, WITH STENOSIS. Acute pleurisy, with effusion; localized empyæma: acute local peritonitis; acute pericarditis: ateleclasis left lung; infarct of spleen and R. kidney.

30 (Johnston)—Male, 55. May 10, 1887. Dr. Wilkins.

CHRONIC ULCERATIVE PHTHISIS, with acute miliary tuberculosis and emphysema; adhesive pleurisy; brown atrophy of heart.

31 (Johnston)—Female, 32. May 12, 1887. Dr. Wilkins.

CHRONIC PHTHISIS OF LUNGS.

31 a (Johnston)—Male, 17½ months. Private.

NASAL AND PHARYNGEAL DIPHTHERIA. Atelectasis R. lung; p. m. invagination of bowel.

32 (Johnston)—Male, 88. May 11, 1887. Dr. MacDonnell.

PYELONEPHROSIS AND HYDRONEPHROSIS. Hypertrophy of prostate; dilatation of bladder; depressed fracture of R. frontal bone (healed).

33 (Johnston)—Female, 12. Private.

PERFORATIVE APPENDICITIS. Pericæcal abscess; acute peritonitis; ascarides.

34 (Johnston)—Female, 35. May 12, 1887. Dr. Wilkins.

CHRONIC ULCERATIVE PHTHISIS. Tuberculous fibrous pleuritis; tubercular ulceration epiglottis; hypertrophy of right ventricle; brown atrophy of heart; nutmeg liver; tuberculous ulceration of intestines; amyloid spleen, liver, kidneys and intestines.

35 (Johnston)—Male, 53. May 16, 1887.

EPITHELIOMA OF ŒSOPHAGUS, infiltrating trachea; pressure on recurrent nerves; purulent bronchitis; chronic adhesive local pleurisy; calcerous bronchial glands; chronic mitral endocarditis: brown atrophy of heart; granular atrophic kidney.

36 (Johnston)—Male, 28. May 18, 1887.

PHLEGMONOUS CELLULITIS and lymphangitis L. arm. Universal adhesive R. pleuritis; multiple apoplexy of lungs; slight sclerotic mitral endocarditis; fatty liver; cloudy swelling of liver and kidneys: condylomata of penis.

37 (Johnston)—Male, 36. May 23, 1887.

TUBERCULOUS ULCERATIVE PHTHISIS. Chronic and acute tuberculous pleurisy; tuberculous pharyngitis? chronic adhesive pericarditis; fatty liver.

38 (Johnston)—Female, 30. May 26, 1887. Dr. Wilkins.

PHTHISIS, WITH CAVITY OF LEFT LUNG. Ochre degeneration of fat; R. pleurisy with effusion; acute broncho-pneumonia; œdema of lungs; abscess and fistula of vulva; hæmorrhoids; hæmorrhagic spot (tuberculous) on epiglottis.

39 (Johnston)—Male, 36. May 27, 1887. Dr. MacDonnell.

PULMONARY PHTHISIS, cavity in left upper lobe; tuberculous pleuritis; adhesive pericarditis; tuberculous peritonitis and enteritis; amyloid of spleen, liver, kidneys, stomach, intestines and bladder.

40 (Johnston)—Female, 17. Nay 27, 1887. Drs. Buller and MacDonnell.

CHRONIC INTERSTITIAL NEPHRITIS. Hypertrophy of heart; mitral stenosis; œdema of lungs, with pulmonary apoplexy; infarct of spleen; pigmentation of peritoneum; retinitis and bulbar neuritis.

41 (Johnston)—Female, 31. June 6, 1887.

RECURRENT SCLEROTIC AND VERRUCOSE ENDOCARDITIS WITH MITRAL STENOSIS. Parenchymatous myocarditis: subinvolution; chronic endometritis; infarcts of lung and kidney; gallstones; cirrhosis of liver infarcts of spleen; status puerperalis.

42 (Johnston)—Male, 40. June 13, 1887. Dr. MacDonnell.

ATHEROMA OF AORTA, DIFFUSE ANEURYSM OF TRANSVERSE ARCH. Dilatation and hypertrophy of heart; infarction of left lung; left hydrothorax; ulcer of R. ankle.

43 (Jonnston)—Male, 57. June 15, 1887. Dr. Wilkins.

CHRONIC PHTHISIS OF LUNGS.

44 (Johnston)—Male, 46. June 16, 1887. Private.

CHRONIC AORTIC AND MITRAL ENDOCARDITIS, WITH STENOSIS. Chronic adhesive peritonitis; induration of spleen and kidneys; cirrhosis of liver; ascites: anasarca; hydrothorax.

45 (Johnston)—Female, 26. June 18, 1887. Dr. Wilkins.

URAEMIC COMA; chronic pleuritis and pericarditis; hogback kidney; multiple angioma of liver; acute hyperplasia of spleen; R. hydrosalpinx; chronic adhesive pelvic peritonitis.

46 (Johnston)—Male, 5. June 18, 1887. Dr. MacDonnell.

DIPHTHERIA of pharynx, larynx and trachea; broncho-pneumonia.

47 (Johnston)—Female, 22. June 20, 1887. Dr. Wilkins.

PULMONARY PHTHISIS WITH CAVITIES. Amyloid of spleen, liver, kidneys and intestines; tuberculous laryngitis; round ulcer of stomach.

48 (Johnston)—Male, 33. June 22, 1887. Dr. Roddick.

FRACTURE OF CERVICAL VERTEBRÆ. Acute double pleurisy; broncho-pneumonia.

49 (Johnston)—Female, 78. June 26, 1887.

COMMINUTED FRACTURE OF RIGHT FEMUR. Chronic bronchitis with emphysema; fat infiltration of heart; senile kidneys; Liebermeister's furrows of liver; papilloma of bladder.

50 (Johnston)—Male, 22. Private.

APPENDICITIS, (perforative) with abscess: general purulent peritonitis; old adhesive perityphlitis and peritonitis; fæcal concretion in appendix.

51 (Johnston)—Male, 21. June 29, 1887. Dr. Wilkins.

PULMONARY PHTHISIS CAVITIES IN BOTH LUNGS. Emphysema and œdema; tuberculous laryngitis; tuberculous enteritis.

52 (Johnston)—Female, 26. June 29, 1887. Dr. Wilkins.

CHRONIC SUPPURATIVE PELVIC CELLULITIS. Subinvolution; thrombosis of pelvic and iliac veins and vena cava; hyperæmia and œdema of lungs.

53 (Johnston)—Male, 50. July 6, 1887. Dr. MacDonnell.

TYPHOID FEVER. Ulceration of small and large intestines: perforation; peritonitis.

54 (Johnston)—Female, 23. July 7, 1887. Maternity.

LACERATION OF LEFT URETER DURING LABOUR. Flat pelvis; fistula of ureter and abscess of left flank; diphtheritic vaginitis; acute pyonephrosis, severe on left side, slight on right. Forceps case.

55 (Johnston)—Male, 42. July 8, 1887. Dr. Roddick.

ACUTE TUBERCULOSIS OF BOTH LUNGS. L. empyæma; tuberculous abscess in liver, with fistula perforating sternum and leaving sinus.

56 (Lafleur)—Male, 23. July 9, 1887.

FRACTURE AND DISLOCATION OF 5TH CERVICAL VERTEBRA.

57 (Johnston)—Male, 34. July 11, 1887. Dr. Wilkins.

TYPHOID FEVER. Ulceration of bowel; bronchitis and emphysema; perichondritis; abscess in spleen.

58 (Johnston)—Male, 27. Aug. 8, 1887.

TYPHOID. Ulceration of ileum; acute hyperplasia of spleen; acute parenchymatous nephritis; p. m. invagination; cloudy swelling of kidneys.

60 (Johnston)—Female, 25. Sept. 6, 1887. Dr. MacDonnell.

ARSENICAL POISONING. Rough on rats. Round ulcer of stomach; chronic follicular enteritis; chronic adhesive peritonitis; perimetritis and salpyngitis.

61 (Johnston)—Male, 55. Sept. 14, 1887. Dr. Wilkins.

ATROPHIC CIRRHOSIS OF LIVER. Double pleuritic adhesions; chronic gastritis; apical cicatrices of lungs; icterus.

62 (Johnston)—Male, 30. Sept. 16, 1887. Dr. MacDonnell.

TYPHOID FEVER, with ulceration and diffuse catarrhal enteritis; acute localized peritonitis (no perforation); acute bronchitis; hyperplasia of spleen.

63 (Johnston)—Male, 43. Sept. 21, 1887. Dr. Wilkins.

CHRONIC INTERSTITIAL NEPHRITIS. Chronic hypertrophic catarrhal gastritis; hypertrophy of heart; Œdema of lungs; R. hydrothorax; anasarca; polypoid adenomata of rectum.

65 (Johnston)—Male, 65. Sept. 22, 1887. Dr. Fenwick.

FRACTURE OF FRONTAL AND SPHENOID BONES, BASE OF SKULL. Laceration and hæmorrhage (extra and inter meningeal) from middle meningeal artery; slight laceration of brain; atheroma of aorta; anchylosis of dorsal vertebræ.

66 (Johnston)—Male, 30. Sept. 22, 1887.

DIPHTHERIA of pharynx and fauces.

67 (Johnston)—Female, 52. Sept. 23, 1887. Dr. Wilkins.

MITRAL STENOSIS. Dilatation of left auricle; fatty degeneration of heart; emphysema; fibromyoma uteri; granular kidneys; infarct of kidney.

68 (Johnston)—Female, 27. Sept. 23, 1887.

TYPHOID FEVER. Acute hyperplasia of spleen; ulceration of intestines; apoplexy of lungs; acute hyperplasia of mesenteric glands; cloudy swelling of liver and kidneys; angioma of liver.

69 (Johnston)—Male, 23. Sept. 24, 1887. Dr. Wilkins.

CHRONIC PULMONARY PHTHISIS. Tuberculous; caseous bronchial glands; miliary tubercles of liver and kidneys.

70 (Johnston)—Male, 74. Sept. 24, 1887. Dr. Fenwick.

HYPERTROPHY OF PROSTATE. Hypertrophy of bladder; chronic catarrhal bronchitis; chronic gastritis; ascarides; icterus; (kidneys not dilated).

71 (Johnston)—Male, 24. Sept. 27, 1887. Dr; Wilkins.

TYPHOID FEVER. Ulceration and perforation of ileum; follicular typhoid ulcer of colon; acute peritonitis; acute hyperplasia of spleen; acute broncho-pneumonia; purulent otitis media.

72 (Johnston)—Male, 27. Sept. 27, 1887. Dr. Wilkins.

TYPHOID FEVER. Ulceration of ileum; hyperplasia of spleen and mesenteric glands; broncho-pneumonia.

73 (Johnston)—Male, 18. Sept. 30, 1887.

TYPHOID FEVER. Slight localized peritonitis; acute swelling of spleen and mesenteric glands; cloudy swelling of kidneys; fatty liver; adenoma of supra-renal; old tuberculous adenitis of glands of neck.

74 (Johnston)—Male, 74. Oct. 4, 1887.

PYÆMIA. ACUTE CELLULITIS and abscess of left leg. Purulent arthritis left ankle, toes and epiphysal end of ribs, chronic subacute verrucose endocarditis of mitral aortic and tricuspid valves. Atheroma of coronaries; chronic bronchitis; emphysema; red atrophy of liver; brown atrophy of heart; acute enlargement and thickening of capsule of spleen; slight interstitial nephritis; hæmorrhoids.

75 (Johnston)—Female, 32. Oct. 10, 1887. Dr. Ross.

ACUTE PERITONITIS and calcification of omentum; chronic endometritis; gallstones; endocarditis; old cicatrices from former ulcerations of colon.

76 (Johnston)—Female, 50. Oct. 10, 1887. Dr. MacDonnell.

CATARRHAL DYSENTERY with follicular ulcers. Healed typhoid ulcers of intestine. Previous typhoid fever, ending in recovery; persistent diarrhœa.

77 (Johnston)—Male, 35. Oct. 14, 1887. Dr. Ross.

CHRONIC PARENCHYMATOUS AND INTERSTITIAL NEPHRITIS. Chronic tuberculosis of lungs, with cavity; Œdema of glottis and right lung; incipient tuberculosis of ileum; anasarca; ascites.

78 (Johnston)—Male, 7. Oct. 14, 1887. Dr. Ross.

NASOPHARYNGEAL DIPHTHERIA.

79 (Johnston)—Male, 35. Oct. 24, 1887.

LEFT EMPYÆMA, with chronic adhesive pleuritis. Right localized pleurisy, with effusion; perforation of sternum by empyæma; tuberculous ulceration of colon; miliary tuberculosis of lungs, liver and kidneys; amyloid spleen; putrid peritonitis.

80 (Johnston)—Female, 27. Nov. 1, 1887.

ACUTE ARSENICAL POISONING: Rough on Rats; death in 7 hours. Chronic mitral endocarditis; chronic (syphilitic?) lymphomata of intestines; gallstones.

81 (Johnston)—Male, 30. Nov. 5, 1887. Dr. Shepherd.

ACUTE TUBERCULOUS MENINGITIS. Nephrotomy; tuberculous cystitis; right tuberculous pyonephrosis; tuberculous vesico rectal perineal fistula; tuberculous peritonitis; miliary tubercles in lungs, liver and kidneys; amyloid liver, spleen and kidneys.

82 (Johnston)—Male, 38. Nov. 10, 1887. Dr. MacDonnell.

CHROIC PULMONARY PHTHISIS; CAVITIES IN BOTH LUNGS. Miliary tuberculosis of right lung, with emphysema; acute and chronic tuberculous pleurisy; amyloid spleen, liver, intestines and kidneys; tuberculous ulceration of ileum, colon and rectum; submucous cyst of bladder; angioma of liver

83 (Lafleur)—Male, 26. Nov. 30, 1887. Dr. Wilkins.

CATARRHAL ULCERATIVE COLITIS.

84 (Johnston)—Male, 55. Dec. 1, 1887. Dr. Ross.

CHRONIC AORTIC ENDOCARDITIS WITH INCOMPETENCY. Dilatation and hypertrophy of both sides of heart; universal adhesive pericarditis; brown induration of lungs; cyanotic induration of kidneys; nutmeg liver; calcification of mesenteric glands and one solitary follicle; hydrothorax; œdema.

85 (Johnston)—Female, 10. Dec. 3, 1887. Dr. Ross.

PHARYNGEAL DIPHTHERIA. Parenchymatous degeneration of heart; acute parenchymatous nephritis.

86 (Johnston)—Male, 23. Dec. 5, 1887. Dr. Roddick.

SEPTIC PNEUMONIA. PHLEGMON AND CARBUNCLE of right side of lip and face; multiple pyæmic infarcts of lung; acute swelling of spleen; acute parenchymatous nephritis.

87 (Johnston)—Male, 60. (Private).

RUPTURE OF LEFT VENTRICLE INTO PERICARDIUM. Fatty infiltration of heart; thrombosis of left anterior coronary artery; cyanotic induration of spleen and kidneys; varicose veins of leg.

88 (Johnston)—Female, 38. Dec. 9, 1887. Dr. Molson.

ACUTE PURULENT MENINGITIS. Puerperal state; acute parenchymatous and chronic interstitial nephritis; acute swelling of spleen; acute and chronic purulent otitis media; thrombus right lateral sinus; gallstone impacted in cystic duct.

89 (Johnston)—Male, 56. Dec. 12, 1887.

FRACTURE OF SKULL. Traumatic softening and hæmorrhage of brain; pulmonary apoplexy; angioma of liver; concretion of prostate.

90 (Johnston)—Male, 50. Dec. 14, 1887.

EARLY GRAY HEPATIZATION OF LEFT LUNG. Acute purulent localized peritonitis left flank and pelvis; catarrhal colitis; acute parenchymatous nephritis, hepatitis and myocarditis; atrophy of left ascending frontal convolution.

91 (Johnston)—Male. Dec. 20, 1887.

ACUTE BRONCHITIS. Hyperæmia of lungs; chronic endocarditis; infarction of spleen. Found dead; suspected opium poisoning.

92 (Johnston)—Female, 3. Dec. 22, 1887. Dr. Major.

DIPHTHERIA of pharynx, larynx and trachea. Atelectasis; broncho-pneumonia; slaty pigmentation of intestinal follicles.

93 (Johnston)—Female, 38. Dec. 23, 1887. Dr. Ross.

TUBERCULOSIS, chronic ulcerative, both lungs; tuberculous meningitis; tuberculous ulceration of intestines; amyloid spleen and intestines.

94 (Johnston)—Male, 4. Dec. 24, 1887. Dr. Buller.

DIPHTHERIA of pharynx, larynx and trachea; tracheotomy; acute vesicular and interstitial emphysema; supernumerary spleen.

95 (Johnston)—Female, 3. Dec. 24, 1887.

DIPHTHERIA of larynx and trachea; acute broncho-pneumonia; accessory spleen.

1888

1 (Johnston)—Male, 30, Jan. 4th, 1888. Dr. Molson.

PULMONARY PHTHISIS. Right tuberculous pleurisy with effusion; miliary tuberculosis of spleen, liver and kidneys; tuberculous perihepatitis and peritonitis; acute tuberculous meningitis; chronic suppurative otitis media.

2 (Johnston)—Male. Private case. Jan. 6, 1888. Dr. Armstrong.

HÆMORRHAGE OF RIGHT CORPUS STRIATUM. Heart and kidneys normal

(Johnston)—Male. Private case. Jan. 6, 1888. Dr. Armstrong.

THROMBOTIC SOFTENING of left internal capsule. Gumma in third left frontal convolution; descending sclerosis of spinal cord.

4 (Johnston)—Private case. Jan. 12, 1888. Dr. Howard.

MEMBRANOUS LARYNGITIS and tracheitis. Beneath this mucosa appears to be intact; sudden death after intubation.

5 (Johnston)—Male 40. Jan. 12, 1888. Dr. Ross.

MILIARY TUBERCULOSIS. Chronic tuberculous caries lumbar vertebræ; acute miliary tuberculosis of lungs kidneys and liver; fatty liver, chronic hyperplasia of spleen; Calcareous and cystic degeneration of choroid plexus; Descending sclerosis of spinal cord: tuberculous ulcer of larynx with adherent slough.

6 (Johnston)—Private case. Jan. 21, 1888. Drs. Schmidt and Buller.

ACUTE HÆMORRHAGIC pachy- and leptomeningitis. Abscess of frontal sinus, following mucous cyst; purulent abscess of left frontal lobe, acute cerebritis of left frontal lobe; chronic atrophic rhinitis.

7 (Johnston)—Male, 67. Jan. 23, 1888. Dr. Shepherd.

ACUTE PYELONEPHROSIS. Acute Parenchymatous; hydronephrosis; dilated ureters; hypertrophy of middle lobe of prostate.

8 (Johnston)—Male, 68. Jan. 26, 1888. Dr. Ross.

ACUTE LOBAR PNEUMONIA, right upper lobe. Acute bronchitis; acute right pleurisy, adhesions both sides; slight cirrhosis of liver and kidneys; spleen small.

9 (Johnston)—Male, 65, Jan. 28, 1888.

SOFTENING OF BRAIN, with sclerosis; broncho-pneumonia; fœtid bronchitis; granular condition of fourth ventricle; ependymitis; softening in pons.

10. (Johnston)—Male, 40. Jan. 29, 1888.

ACUTE LOBAR PNEUMONIA right upper lobe. Acute pleurisy; slight granular kidneys.

11 (Johnston)—Male, 18. Jan. 30, 1888. Dr. Roddick.

TUBERCULOSIS CARIES OF DORSAL and lumbar vertebræ. Double psoas abscess; acute left pleurisy; chronic adhesive right pleurisy, non-tuberculous; acute swelling of spleen; slaty pigmentation of follicles of intestines.

12 (Johnston)—Male, 4 hours. Jan. 30, 1888. Dr. J. C. Cameron.

HÆMORRHAGE OF longitudinal sinus. Atelectasis of lungs.

13 (Johnston)—Male, 34. Feb. 8, 1888. Dr. Ross.

CANCEROUS ULCER OF STOMACH, invading left lobe of liver. Amyloid spleen, intestines and liver.

14 (Johnston)—Male, 24. Feb. 9, 1888. Dr. Bell.

AMPUTATION OF left femur sloughing of stump. Secondary hæmorrhage; artery sloughed through at point of ligation; pyæmic abscess in left pectoral region; fibrous anchylosis left hip; anæmia of organs.

15 (Johnston)—Male, 6. Feb. 10, 1888. Dr. Ross.

DIPHTHERIA of pharynx, larynx and trachea; broncho-pneumonia; intubation.

16 (Johnston)—Female, 40. Feb. 13, 1888. Drs. Molson and Stewart.

EXOPHTHALMIC GOITRE thrombosis of left vertebral and inferior cerebellar arteries, Hæmorrhagic cyst in pons (hæmatoma) slight chronic aortic endocarditis; infarction of spleen; cystic bronchocele.

17 (Johnston)—Female, 25. Feb. 15, 1888. Dr. Molson.

SUBACUTE TUBERCULOUS MENINGITIS. Slight acute broncho-pneumonia; chronic adhesive pleurisy; gestation.

18 (Johnston)—Female. Feb. 17, 1888.

ACUTE AORTIC AND MITRAL ENDOCARDITIS. Nutmeg liver; anasarca; acute parenchymatous myocarditis.

19 (Johnston)—Female, 21. Feb. 17, 1888.

VALVULAR HEART DISEASE. Dilatation of right ventricle; emphysema; nutmeg liver; hyperæmia of intestines; anasarca; hydrothorax; ascites; chronic adhesive perimetritis.

20 (Johnston)—Female, 20. Feb. 25, 1888. Dr. Ross.

PERFORATION OF ULCER OF STOMACH; general acute peritonitis; two other ulcers of stomach; gestation third month.

21 (Johnston)—Male, 20. Feb. 24, 1888. Dr. Bell.

PYÆMIA. Sutured right patella; abscess and phlegmon right leg; acute purulent arthritis right ankle and knee; acute verrucose infectious endocarditis; miliary abscess of heart; infarction of spleen, kidneys and left corpus striatum; cyst size of walnut in right lobe of liver; superficial ulcer of right shin.

22 (Johnston)—Male, 43. Feb. 25, 1888. Dr. Ross.

ACUTE LOBAR PNEUMONIA right upper lobe. Gummatous hepatitis, old.

23 (Johnston)—Male, Feb. 28, 1888.

ANEURYSM OF ABDOMINAL AORTA. Erosion of the body of the 3rd lumbar vertebra; dilatation of heart; atheroma of aorta; general arterio sclerosis; slight interstitial nephritis; chronic bronchitis; slight œdema of lungs.

24 (Lafleur)—Male, 1½. Feb. 28, 1888. Dr. Molson.

DIPHTHERIA OF PHARYNX, larynx and trachea. Tracheotomy; interstitial emphysema; atelectasis; broncho-pneumonia.

25 (Johnston)—Male, 60. Feb. 28, 1888. Dr. Roddick.

Railroad accident. FRACTURE OF RIBS, 7 to 11 right; laceration of pleura and right lung; traumatic pneumonia; bronchopneumonia; slight hæmorrhage into cerebral ventricles.

26 (Johnston)—Female, 45. March 2, 1888. Dr. Ross.

CHRONIC PARENCHYMATOUS NEPHRITIS. Chronic mitral and tricuspid endocarditis; cirrhotic and nutmeg liver; hæmorrhages of spleen; acute gastro-enteritis; hypertrophic endometritis.

27 (Johnston)—Male, 60. March 5, 1888. Dr. Ross.

PULMONARY PHTHISIS. Cavity in right lung; emphysema left lung; acute miliary tuberculosis both lungs; right tuberculous adhesive pleurisy; tuberculous ulcer of larynx; tuberculous enteritis; fatty liver; miliary tuberculosis of kidneys and spleen.

28 (Johnston)—Female, 80. March 5, 1888.

INOCULITION TUBERCULOSIS of subcutaneous tissue and muscles of left arm; of axillary glands; extension to left lung; brown atrophy of heart; atheroma of valves; chronic hypertrophic catarrhal colitis with polypi; polypoid gastritis; annular ulcer of pylorus.

29 (Johnston)—Female, 4. March 5, 1888. Dr. Ross.

DIPHTHERIA nose, pharynx and larynx (hæmorrhagic), old infarct of lung; acute hæmorrhagic broncho-pneumonia; infarct of spleen; emphysema of peritoneum; epi and endocardial ecchymoses; chronic mitral and aortic endocarditis.

30 (Johnston)—Female, 7. March 7, 1888.

DIPHTHERIA of pharynx, larynx and trachea. Acute inflation of lungs; Broncho-pneumonia; cloudy swelling of kidneys; follicular enteritis; ascarides.

31 (Johnston)—Female, 5. March, 7, 1888. Dr. Ross.

DIPHTHERIA of pharynx, larynx and trachea atelectasis; broncho-pneumonia; cloudy swelling of kidneys.

32 (Johnston)—Male, 50. March 12, 1888. Dr. Molson.

PNEUMONIA. Late red hepatization upper and middle lobe right lung; acute right pleurisy (fibrinous); acute bronchitis; cloudy swelling of kidneys and heart.

33 (Johnston)—Male, 25. March 12, 1888. Dr. Molson.

No organic disease found to account for death.

34 (Lafleur)—Male, 43. April 2, 1888. Dr. Bell.

FRACTURE OF RIGHT LEG. Syncope.

35 (Lafleur)—Male, 40. April 9, 1888. Dr. Molson.

PHTHISIS BOTH LUNGS. Tuberculous meningitis.

36 (Lafleur)—Male, 67. April 10, 1888. Dr. Bell.

GENERAL TUBERCULOSIS. Right tuberculous pyelitis.

37 (Lafleur)—Male, 65. April 10, 1888. Dr. Shepherd.

PNEUMONIA base of right lung, commencing pneumonia right upper lobe; pyelonephritis purulent; chronic cystitis; enlarged prostate; infiltration of urine with gangrene of scrotum and perineum.

38 (Lafleur)—Male, 21. April 12, 1888. Dr. MacDonnell.

POTASSIUM BICHROMATE POISONING. Acute gastro enteritis; methæmoglobimuria.

39 (Lafleur)—Male, 44. April 15, 1888. Dr. Bell.

SUPPURATIVE APPENDICITIS (chronic) with sinuses. Commencing perforative septic peritonitis; abscesses of liver; sero-fibrinous pericarditis; slight fatty kidney.

40 (Lafleur)—Female, 75. April 23, 1888.

CIRRHOSIS OF LIVER AND KIDNEYS, MULTILOCULAR CYST RIGHT OVARY. Cystic change of left ovary; fibromyoma uteri; senile atrophy of heart.

41 Not entered.

42 (Lafleur)—Male, 47. April 27. Dr. MacDonnell.

PNEUMONIA lower lobe both lungs; commencing pneumonia left upper lobe and right upper lobe; chronic parenchymatous nephritis: spleen firm 620 grams.

43. (Lafleur)—Male, 61. May 7, 1888. Dr. Bell.

ALVEOLAR SARCOMA of 9th and 10th dorsal vertebræ. Secondary in liver; broncho-pneumonia right upper lobe; gray hepatization right lower lobe; chronic interstitial nephritis; bedsore.

44 to 46 Not entered.

47 (Lafleur)—Male, 35. May 26, 1888. Dr. Macdonnell.

ACUTE TUBERCULOSIS with left lung; acute tuberculosis right lung; tuberculous ulcers throughout intestines; fatty liver.

48 Female, 60. May 26, 1888.

Not entered.

49 (Lafleur)—Female, 27. May 30, 1888. Dr. Wilkins.

TUBERCULOSIS with cavities; polypus fundus uteri at placental site; tuberculous ulcers of intestine and rectum; localized adhesive peritonitis.

50 Not entered.

51 (Lafleur)—Female, 29. June 1, 1888. Dr. Wilkins.

CHRONIC PARENCHYMATOUS NEPHRITIS. Slight interstitial nephritis; diphtheritic ulcerative colitis; right hydrothorax; chronic adhesive peritonitis; right pyosalpinx; chronic enlargement of spleen; gumma of spleen; warty excrescences of descending arch of aorta.

52 (Lafleur)—Male, 37. June 2, 1888. Dr. Shepherd.

TUBERCULOUS ULCER OF TONGUE. Removal of tongue; acute general miliary tuberculosis.

53-54 Not entered.

55 (Lafleur)—Male, 5. June 21, 1888.

DIPHTHERIA of nose, pharynx and larynx. Broncho-pneumonia; atelectasis.

56 (Lafleur)—Male, 32. June 27, 1888. Dr. MacDonnell.

PYÆMIA. Subpectoral abscesses; purulent arthritis; abscess of right lung.

57 (Lafleur)—Male, 10. June 28, 1888.

SCLEROTIC AND VERRUCOSE mitral endocarditis. Chronic interstitial nephritis; dilatation and hypertrophy of heart; nutmeg liver; œdema of lungs; double hydrothorax; hydropericardium; ecchymoses of bladder; anasarca.

58 (Lafleur)—Male, 70. June 28, 1888. Dr. MacDonnell.

FATTY AND CIRRHOTIC LIVER. Fatty degeneration and fat infiltration of heart; icterus; œdema of lungs; muco-purulent bronchitis; contracted granular kidneys; right inguinal hernia.

59 Not entered.

60 (Lafleur)—Male, 72. July 30, 1888. Dr. Shepherd.

STRANGULATED HERNIA. Radical operation; constriction of eight inches of intestine; acute lobar pneumonia; purulent bronchitis; caseous nodules apex and bronchial glands.

61-62 Notes not entered.

63 (Lafleur)—Female, 60. Aug. 4, 1888. Dr. Wilkins.

MULTIPLE PURPURIC ECCHYMOSES of skin, kidneys, bladder, stomach, intestines, pleura and pericardium; broncho-pneumonia; moma uteri.

64 (Lafleur)—Male, 32. Sept. 10, 1888. Dr. Roddick.

SUICIDE. Two bullet wounds left auditory canal; fracture left petrous bone; acute purulent meningitis; broncho-pneumonia.

65 (Lafleur)—Female. Sept. 18, 1888. Dr. Wilkins.

SYPHILIS. Gumma of lung and liver; ulceration of intestine; amyloid liver, spleen and kidneys.

66 (Lafleur)—Male, Sept. Dr. Wilkins.

ACUTE RIGHT LOBAR PNEUMONIA. Commencing pneumonia left base; gangrene of right lung; purulent thrombus cerebral sinuses; and veins of Galen; superficial encephalitis; hæmorrhagic spots in corpus callosum; middle ear normal.

67 (Lafleur)—Male, 16. Sept. 18, 1888. Dr. Wilkins.

TYPHOID FEVER ulceration of intestines.

68 (Lafleur)—Male, 79. Sept. 28, 1888. Dr. MacDonnell.

HYPERTROPHY and dilatation of heart; fatty degeneration. Hydrothorax; œdema of lungs; anasarca; cyanotic induration of kidneys; fatty liver; old scar on prepuce; slight double hernia; external hœmorrhoides.

69 (Lafleur)—Female, 47. Sept. 21, 1888. Dr. Roddick.

CARCINOMA OF RECTUM. Secondary in inguinal glands and liver; chronic bronchitis.

70 (Lafleur)—Male, one week. Sept. 24, 1888. Maternity.

CAPILLARY BRONCHITIS. Asphyxia; froth in trachea; aspiration of food.

71 (Lafleur)—Male, 60. Sept. 29, 1888.

CEREBRAL HÆMORRHAGE left corpus striatum; arterio-sclerosis; slight interstitial nephritis; slight hypertrophy of left ventricle.

72 (Lafleur)—Male, 19. Oct. 5, 1888. Dr. Ross.

APPENDICITIS. Perforation; acute general purulent peritonitis; cloudy swelling of heart muscle.

73 to 77 Not entered.

78 (Lafleur)—Male, 17. Nov. 10, 1888. Dr. Molson.

PHITHISIS. Cavities both lungs; anæmia; fatty liver and kidneys; brown-atrophy of heart; tuberculous enteritis; miliary tubercles in suprarenals.

79 (Lafleur)—Male, 52. Nov. 16, 1888. Dr. Bell.

CHRONIC ABSCESS OF PANCREAS. Obstructive jaundice; sero-fibrinous peritonitis; acute fibrinous pericarditis; calcified nodule lower lobe right lung; brown-atrophy of heart; sclerosis of valves; abscess of tip of vermiform appendix.

80 to 82 Not entered.

83 (Lafleur)—Female, 30. Nov. 30, 1888. Dr. Gardner.

ACUTE RIGHT lobar pneumonia. Subinvolution of uterus; parenchymatous myocarditis, slight fatty kidney; slight muco enteritis.

84 (Lafleur)—Female, 55, Dec. 6, 1888. Dr. Ross.

ACUTE LOBAR PNEUNONIA right upper lobe; acute diphtheritic entero-colitis; chronic cervical endometritis; fatty heart.

85 Not entered.

86 (Lafleur)—Female, 23. Dec. 12, 1888. Dr. Ross.

PHARYNGEAL DIPHTHERIA. Broncho pneumonia.

87 Not entered.

88 (Lafleur)—Male, 65. Dec. 20, 1888. Dr. Ross.

ACUTE FIBRINO purulent peritonitis. Cause not apparent: Sclerosis aortic valves; calcification; fibroid nodule left apex.

89-90 Not entered.

1889

1 (Lafleur)—Female, 50. Jan. 11, 1889. Dr. Ross.

CEREBRAL SOFTENING OF BOTH CRURA (thrombotic ?). Slight chronic interstitial nephritis; arterio-sclerosis; catarrhal enteritis; chronic adhesive pelvic peritonitis; brown atrophy of liver; caseous nodule in left lung; polypus of cervix uteri.

2 (Lafleur)—Male, 23. Jan. 25, 1889. Dr. Ross.

ACUTE SUPPURATIVE NEPHRITIS. Acute hæmorrhagic cystitis; acute enlargement of spleen; acute bronchitis.

3 (Lafleur)—Female, 35. Jan. 29, 1889.

TUBERCULOUS PERITONITIS. Acute miliary tuberculosis; bedsores; eczema about genitals.

4 (Lafleur)—Female, 17. Feb. 25, 1889.

VALVULAR HEART DISEASE.—Mitral sclerosis; dilatation and hypertrophy of heart; icterus; ascites; bedsore.

5 (Finley)—Female, 42. April 5, 1889. Dr. Wilkins.

CIRRHOSIS OF KIDNEY. Alcoholism; fatty and cirrhotic liver; fatty degeneration of heart; atheroma of aorta; ascites; anasarca; œdema of pia; encapsuled trichinosis of muscle (old).

6 (Finley)—Female, 40. April 14, 1889. Dr. Wilkins.

ULCER OF STOMACH with open artery in base. Fatty and cirrhotic liver alcoholism; cirrhotic kidneys; hæmorrhage in stomach and intestine; fatty degeneration of heart; ossifying pachymengitis; gall stones.

7 (Finley)—Female, 42. April 22, 1889. Dr. Stewart.

PUERPERAL SEPTICÆMIA. Purulent parametritis; abscess in both broad ligaments; no peritonitis; acute enlargement of spleen with infarct; slight interstitial nephritis; cloudy swelling of kidneys and liver; pyæmic abscesses in pectoral muscles.

8 (Finley)—Female, 40. April 30, 1889.

CARBOLIC ACID POISONING. Quantity unknown; 48 hours duration; punctiform hæmorrhages of stomach; commencing parenchymatous nephritis.

9 (Finley)—Female, 52. April 30, 1889. Dr. Roddick.

CANCER OF ASCENDING COLON infiltrating abdominal wall; lacing lobe of liver; thrombosis of iliac veins; acute purulent bronchitis; right broncho-pneumonia; acute right pleurisy.

10 (Finley)—Female, 55. April 30, 1889.

ACUTE LOBAR PNEUMONIA (left). Slight interstitial nephritis; hypertrophy of heart; lacing lobe of liver; œdema of glottis.

11 (Finley)– Male, 52. April 30. Drs. Ross and Shepherd.

ADENO CARCINOMA of sigmoid flexure with external and vesical fistulæ; secondary cancer of liver and retroperitoneal glands; old tuberculous pyelonephritis (right); caseous nodules in lungs.

12 Not entered.

13 (Finley)—Male, 45. May 8, 1889. (Coroner's case).

RUPTURE OF ILEUM. Bruising of abdomen; general peritonitis.

14 (Finley)—Female, 24. May 9, 1889. Dr. Sutherland.

PYÆMIA. Retropharyngeal abscess; thrombosis internal jugular and lateral sinus; abscess of dura mater; collapse of lungs; sclerosis mitral valve; cloudy swelling of liver and kidneys; acute enlargement of spleen; parovarian cysts; hydrosalpinx.

15 (Finley)—Female, 46. May 12, 1889. Dr. Stewart.

CANCER OF PYLORUS infiltrating colon; secondary in liver: fusion of pulmonary valves; aortic endocarditis with incompetence; angioma of liver; caseous nodules in apices.

16 (Finley)—Female, 19. May 26, 1889. Dr. Wilkins.

GENERAL PURULENT PERITONITIS. Extreme cirrhosis of kidneys; acute plastic pleurisy; hydrosalpinx; caseous nodules of lungs.

17 (Finley)—Male, 22. June 2, 1889. Dr. Stewart.

CHRONIC PHTHISIS WITH CAVITIES. Tuberculous enteritis; early tubercle of larynx; catarrhal gastritis; p.m. digestion and perforation of œsophagus.

18 (Finley)—Female, 18. June 2, 1889. Dr. Shepherd.

STRANGULATED INGUINAL HERNIA. No gangrene or peritonitis.

19 (Finley)—Male, 20. June 5, 1889. Dr. Stewart.

ACUTE PARENCHYMATOUS NEPHRITIS. Acute glomerulonephritis; hypertrophy and dilatation of heart; commencing atheroma of aorta; œdema of lungs; hyperæmia of intestines.

20 (Finley)—Male, 61. June 5, 1889. Dr. Stewart.

CIRRHOSIS OF STOMACH with ulceration. Cirrhotic kidneys; gall stones; tabes dorsalis.

21 (Finlay)—Male, 40. June 9, 1889. Dr. Stewart.

MULTIPLE LYMPHOSARCOMA? of pleura and peritoneum; sub acute pleurisy and pericarditis with effusion; tuberculosis?

22 (Finley)—Female, 36. May 14, 1889. Dr. Wilkins.

CHRONIC ULCERATIVE COLITIS. Fatty liver; chronic adhesive peritonitis.

23 (Finley)—Male, 66. June 10, 1889. Dr. Wilkins.

General Arteriosclerosis. Occlusion of ventricles of brain; miliary aneurysms; obliterative endarteritis; hypertrophy and dilatation of left heart; sclerosis of valves; atrophy of liver; dilatation of stomach; kidneys normal.

24 (Finley)—Male, 1½. June 20, 1889. Dr. Burland.

Perforative Appendicitis. Fœcal concretions in appendix; acute septic peritonitis.

25 (Finley)—Male, 47. June 24, 1889. Dr. Stewart.

Angiosarcoma Right Crus Cerebri.

26 (Finley)—Male, 25. June 24, 1889. Dr. Wilkins.

Typhoid Fever. Ulceration of intestines; perforation; commencing general peritonitis; acute enlargement of spleen.

27 (Finley)—Female, 21. June 25. Dr. Wilkins and Shepherd.

Right Empyæma. Thoracocentesis; collapse of lung; recent and old pleurisy; fatty liver.

28 (Finley)—Male, 27. June 27, 1889.

Valvular Disease of Heart. Aortic incompetence; old and recent aortic endocarditis; slight sclerosis of mitral value; universal synechiæ of pericardium; hypertrophy and dilatation of heart; œdema of lungs; spleen large and firm.

29 (Finley)—Male, 26. July 2, 1889. Dr. Stewart.

Sunstroke. Alcoholism; fluidity of blood, well marked rigidity in lower extremities; absent in upper 17 hours p.m. Granular and fatty degeneration with acute dilatation of both auricular and ventricular orifices; Punctiform hæmorrhages beneath epicardium; œdema and hyperæmia of lungs; enlargement of solitary follicles throughout intestines; early decomposition of organs.

30 (Finley)—Male, 50. July 9, 1889. Dr. Bell.

Scirrhus Adenocarcinoma of hepatic flexure of colon, with stricture; trichinosis; fatty liver with secondary cancer.

31 (Finley)—Female, 34. July 10, 1889. Dr. Stewart.

Acute Parenchymatous Nephritis. Pregnancy; fatty liver; catarrhal ulcers of colon.

32 (Finley)—Male, 55. Aug. 9, 1889. Dr. Wilkins.

Typhoid Fever. Ulceration of intestines (3rd week); parenchymatous degeneration of heart and liver; acute enlargement of spleen; small cavity at each apex; ununited fracture right external condyle; bullet embedded in frontal bone.

33 (Finley)—Female, 17. Aug. 13, 1889. Dr. MacDonnell.

Typhoid Fever. Ulceration of ileum and large intestine; large soft spleen; granular degeneration of liver and kidneys; œdema of lungs; slight chronic endocarditis of tricuspid and mitral valves.

34 (Finley)—Female, 34. Aug. 13, 1889. Dr. Stewart.

CASEOUS PNEUMONIA; left lung cavites at apex; œdema and caseous pneumonia right lung; universal pleural synechiæ; amyloid spleen liver and kidneys without enlargement; amyloid intestines.

35 (Finley) Male, 51. Aug. 23, 1889. Dr. MacDonnell.

ATROPHIC CIRRHOSIS OF LIVER. Jaundice; granular and fatty degeneration of heart; commencing atheroma of aortic valves; dilatation of both ventricles and auricular orifices; right hydrothorax; recent infarct of lung; old infarct of spleen and right kidney; gastro-intestinal catarrh; prostatic calculi; syphilitic ulceration and scarring of legs.

36 (Finley)—Male, 30. Aug. 27, 1889. Dr. Fenwick.

ABSCESS OF LIVER. Dysenteric ulceration of large intestine: broncho-pneumonia.

37 (Finley)—Female, 56. Sept. 4, 1889. Dr. MacDonnell.

ACUTE CATARRHAL ENTEROCOLITIS. Dilatation of stomach; brown atrophy of liver; pleural adhesions; calcified apical nodules; calcified and caseous bronchial glands; fatty degeneration of heart with slight dilatation of ventricles; no poison found chemically; suspected ptomaine poison; old perihepatic adhesions.

38 (Finley)—Male, 38. Sept. 10, 1889. Dr. Fenwick.

PYÆMIA. Left empyema; right broncho-pneumonia; acute plastic pleurisy; parenchymatous degeneration of liver and spleen; comminuted fracture of left thigh; fatty degeneration of right ventricle; collapse; induration and œdema of left lung.

39 (Finley)—Female, 19. Sept. 10, 1889.

TYPHOID FEVER (14th day). Swelling of Peyer's patches and of solitary follicles of large intestine to within three inches of rectum; cloudy swelling of liver; enlargement of spleen and mesenteric glands; atelectasis and hyperæmia of bases of lungs.

40 (Finley)—Male 25. Dr. Blackader.

ABSCESS OF BRAIN, left temporal lobe; perforation of left squamous bone.

41 (Finley)—Male, 45. Sept. 12, 1889. Dr. Wilkins.

CASEOUS PNEUMONIA. Caseous peribronchitis and cavity left lung; right lung, no cavity; slight tuberculous ulceration of ileum; commencing tuberculosis of larynx.

42 (Finley) Male 23. Sept. 14, 1889.

TYPHOID FEVER (16th day). Enlargement of intestinal follicles with reticular ulceration; enlargement of spleen and mesentric glands.

43 (Finley)—Male, 65. Sept. 15, 1889. Dr. MacDonnell.

VALVULAR HEART DISEASE. Hypertrophy of heart, bilateral; sclerosis of valves; nutmeg liver; ascites hydrothorax; œdema; catarrhal gastritis cyanotic induration of kidneys.

44 (Johnston)—Infant, 3 weeks. Sept. 27, 1889.

MARASMUS. No special change in organs; eczema about buttocks; syphilis?

45 (Williams)—Male, 25 days. Sept. 30, 1889.

MARASMUS. No special change in organs.

46 (Williams)—Male, 1 month. Sept. 28, 1889.

MARASMUS. Rachitis.

47 (Johnston)—Male, 50. Oct. 1, 1889. Dr. MacDonnell.

GOUT; CHRONIC INTERSTITIAL NEPHRITIS. Severe urate deposit in kidneys; extensive uratic arthritis and tenosynovitis throughout the whole body; tophi; follicular abscess of pharynx; enlargement of posterior cervical glands; acute left pleurisy with effusion; ecchymosis of mucosa of stomach; melæna.

48 (Johnston)—Private case. Oct. 4, 1889.

MEDULLARY CANCER OF STOMACH in middle region, greater curvature; secondary cancer of liver.

49 (Johnston)—Male, 35. Oct. 5, 1889.

TYPHOID FEVER. Ulceration of ileum, colon and appendix; a little blood in intestine above valve; slaty pigmentation of Peyer's patches; acute swelling of spleen and mesenteric glands; hyperæmia and partial atelectasis of both lungs at bases; patency of foramen ovale; slight hyaline degeneration of recti abdominales; fatty liver; ecchymoses on surface.

50 (Johnston)—Male, 74. Oct. 14, 1889. Dr. Molson.

INTERSTITIAL NEPHRITIS. Acute and chronic bronchitis; dilatation of heart with fatty degeneration; atheroma; nutmeg liver; chronic adhesive peritonitis; old tuberculosis at apices of lungs.

51 (Johnston)—Male, 27. Oct. 15, 1889. Dr. Roddick.

FRACTURE OF LEFT OS INNOMINATUM. Dislocation of both sacro-iliac articulations; Pott's fracture of left leg; laceration of intestines.

52 Johnston)—Female, 40. Oct. 21, 1889.

ACUTE LEFT LOBAR PNEUMONIA with pleurisy; multiple broncho-pneumonia; large soft spleen; fatty liver.

53 (Johnston)—Male, 28. Oct. 27, 1889. Dr. Molson.

MASTOIDITIS, phlebitis of right lateral sinus and internal jugular. Chronic purulent right otitis media. Acute swelling of spleen with multiple infarcts; accessory spleen; acute parenchymatous nephritis; acute and chronic local peritonitis; anæmia; slight catarrhal enteritis; hyperæmia of lungs with slight pneumonia.

54 (Johnston)—Female, 10. Oct. 29, 1889. Dr. MacDonnell.

ACUTE MENINGITIS (non-tuberculous). Sabacute pachymeningitis, with distension of ventricles; acute pneumothorax from localized necrosing pleuritis of left lung; hæmorrhage into papillary muscles; optic neuritis.

55 (Johnston)—Male, 40. Oct. 31, 1889. Dr. Bell.

FRACTURE OF laminæ of 5th and 6th vertebræ. Laminectomy; partial laceration and hæmorrhage of spinal cord.

56 (Johnston)—Male, 57. Oct. 31, 1889. Dr. MacDonnell.

SCIRRHOUS CANCER OF PYLORUS; secondary cancer of right kidney and posterior mediastinal glands.

57 Female, 45. Nov. 4, 1889. Dr. Molson and Stewart.

DIPHTHERIA of pharynx, larynx, trachea and bronchi. Emphysemia and broncho-pneumonia; acute hæmorrhagic nephritis; myoma uteri.

58 (Johnston)—Male, 27. Nov. 6th, 1889. Dr. MacDonnell.

ACUTE LOBAR PNEUMONIA. Hæmorrhagic infarction of right lung; acute pleurisy; acute croupous tracheitis; parenchymatous nephritis; foreign body in appendix vermiformis.

59 (Johnston)—Private case. Nov. 13, 1889. Dr. Browne.

RUPTURE OF HEART. Hæmorrhage into pericardium; myomalacia cordis; fat infiltration of heart; atheroma of coronaries.

60 (Johnston)—Male, 67. Nov. 14, 1889. Dr. Roddick.

MELANOTIC SARCOMA OF SKIN OF LEFT GROIN. Secondary in inguinal glands; universal adhesive pericarditis; mural thrombus with white softening of right ventricle; grey degeneration of cœliac ganglia; pigmentation of skin of forehead; chronic granular kidneys; thickening of spleen capsule; hyaline thickening of splenic artery; hæmorrhoids; complete fistula in ano.

61 (Johnston)—Female, 27. Nov. 15, 1889. Dr. Molson.

ACUTE MILIARY TUBERCULOSIS. Anæmia; broncho-pneumonia; old apical cavities; enlarged spleen; tuberculosis of bronchial glands.

62 (Johnston)—Female, 63. Nov. 18, 1889.

ATROPHIC CIRRHOSIS OF LIVER. Emphysema; obsolete tuberculosis of apices; brownish rash on legs; pedunculated cyst of right ovary; ascites.

63 (Johnston)—Male, 50. Private case.

CHRONIC INTERSTITIAL NEPHRITIS. Chronic ulcerative catarrhal colitis; enlargement of suprarenal capsules; fibrosis of cœliac plexus.

64 (Johnston)—Female, 22. Nov. 24, 1889.

TYPHOID FEVER (stage of healing with relapse). Multiple follicular ulceration of stomach with hæmorrhagic erosions; acute parenchymatous nephritis; abortion; lactation; suppurative left mastitis; fatty degeneration of intima of aorta; parenchymatous myocarditis; slight laceration of cervix uteri.

65 (Johnston)—Female, 40. Nov. 30, 1889. Dr. MacDonnell.

ACUTE LOBAR PNEUMONIA. Chronic insterstitial pneumonia with organization of the exudate; chalicosis; acute purulent bronchitis; caseous bronchial glands; brown atrophy of heart; slight cloudy swelling of kidneys.

66 (Johnston)—Male, 49. Dec. 1, 1889. Dr. Molson.

ATROPHIC CIRRHOSIS OF LIVER. Acute tuberculous pleurisy and peritonitis; chronic pulmonary tuberculosis; hæmatoma in right iliac fossa (course not established); brown atrophy of heart; atheroma of aorta; cyanotic induration of kidneys; hæmorrhagic infarction of spleen; ulceration of ileum (tuberculous).

67 (Johnston)—Male, 55. Dec. 3, 1889. Dr. MacDonnell.

CANCER OF PYLORUS. Secondary in liver, lymph glands and peritoneum; ascites; chronic hypertrophic cystic gastritis; chronic fœtid bronchitis; broncho pneumonia.

68 (Johnston)—Male, 40. Dec. 7, 1889. Dr. Bell.

COMPOUND FRACTURE of left tibia and fibula. Exostosis of skull cap; osteophytes of dura mater.

69 (Johnston)—Female, 44. Dec. 12, 1889. Dr. MacDonnell.

MULTIPLE THROMBOSIS AND RECURRENT PULMONARY EMBOLISM. Thrombosis of femoral vein; subacute peritonitis; localized right adhesive pleurisy; dilatation of both fallopian tubes; interstitial pancreatitis with cysts; ecchymosis of stomach; end-thrombosis of gastric veins; erythema nodosum; ascites.

70 (Johnston)—Female, 17. Dec. 19, 1889.

MULTIPLE GANGRENE OF RIGHT LUNG. Chronic mitral and acute mitral and aortic endocarditis; universal pericardial synechiæ; hypertrophy and dilatation of both sides of heart; obliterative thrombosis of left internal jugular, vertebral and innominate veins; thrombosis of left femoral vein; embolism of pulmonary arteries; acute bronchitis; acute right pleurisy with effusion; red atrophy of liver; cyanotic induration of kidneys; ascites; anasarca.

71 (Johnston)—Female, 4. Dec. 20, 1889.

DIPHTHERIA of pharynx, larynx, trachea and main bronchi. Hyperæmia of both lungs; slight broncho-pneumonia; cloudy swelling of kidneys; slaty pigmentation of Peyer's patches.

72 (Johnston)—Male, 55. Dec. 21, 1889. Dr. Molson.

Chronic mitral endocarditis; dilatation and hypertrophy both sides of heart; mural thrombi; brown induration with multiple infarcts; double hydrothorax; cyanotic atrophy of liver; chronic interstitial nephritis.

73 (Johnston)—Male, 24. Dec. 23, 1889. Dr. Roddick.

LACERATION OF URETHRA behind bulb. Moderate stricture of urethra; congestion of kidneys? cloudy swelling of liver; ecchymoses of stomach and heart; calcification of bronchial glands; toxæmia. (?)

74 (Johnston)—Male, 55. Dec. 28, 1889. Dr. MacDonnell.

CEREBRAL HÆMORRHAGE (recurrent). Chronic interstitial nephritis; albuminuric retinitis; atrophy of tongue; arterio sclerosis; hypertrophy of heart; chronic mitral endocarditis; old healed tubercles at left apex; acute broncho pneumonia; emphysema; slight brown induration of lungs.

75 (Johnston)—Male. Dr. MacDonnell.

CHRONIC INTERSTITIAL NEPHRITIS. Hypertrophy of heart with dilatation.

1890

1 (Finley)—Male, 55, Jan. 22nd, 1890. Dr. MacDonnell.

ACUTE LOBAR PNEUMONIA, left upper lobe. Hypertrophy and dilatation of heart; chronic aortic endocarditis with fusion of segments; acute endocarditis with small aneurysm of sinus valsalæ and of pars membranosa; slight mitral endocarditis; cyanotic induration of kidnies and spleen infarct of spleen.

2 (Johnston)—Male, 49, Feb. 3rd, 1890. Dr. MacDonnell.

ACUTE LOBAR PNEUMONIA, left base. Acute fibrinous pleurisy; emphysema; acute parenchymatous nephritis and chronic interstitial nephritis; pyelitis and hydronephrosis; chronic purulent prostatitis; brown atrophy of heart; fibrous nodule, free in peritoneum. Nothing abnormal in pancreas or brain.

3 (Johnston)—Female, 60, Feb. 2nd, 1890. Private case.

CHRONIC MITRAL ENDOCARDITIS, with stenosis. Dilatation and hypertrophy of heart; slight interstitial nephritis; ascites; hydrothorax.

4 (Johnston)—Male, 44, Feb. 6th, 1890. Dr. MacDonnell.

ANEURYSM OF TRANSVERSE ARCH OF AORTA. Moderate compression of trachea; hyperæmia of lungs; cyst in wall of gall bladder; phlebotomy.

5 (Johnston)—Male, 33. Dr. Bell.

STRICTURE OF URETHRA; chronic cystritis; prostatitis; pyelonephritis; emphysema of lungs; thickening of capsule of spleen.

6 (Johnston)—Male.

CHRONIC SUPPURATIVE TUBERCULOUS URETHRITIS. Diphtheritic hæmorrhagic cystitis; multiple chronic interstitial nephritis; obsolete apical tuberculosis; chronic interstitial pneumonia right lung; brown atrophy of heart and liver; bed sores.

7 (Johnston)—Female, 25, Feb. 8th, 1890. Dr. MacDonnell.

INFLUENZA. Multiple Miliary Hæmorrhages of white substance of brain, especially in internal capsule; bronchopneumonia; acute glomerulo nephritis.

8 (Johnston)—Male, 37, Feb. 8th, 1890. Dr. Bell.

DOUBLE HERNIA. Radical operation. Slaty pigmentation of peritoneum in iliac regions; bronchopneumonia; slight fatty liver; cyst in left corpus callosum (lymphatic cyst).

9 (Johnston)—Male, 23, Feb. 10th, 1890.

ACUTE TUBERCULOUS MENINGITIS. Chronic pulmonary tuberculosis; excision of left testicle (for tubercle); atrophy of left seminal vesicle; chronic abcess in right kidney and left pectoral region; calcification in tonsils.

10 (Johnston)—Male, 50, Feb. 10th, 1890. Dr. MacDonnell.

CRLEBRAL HÆMORRHAGE ANEURYSM; rupture of angioma of liver; slight interstitial nephritis arteriosclerosis; hypertrophy of heart.

11 (Johnston)—Male, 34, Feb. 19th, 1890. Dr. MacDonnell.

ANEURYSM OF ARCH. Rupture into posterior mediastinum, with formation of false aneurysm, which ruptured into stomach; hæmorrhage into sheath of vagus nerve; recent and old infarcts of lungs; enlargement of spleen; blood clot in stomach and duodenum.

12 (Johnston)—Feb. 20th, 1890.

CHRONIC PHTHISIS with cavities (both lungs); caseous pneumonia; universal adhesive pleurisy.

13 (Johnston)—60. Private case.

PYÆMIC ABSCESSES in great omentum, mesorectum and gastro hepatic omentum; abscesses in spleen; thrombi in both iliac veins; pneumonia.

14 (Johnston)—Female, 50, Feb. 28th, 1890. Dr. Molson.

PULMONARY EMBOLISM AND MULTIPLE GANGRENE OF LUNG. Right pleurisy with effusion; nutmeg liver; myoma uteri; anomaly of renal vessels, branch of renal artery given off from aorta, a bifurcation, hypertrophy and dilatation of heart.

15 (Johnston)—Male, 19, March 2nd, 1890. Dr. Bell.

ULCERATIVE MALIGNANT ENDOCARDITIS of mitral and aortic valves; miliary embolic suppurative nephitis; acute septic miliary infarcts of spleen; stricture of urethra; fistula in perineo; hyperœmia and atelectasis base of right lung; chronic adhesive local peritonitis and pleuritis without œdema of arachnoid; streptococcus infection.

16 (Johnston)—Male, 22, March 4th, 1890. Dr. MacDonnell.

ACUTE DOUBLE PLEURISY. PHLEGMON of deep cervical fascia and mediastinum; acute pericarditis; chronic enlargement of spleen; cloudy swelling of kidneys; chronic local peritonitis; caseation and calcification of bronchial glands.

17 (Johnston)—Male, 28, March 7th, 1890.

ACUTE MENINGITIS. Right purulent otitis media; gonorrhœa; caseation of bronchial glands; hyaline appearance of kidneys without amyloid reaction.

18 (Johnston)—Female, 42, March 10th, 1890. Dr. Bell.

BALL THROMBUS L. AURICLE, CHRONIC MITRAL ENDOCARDITIS, with stenosis; dilatation of left auricle, embolism and secondary thrombosis of iliac arteries; gangrene of left foot and leg; acute synovitis of left knee; nutmeg liver; brown induration of lungs; infarcts of kidneys and spleen; hydrothorax.

19 (Johnston)—Private case.

SUPPURATIVE CHOLECYSTITIS, with perforation; gall stones; chronic adhesive peritonitis and acute general purulent peritonitis; bronchopneumonia.

20 (Johnston)—Male, 32, March 21st, 1890.

COLLOID CANCER OF BLADDER. Secondary in pelvic glands; cystotomy operation; double hydronephrosis; anasarca hydrothorax; hydropericardium œdema of lungs; bronchopneumonia; hypertrophy of heart with dilatation; fatty degeneration of heart.

21 (Johnston)—Male, 54, March 24th, 1890. Dr. MacDonnell.

ACUTE LOBAR PNEUMONIA right lower and middle lobes. Chronic interstitial pneumonia; chronic adhesive pleurisy and acute fibrinous pleurisy; hypertrophy of heart; hypertrophy and cirrhosis of kidneys.

22 (Johnston)—Female, 20, March 31st, 1890. Private.

CHRONIC ULCER OF STOMACH. Perforation; acute peritonitis; anæmia.

23 (Johnston)—Female, 62. Dr. Molson.

CANCER OF STOMACH AT FUNDUS. Secondary in portal glands; perisplenitic abscess; acute peritonitis; bronchopneumonia; brown atrophy of heart; arteriosclerosis nodosa.

24 (Johnston)—Female, 14, March 31st, 1890. Dr. Bell.

RETROPHARYNGEAL AND RETROŒSOPHAGEAL CASEOUS TUBERCULOUS ABSCESS. Œdema and emphysema about neck; acute right pleurisy; acute (tuberculous?) peritonitis; infiltration of sheaths of pneumogastric nerves; no caries of cervical vertebræ; constriction of œsophagus.

25 (Johnston)—Male, 55, April 10th, 1890. Dr. Ross.

ANEURYSM OF AORTA in ascending arch. General arteriosclerosis of aortic valve; dilatation and hypertrophy with fatty degeneration of heart; chronic adhesive pleurisy; interstitial pneumonia; calcified bronchial glands; chronic enlargement of spleen; cyanotic induration of kidneys and liver; Liebermeister's furrows; angioma of liver.

26 (Johnston)—Male, 61, April 10th, 1890. Dr. Fenwick.

SUICIDE, by revolver wound of brain. Fracture of right orbital plate, right lachrymal and nasal bones; suppuration in track of wound; abscess in right antrum of Highmore; chronic hypertrophic rhinitis; laceration of anterior frontal region, right side; acute septic meningitis.

27 (Johnston)—Female, 3½.

ACUTE BRONCHO-PNEUMONIA. Catarrhal laryngitis.

28 (Johnston)—Male, 50, April 22nd, 1890. Dr. MacDonnell.

SUB-ACUTE LEFT PLEURISY AND EMPYEMA. Chronic aortic endocarditis with hypertrophy and dilatation; chronic adhesive pericarditis; cyanotic induration of kidneys and spleen with thickening of spleen capsule; nutmeg liver; chronic local peritonitis; right inguinal hernia; right hydrocele; necrosing ostitis l. tibia; thrombosis right branch of hepatic artery.

29 (Johnston)—Female, April 22nd, 1890. Dr. Ross.

CHRONIC PHTHISIS, with cavity. Tuberculous laryngitis and tracheitis; chronic adhesive pleurisy and recent tuberculous pleurisy; tuberculous bronchial glands; tuberculous ulceration small and large intestine; miliary tubercles of kidney and liver; amyloid stomach, bladder, liver, kidney, and pancreas; infarct and tubercle of spleen; bed sores; patency of foramen ovale.

30 (Johnston)—Male 57, April 26th 1890. Dr. Wilkins.

DOUBLE ACUTE PNEUMONIA, of upper lobe; acute left pleurisy; slight chronic endocardits; slight brown atrophy of heart; diverticula of colon; fatty liver; cloudy swelling of kidneys.

31 (Johnston)—Female, 50, May 3, 1890.

CANCER OF UTERUS, infiltrating sigmoid flexure and rectum; secondary cancer of liver; chronic pulmonary tuberculous, miliary tuberculosis of liver; tuberculous peritonitis; cancer of R. ureter R. hydronephrosis; brown atrophy of heart; milk patch.

32 (Johnston)—Male, 22, May 5, 1890. Dr. Ross.

DUODENAL ULCER, perforation, peritonitis and R. pleurisy.

33 (Johnston)—Female, May 5, 1890. Dr. Shepherd.

RENAL CALCULUS, nephrectomy left kidney; pyelonephritis on R. side,

34 (Johnston)—Private Case. Dr. Buller.

SARCOMA OF PITUITARY GLAND, large round-celled; optic neuritis and atrophy.

35 (Johnston)—EMPHYSEMA and œdema of lungs; chronic and acute bronchitis; nutmeg liver, interstitial nephritis (diffuse); dilatation of heart with fatty degeneration; fusion of aortic cusps and sclerosis.

36 (Johnston)—Female 33.

ACUTE LEFT LOBAR PNEUMONIA, both lobes; acute pericarditis; acute R. pleurisy; parenchymatous nephritis and hepatitis; acute swelling of spleen; subinvolution; puerperal state; thrombi beneath placental site, firm acute aortic endocarditis with perforation, and long fibrinous tag attached.

37 (Johnston)—Female, 33.

ACUTE GENERAL PERITONITIS. Chronic and acute parametritis; gangrenous cyst in region of left fallopian tube; cystoma left ovary; R. pyosalpynx.

38 (Johnston)—Male, 10, May, 20, 1890. Private case.

TUBERCULOUS MENINGITIS. Brain only examined.

39 (Johnston)—Male, 49, May 29, 1890. Dr. Ross.

CEREBRAL PLUMBISM. Atrophy of brain cortex; neuro-retinitis; hæmorrhage into pons; enlarged spleen; kidneys not cirrhotic.

40 (Johnston)—Male, 42, June 5, 1890.

CHRONIC PHTHISIS WITH CAVITY. Interstitial pneumonia; tubercles in spleen and wall of intestines, Cyst R. epididymis; tuberculous laryngitis; slight mitral endocarditis; tubercles in liver; caseous abscess in wall of bladder.

41 (Johnston)—Male, 7 months. Dr. Corsan.

CHRONIC TUBERCULOSIS, PART OF R. LUNG; chronic pleurisy; acute miliary tuberculous liver, spleen and right lung; caseous epigastric and bronchial glands; tuberculosis of heart muscle and endocardium of mitral ring.

42 (Johnston)—Male, 43, July, 27, 1890.

RIGHT PYELONEPHRITIS. Nephrotomy, purulent cystitis; old stricture of urethra; old mitral endcarditis; old infract of spleen.

43 (Johnston)—Male, 67, July 30, 1890.

ACUTE LOBAR PNEUMONIA. Red hepatization right lower lobe; old infarcts of lung with emphysema of both lungs; hypertrophy and dilatation of heart; fatty liver; infarcts spleen and kidneys.

44 Female (29) August 5, 1890. Dr. Ross.

OBSTRUCTIVE JAUNDICE; acute parenchymatons nephritis; gall stones; liver 2,750 grms.; soft

45 Male (24) Aug 12, 1890. Dr. Shepherd.

RIGHT PYCLONEPHRITIS. Left hydronephrosis; stricture of urethra.

46 Not entered.

47 (Johnston)—Male, 23.

TYPHOID FEVER. Ulceration of solitary follicles in large intestine and of Peyers patches; acute enlargement of spleen and mesenteric glands; cloudy swelling of liver.

48 (Johnston)—Male, 61, Sept. 14, 1890.

CHRONIC PHTHISIS, with cavity pulmonary hæmorrhage; recurrent fibro-sarcoma of skin of abdomen.

49 (Johnston)—Male, 20, Sept. 24, 1890.

ACUTE PNEUMONIA, right lung; acute double pleurisy; small hæmatomata beneath capsule of spleen.

50 (Johnston)—Male, Sept. 24, 1890. Private case.

CEREBRAL HÆMORRHAGE; fine aneurysin of inferior cerebellar artery; slight interstitial nephritis; pressure upon medulla.

51 (Johnston)—Female, 39, Oct. 5, 1890. Dr. Bell.

MALIGNANT ADENOMA OF R. KIDNEY and suprarenal; gangrenous and putrid R. perinephritis; old apical tubercles both lungs with acute miliary tubercle apex of R. lung, and acute R. pleurisy; cystoma R. ovary. Anæmia.

52 (Johnston)—Male, 52, Oct. 8, 1890. Dr. Bell.

EPITHELIOMA OF TONGUE. Secondary in lungs; excision of tongue; multiple necrosing pneumonia; arteriosclerosis.

53 (Johnston)—Female, 60, Oct. 16, 1890. Dr. Wilkins.

CANCER OF STOMACH, diffuse form with cirrhosis and diminution in size; chronic stenosing mitral endocarditis; brown atrophy heart and liver; calculus of left kidney.

54 (Johnston)—Male, 36, Oct. 22, 1890. Dr. Wilkins.

SYPHILITIC ULCERATION OF LARGE INTESTINE, adhesive perityphlitis from ulcer of rectum and descent of rectum on right side; syphilitic ostitis and periostitis of tibia and cranium; œdema of lungs; bronchopneumonia; amyloid kidney and chronic parenchymatous nephritis; ulceration of pharynx; cirrhosis of liver; fœcal concretion in appendix; local adhesive peritonitis.

55 (Johnston)—Female, 23, Oct. 28, 1890. Private case.

CHRONIC ULCER OF STOMACH and perforation. Acute purulent perforative peritonitis. Parenchymatous swelling of kidneys and liver, spleen and heart.

56 (Johnston)—Male, Nov. 7, 1890. Verdun Asylum.

DIFFUSE SCLEROSIS OF BRAIN, thickening and opacity of pia; subdural nodule over R. hemisphere; enlargement of supra renals.

57 (Johnston)—Female, 51, Nov. 20, 1890. Dr. Gardner.

SCARCOMA OF UTERUS, abdominal hysterectomy; acute peritonitis with serous effusion; cysts of spleen; arterio sclerosis; elongation left lobe of liver.

58 (Johnston)—Male, 50, Nov. 20, 1890. Dr. MacDonnell.

DIABETES, acetonuria; chronic phthisis; slight interstitial pleuritis; brain normal.

59 (Johnston)—Male, 50, Nov. 22. Private case.

DIABETES, acetonuria; thickening of pia; brain tissue firm but no focal disease; pancreas normal; kidneys large and firm, contain a few cysts.

60 (Johnston)—Male, 40, Dec. 6, 1890. Dr. MacDonnell.

CHRONIC MITRAL AND AORTIC ENDOCARDITIS. Dilatation and hypertrophy of heart; universal adhesive pericarditis; cyanotic induration of lungs, liver and kidneys; old infarcts in spleen and kidneys; anasarca; ascites; R. hydrothorax; chronic left otitis media.

61 (Johnston)—Male, 4, Dec. 6, 1890, Dr. Corsan.

ACUTE BRONCHO-PNEUMONIA and catarrhal rhinitis: polypoid thrombus of R. auricle.

62 (Johnston)—Male, Dec. 9. Private case.

CHRONIC PHTHISIS, with cavity of left lung. Left tuberculous pleurisy with effusion; amyloid spleen, liver and kidney.

63 (Johnston)—2½ years, Dec. 10. Private case.

ACUTE BRONCHO-PNEUMONIA, thrombus in longitudinal sinus and right renal vein.

64 (Johnston)—Male, 34, Dec. 20. Dr. Molson.

GONORRHŒAL ULCERATIVE ENDOCARDITIS of triscuspid and aortic valves involving septum membranaceum; slight dilatation and hypertrophy of heart; chalicosis lungs with slaty pigmentation; chronic bronchitis and bronchiectasis; chronic interstitial pneumonia disseminated; chronic purulent urethritis; enlargement of spleen, liver and kidneys.

65 (Johnston)—Male, 30, Dec. 14, 1890. Dr. Molson

EMPHYSEMA. Dilatation and hypertrophy of heart; cyanotic induration of kidney; multiple sclerosis of brain with superficial hæmorrhages.

66 (Johnston)—Male, 40.

CHRONIC PARENCHYMATOUS NEPHRITIS. Hypertrophy of left ventricle; gliosis; old apoplectic cyst; uræmia.

67 (Finlay)—Male, 54.

STRICTURE OF URETHRA; ENLARGED PROSTATE; cystitis; hydronephrosis; hypertrophy of left ventricle; Liebermeister's furrows.

68 (Finlay)—Male, 21, Dec. 24, 1890.

PROFOUND ANÆMIA; dilatation and hypertrophy of heart; fatty heart; petechiæ mucosa of colon.

69 Male, 19, Dec. 30, 1890. Dr. MacDonnell.

APPENDICITIS, perinephritic abscess; acute purulent peritonitis; acute swelling of spleen, kidneys and liver.

1891

[1 to 19—Records lost].

20 (Johnston)—Male, 28. March 9th, 1891. Dr. MacDonnell.

ADENOID CANCER OF ILEUM near valve; chronic adhesive peritonitis with ulceration and fistula of ileum; cloudy swelling and slight interstitial nephritis.

[21 to 28—Records lost].

29 (Finley)—Male, 55. April 22nd, 1891. Dr. Ross.

CORTICAL HÆMORRHAGE OF BRAIN. Atrophy and hydronephrosis of R. kidney; enlargement (compensatory) of left kidney; fatty and cirrhotic liver; enlarged spleen; old fibroid nodules and adhesions of R. apex; calcareous bronchial glands; œdema of lungs; atheroma of abdominal aorta.

30 (Finley)—Male. 23. May 3rd, 1891.

ACUTE PNEUMONIA, lower lobe of R. lung; acute bronchitis; hypertrophy of left ventricle.

31 Finley)—Male, 59. May 4th, 1891. Dr. Wilkins.

PRIMARY PURULENT PERICARDITIS; slight fibrinous pleurisy.

32 (Finley)—Male, 48. May 11th, 1891. Dr. Wilkins.

ACUTE NEPHRITIS. Hypertrophy of left ventricle with slight fibrosis tip of papillary muscle; right hydrothorax; hydropericardium; œdema of lungs.

[33—Record lost.]

34 (Finley)—Female, 24. May 13th, 1891. Dr. Wilkins.

TUBERCULOUS MENINGITIS. Caseous nodule in cerebellum; bronchopneumonia left lower lobe; cystoma of ovaries; no appearance of miliary tubercles in organs.

35 (Finley)—Female. May 11th, 1891. Maternity.

ACUTE MENINGITIS of pia and dura over left convolutions; no spinal meningitis; no cause discovered; cloudy swelling of liver, kidneys and spleen.

36 (Finley)—Male, 34. May 16th, 1891. Dr. Wilkins.

ACUTE ASCENDING PARALYSIS; no lesions of organs noted at autopsy; brain and cord normal.

37 (Finley)—Male, 42. June 1st, 1891. Dr. Ross.

PNEUMONIA OF RIGHT BASE WITH ACUTE PLEURISY. Cirrhotic kidneys; hypertrophy of left ventricle; cirrhosis of liver; chronic necrosing ostitis of skull cap (syphilitic ?).

38 (Finley)—Female, 18. June 7th, 1891. Dr. Ross.

ACUTE MULTIPLE PNEUMONIA, RIGHT UPPER LOBE. Acute swelling of spleen, liver and kidneys; thrombosis of right iliac vein; catarrhal ulceration of large intestine; old thickening of mitral valves; old infarcts of kidneys.

39 (Finley)—Child, 6 days. May 18th, 1891.

ACUTE CEREBRO SPINAL MENINGITIS. Child of No. 35, 1891.

40 (Finley)—Male, 52. June 20th, 1891. Dr. Ross.

PERFORATIVE APPENDICITIS. Localized purulent peritonitis; unusual site of appendix; patent tunica vaginalis with communicated peritonitis; cellulitis of abdominal wall; acute right pleurisy; slight atheroma of aortic valves.

40 a (Finley)—Female, 52. June 22, 1891. Dr. Ross.

PRIMARY CANCER OF BREAST. Secondary in vertebræ, liver, ribs and pleuræ.

41 (Finley)—Female, 4½. June 23, 1891. Dr. Corson.

DIPHTHERIA of pharynx, larynx and trachea; broncho-pneumonia; cloudy swelling of liver.

42 (Finley)—Male, 52. July 7, 1891. Dr. Armstrong.

PYÆMIA. Right empyema; acute left pleurisy; miliary abscesses heart and kidneys; stricture of urethra; chronic dilatation and hypertrophy of bladder: slight atheroma of aortic and mitral valves; gummatous scars of liver and skin; gallstones.

43 (Finley)—Male, 51. July 27, 1891. Dr. Molson.

CANCER OF ŒSOPHAGUS. (Aspiration?) pleuro-pneumonia; acute general peritonitis; secondary cancer of liver, mesentery and omentum; angioma of liver.

44 (Finley)—Female, 22. July 28, 1891. Drs. Shepherd and Birkett.

ABSCESS OF LEFT LOBE OF CEREBELLUM. Chronic inflammation of left tympanum and mastoid; lateral sinus normal. Only head examined.

45 (Finley)—Verdum Asylum case. Dr. Burgess.

SARCOMA OF BRAIN lying to right side of pons and replacing right lobe of cerebellum.

46 (Finley)—Male, 36. Aug. 8, 1891.

FRACTURE OF RIGHT SQUAMO-TEMPORAL BONE. Fracture of right femur and right side of pelvis. Fell down elevator shaft.

47 (Finley)—Male, 58. Sept. 30, 1891. Dr. Armstrong.

TETANUS. Crush of leg; (Ry. accident); amputation twice—first time wound very foul and flaps sloughed; some interstitial nephritis; no tetanus bacilli found in wound; organs showed nothing special.

48 (Finley)—Male, 2½.

ACUTE MENINGITIS. Greenish gelatinous lymph over frontal and parietal lobes; no evidence of tubercles at vessels; interpeduncular space free; tympana normal; non-tubercular.

1892

1 to 19—Reports missing.

20 (Finley)—Female, 40. April 3, 1892. Dr. Armstrong.

Cerebral Hæmorrhage R. Arteriosclerosis; hypertrophy of left ventricle; cirrhotic kidney; R. kidney converted into a mass of small cysts at its lower end.

21 (Finley)—Infant. April 9, 1892. Maternity.

Pyæmia. Suppurative omphalo-phlebitis; peritonitis; multiple abscesses; parenchymatious degeneration of liver, spleen and kidneys.

22 (Finley)—Male, 40. April 24, 1892. Private.

Fibroid Heart. Atheroma of aorta; atheroma of coronaries.

23 (Finley)—Male, 24. April 27, 1892. Dr. J. A. Hutchinson.

Depressed Fracture of Skull. Abscess of left frontal lobe of brain; staphylococcus pyogenes aureus infection.

24 (Finley)—Female, 50. April 28, 1892. Dr. Ross.

Abscess of Liver. Impacted gallstone; contraction of gall-bladder; dilatation of bile ducts; no jaundice.

25 (Finley)—Male, 60. May 11, 1892. Dr. Wilkins.

Pyæmia. Abscesses of neck near thyroid; cirrhosis of liver.

26 (Finley)—Male. May 11, 1892.

Sacculated Aneurysm of Descending Arch of Aorta. Rupture into œsophagus.

27 (Finley)—Male, 48. May 19, 1892. Dr. Ross.

Acute Right Lobar Pneumonia. Calcified adhesions left pleura; milk patches; fatty liver.

28 (Finley)—Female, 34. May 26, 1892. Dr. Finley.

Typhoid Fever. Perforation and Peritonitis. Cloudy swelling of liver and kidneys; spleen small 95 grms; commencing atheroma of aorta.

29 (Finley)—Male, 35. May 27, 1892. Dr. Ross.

Miliary Tuberculosis of Lungs. Caseous tubercles in epididymis and each lobe of prostate; sebaceous cyst of back.

30 (Finley)—Male, 33. May 31, 1892. Dr. Ross.

Angina Pectoris. Atheroma of aorta; atheroma and stenosis of coronary arteries; dilatation and fatty degeneration of heart.

31 (Finley)—Male, 28. June 2, 1892. Drs. Stewart and Ross.

Mitral Stenosis. Hypertrophy and dilatation of left auricle; cyanotic induration of lungs.

32 (Finley)—Male, 35. June 8, 1892. Dr. Armstrong.

SPINAL MENINGITIS AND COMPRESSION MYELITIS. Gummatous tumor of cord; gummata in testes, liver, spleen and lungs.

33 (Finley)—Male, 62. June 15, 1892. Dr. Ross.

PRIMARY CANCER IN BILE DUCTS, secondary in liver and glands of portal fissure, also in retro-abdominal and retro-thoracic glands; jaundice.

34 (Finley)—Female, 15. June 16, 1892. Dr. Wilkins.

TUBERCULOSIS OF BOTH LUNGS, WITH CAVITIES. Tuberculous salpingitis; tuberculous peritonitis and tuberculous ulceration of intestines; left pneumothorax.

35 (Finley)—Male, 66. June 19, 1892. Dr. Ross.

ADENOID CANCER OF STOMACH. Secondary cancer of liver; recent peritonitis with effusion; minute fibroid patch in heart; adhesions of bases of lungs.

36 (Finley)—Female, 34. June 21, 1892. Dr. Wilkins.

MILIARY ABSCESSES OF LIVER. Gallstones; dilated bile-ducts; contracted and thickened gall-bladder; old appendicitis.

37 (Finley)—Male, 41. June 21, 1892. Dr. Ross.

TUBERCULOSIS. Tuberculous caries of spine, lumbar region; psoas abscess (double caseous); miliary tuberculosis of lungs, kidneys, liver and spleen; tuberculous meningitis and pericarditis.

38 (Finley)—Male, 64. June 27, 1892. Dr. Bell.

ENLARGED PROSTATE; PYELITIS AND MULTIPLE ABSCESSES OF KIDNEYS. Superficial necrosis of prostate; hypertrophy of bladder.

39 (Finley)—Female, 71. June 28, 1892. Private.

HÆMORRHAGIC INFARCTION AND RUPTURE OF HEART. Sudden death. Calcareous degeneration of coronary arteries, with thrombosis; senile atrophy of kidneys; polypus of cervix uteri.

40 (Finley)—Female, 32. June 30, 1892. Drs. Buller and Stewart.

PHLEGMONOUS ERYSIPELAS OF LEFT LEG AND THIGH. Aortic stenosis; hypertrophy of left ventricle; atheroma of aorta; fibroid testicle (syphilis?).

41 (Finley)—Female, 5. July 3, 1893. Dr. Wilkins.

DIPHTHERIA of larynx and trachea; broncho-pneumonia; inflation of lungs; slight mitral endocarditis.

42 (Finley)—Female, 23. July 10, 1892. Private.

EMBOLI IN VENA CAVA AND PULMONARY ARTERIES. Thrombosis of left iliac vein; œdema of lungs; anæmia; recent pregnancy.

43 (Finley)—Male, 19. July 13, 1892. Dr. Ross.

TUBERCULOSIS. Tuberculous caries of dorsal vertebræ; tubercles of lungs, liver, kidneys and spinal membranes; compression myelitis.

44 (Finley)—Male, 48. July 19, 1892.

SUPPURATIVE NEPHRITIS. Stricture of urethra; slight hypertrophy of bladder; chronic cystitis; abscess of scrotum; fibroid testicles; emphysema; hypertrophy of heart; atheroma of aorta.

45 (Finley)—Female, 33. July 23, 1892. Dr. Wilkins.

ACUTE PERITONITIS and pleurisy; biliary calculi; perforation of gall-bladder; ovaries enlarged and fibroid.

46 (Finley)—Male, 11. July 28, 1892. Dr. Wilkins.

TUBERCULOSIS of mesenteric glands, intestines and lungs; acute mitral endocarditis.

47 (Finley)—Male, 23. Aug. 1, 1892. Dr. Shepherd.

APPENDICITIS. Pelvic abscess; laparotomy.

48 (Finley)—Female, 21. Aug. 2, 1892. Dr. Ross.

PHTHISIS. Large cavities at both apices; tuberculous ulcers of colon; chronic adhesive pelvic peritonitis.

49 (Finley)—Female, 50. Aug. 4, 1892. Dr. Finley.

GLIOMA R. OPTIC THALAMUS. Uterine fibroid; atrophy of organs.

50 (Finley)—Male, 31. Sept. 31, 1892. Dr. Stewart.

PULMONARY PHTHISIS OF BOTH LUNGS, WITH CAVITIES. Chronic pleurisy; serous pericarditis; atrophy of right shoulder muscles; suppurative arthritis right shoulder.

51 (Finley)—Female, 20. Sept. 4, 1892. Dr. Wilkins.

PYÆMIA. Typhoid fever; suppurative infarctive nephritis; dilatation of R. ureter; subinvolution of uterus; puerperal state; eleventh day after miscarriage (still-born 7 months); swelling of spleen with infarcts; parenchymatous degeneration of liver; acute R. pleurisy.

52 (Finley)—Female, 50. Sept. 5, 1892. Dr. Stewart.

BRONCHO-PNEUMONIA. Hypertrophy of heart with adhesive pericarditis; atheroma of aortic valves; atheroma of aorta.

53 (Finley)—Female, 28. Sept. 9, 1892. Dr. Ross.

CARBUNCLES; no other gross lesions.

54 (Finley)—Female, 48. Sept. 11, 1892. Dr. Shepherd.

MULTIPLE, ANEURYSMS OF SUPERIOR MESENTERIC ARTERY, WITH RUPTURE INTO PERITONEAL CAVITY. Dissecting aneurysm of abdominal aorta; aneurysm of right subclavian; early interstitial nephritis; hypertrophy and fibroid change of left ventricle.

55 (Finley)—Female, 58. Sept. 14, 1892. Dr. Armstrong.

STRANGULATED HERNIA. Hæmorrhagic infiltration of ileum at one point; atheroma of aortic valves.

56 (Lafleur)—Female, 3. Oct. 28, 1892.

SCARLATINA. Pseudomembranous pharyngitis, rhinitis and laryngitis; localized pneumonia of left lung; tuberculous adenitis bronchial glands; bilateral purulent otitis media.

57 (Lafleur)—Male, 3. Nov. 12, 1892.

DIPHTHERIA of tonsils, pharynx and uvula, larynx and trachea; broncho-pneumonia and lung inflation; p.m. invagination of small bowel.

58 (Lafleur)—Male, 43. Nov. 22, 1892.

CHRONIC INTERSTITIAL NEPHRITIS. Chronic right pleurisy; emphysema and œdema of both lungs; chronic gastritis; chronic purulent bronchitis; fatty liver.

59 (Adami)—Female. Nov. 23, 1892.

PYÆMIA. Retained suppurati ng placenta; pyæmic infarct lower lobe right lung; right empyæma; varicose veins.

60 (Adami)—Female, 32. Nov. 23, 1892.

NECROSIS (GANGRENE?) OF LUNG. Old pleurisy with adhesions; interstitial nephritis; dilatation and hypertrophy of heart with sclerosing endocarditis.

61 (Finley)—Male, 26. Dec. 3, 1892. Dr. Bell.

HÆMORRHAGIC PANCREATITIS. Fat necrosis; fatty liver; large firm kidneys; a few ounces of brownish coloured fluid in peritoneum; no peritonitis.

1893

1 to 10—Reports missing.

11 (Adami)—Male, 88. Jan. 23, 1893. Dr. Bell.

CARCINOMA OF PROSTATE extending to the bladder, where it presents characters of an alveolar sarcoma; dilated bladder; secondary cancer in left inguinal glands and (around common iliac arteries); retroperitoneal glands; hydronephrosis; hydrothorax and œdema of lungs; atheroma of aorta, carotids and cerebral arteries.

12 (Adami)—Male, 47. Feb. 10, 1893.

SOFTENING OF LEFT ASCENDING PARIETAL CONVOLUTION. Hypostatic congestion and atelectasis of lungs; fatty liver; thickening of aortic valves; fatty change of kidneys; bedsores.

13 (Adami)—PERNICIOUS ANÆMIA.

14 (Adami)—Male, 31. Feb. 22, 1893.

Chronic Fibrous Pachymeningitis near ascending parietal convolution (lower end) with adhesion to pia; heart enlarged and fatty; perihepatitis fibrosa. Paralysis L. leg.

15 (Adami)—Male, 35. Feb. 24, 1893. Dr. Stewart.

Arteriosclerosis. Hypertrophy and dilatation of heart; embolic infarct of left ventricle; interstitial nephritis; white infarcts of kidneys.

16 (Adami)—Female, 72. Feb. 8, 1893. Dr. Stewart.

Hypertrophy of Heart and Chronic Endocarditis of Mitral and Aortic Valves. Hypertrophic ostitis inner table of frontal bone; ivory exostosis of R. petrous bone; calcareous change of arteries at base of brain; hydrothorax; calcification of coronaries; congestion of stomach and intestines; rib indentations on liver; ovaries look cartilaginous; old fracture of R. 3rd, 4th and 5th ribs; œdema of lungs.

17 (Adami)—Female, 66. March 30, 1893. Dr. Molson.

Tumor of Corpus Callosum L. Frontal Region. Atheroma of abdominal aorta; aneurysmal dilatation of sinuses of valsalva; brown atrophy of heart.

18 (Adami)—Male, 71. April 3, 1893.

Pyelonephritis. Phosphatic concretions of bladder; general hypertrophy of prostate; perineal cystotomy; hypertrophy of heart; arteriosclerosis.

19 (Adami)—Male, 8 years. April 8, 1893.

Tuberculous Meningitis. Acute miliary tuberculosis; tuberculous mesenteric gland; miliary tubercles of spleen and liver and lungs; broncho-pneumonia.

20 (Adami)—Male, 37. April 11, 1893.

Acute Tubercular Broncho-Pneumonia of Right Lung. Symmetrical sclerosis of Gall's column; in lower dorsal region similar degeneration of crossed pyramidal tract; milk spots of heart; dilatation; cavity left apex; constriction anterior of right lobe of liver; tuberculous ulceration of intestines; slight atheroma of aorta; syphilitic gumma of cord.

21 (Adami)—Male, 21. April 13, 1893.

Aortic Incompetence. Chronic general adhesive pericarditis; dilatation and hypertrophy of heart; persistent foramen ovale; congestion and œdema of lungs; nutmeg liver; hogback kidneys; anasarca.

22 (Adami)—Female, 73. April 15, 1893.

Multiple Softening of Cortex of R. Hemisphere. Atheroma of cerebral arteries; brown atrophy of liver; accessory spleen; cirrhosis of kidneys; degeneration of supra-renals (gray mottled); dilated and atrophied stomach.

23 (Adami)—Female, 28. April 17, 1893.

MILIARY TUBERCULOSIS OF PIA AND CORTEX. Old phthisis both apices; large cavity of left upper lobe; acute tuberculous broncho-pneumonia; tuberculous right supra-renal; miliary tuberculosis of liver and kidneys.

24 (Adami)—Male, 25. May 1, 1893.

ACUTE PNEUMONIA. Old pneumonia of left upper lobe; gangrene; catarrhal jaundice.

25 (Adami)—Female, 55. May 3, 1893. Dr. Blackader.

PYONEPHROSIS. Left hydronephrosis with commencing pyonephrosis; right pyonephrosis with multiple abscesses and purulent perinephritis; cystitis; mixed urate and oxalate calculi; obstruction of ureters.

26 (Adami)—Female, 37. May 9, 1893.

WOUND INFECTION OF RIGHT ARM. Septicæmia; congestion of lower lobe of right lung; congestion of kidneys with parenchymatous nephritis and fatty degeneration; fatty liver; hyperostosis frontal bone.

27 (Adami)—Male, 44. May 16, 1893.

ACUTE PNEUMONIA of lower lobe of left lung advancing in upper lobe; acute left pleurisy; early interstitial nephritis with acute parenchymatous nephritis.

28 (Adami)—Male, 44. May 17, 1893. Dr. Blackader.

EMPHYSEMA. Old tuberculosis both apices; bronchitis; pulmonary œdema and congestion at bases; dilatation and hypertrophy right heart; patent foramen ovale; nutmeg liver; interstitial nephritis; congestion of upper part of intestines; congestion and hæmorrhage of pancreas.

29 (Adami)—Male, 24. May 31, 1893.

ACUTE LOBAR PNEUMONIA right upper and left lower lobes; acute fibrinous pleurisy (double); acute parenchymatous nephritis.

30 (Johnston)—Male, 60. May 31, 1893.

ACUTE LOBAR PNEUMONIA right lung. Disseminated broncho-pneumonia left lung; atheroma and dilatation of aorta; chronic interstitial myocarditis; acute catarrhal gastritis, colitis and cholecystitis; fatty liver; chronic interstitial nephritis; alcoholism.

30A (Adami)—Male, 3. June 5, 1893.

SCARLATINA. Abscess of neck; suppurative lymphangitis; acute parenchymatous nephritis; acute enlargement of spleen; acute pharyngitis; acute and chronic purulent right otitis media and interna; acute right mastoiditis.

31 (Adami)—Male, 50. June 22, 1893.

ANGINA LUDOVICI. Septicæmia; fatty degeneration of heart.

32 (Adami)—Male, 39. June 22, 1893.

HERNIA. Radical cure; external right iliac pricked and ligatured; wound remaining open and suppurating; marasmus.

33 (Adami)—Female, 52. June 24, 1893. Dr. Blackader.

BRONCHITIS AND EMPHYSEMA. Subacute duodenitis and jejunitis; cyst in brain; chronic adhesive pleurisy; perihepatitis; chronic tuberculosis right apex.

33A (Adami)—Male, 37. June 27, 1893.

ACUTE HÆMORRHAGIC PERICARDITIS. Chronic adhesive pericarditis; congestion and œdema of lungs; bedsores.

34 (Adami)—Female, 60. June 30, 1893.

EMPHYSEMA OF BOTH LUNGS. Chronic bronchitis; adhesions at apex and left base; fatty degeneration of heart with hypertrophy; atheroma of coronaries and aorta; granular kidneys; adhesions and calcareous deposit round left ovary; senile atrophy of intestinal walls; hæmorrhages of pancreas.

35 (Johnston)—Male, 33. July 10, 1893.

FATTY AND CIRRHOTIC LIVER. Jaundice; double hydrothorax chronic parenchymatous and interstitial nephritis; hypertrophy of left heart; chronic granular pharyngitis; broncho-pneumonia; œdema of lungs.

36 (Johnston)—Female, 3. July 13, 1893.

ACUTE TUBERCULOUS MENINGITIS. Chronic double otitis media (purulent); miliary tuberculosis of lungs, spleen and liver; tuberculous ulceration of colon; chronic tuberculosis of bronchial glands.

37 (Johnston)—Female, 50. July 13, 1893. Dr. Armstrong.

CHRONIC INTERSTITIAL NEPHRITIS. Acute catarrhal ulceration of colon; pigmentation of peritoneum; myoma uteri; adhesive pleurisy and pericarditis; chronic bronchitis; slight hypertrophy and dilatation of heart; anasarca.

38 (Johnston)—Male, 32. July 14, 1893. Dr. Lafleur.

ACUTE LOBAR PNEUMONIA, upper and lower lobes right lung. Imperfect separation of lobes; acute right fibrinous pleurisy; slight interstitial chronic hepatitis; slight cloudy swelling of liver and kidneys; acute enlargement of spleen; slight catarrhal colitis; gall-stone.

39 (Johnston)—Female, 18. July 23, 1893. Dr. Wilkins.

TYPHOID FEVER. Ulceration of ileum stage of healing; perforative appendicitis; suppurative perityphlitis with perforation into cæcum; acute septic pelvic peritonitis; chronic adhesive perimetritis; acute parenchymatous nephritis and hepatitis.

40 (Johnston)—Female, 32. July 26th, 1893.

GENERAL TUBERCULOSIS. Hæmorrhages of brain, meninges, stomach and intestines; acute hæmorrhagic tubercular peritonitis; acute tubercular pleurisy; hæmorrhage of myocardium; chronic tuberculous enteritis; acute hæmorrhagic cystitis; left pyelonephritis; cirrhosis of liver and kidneys; miliary tuberculosis of liver; solitary tubercle of brain. Purpura.

41 (Johnston)—Female, 42. July 28, 1893. Dr. Gardner.

PYOSALPINX. Laparotomy; hernia of wound; operation followed by constipation and diarrhœa; small abscess of abdominal wall; healed laparotomy incision; local adhesive peritonitis; chronic pelvic peritonitis with recto-vesical fistula receding from region of left fallopian tube; catarrhal cystitis and enteritis; fatty heart; cloudy swelling of liver and kidneys.

42 (Johnston)—Male, 53. July 28, 1893.

CARCINOMA OF RIGHT KIDNEY. Secondary cancer in liver and retroperitoneal glands; perihepatitis; cyst of liver; secondary cancer of left supra-renal; necrosis of right supra-renal; bronzing of skin with slight leucoderma; brown atrophy of heart.

43 (Johnston)—Female, 7. August 5, 1893. Dr. Spier.

TUBERCULOSIS OF BRAIN. Solitary tubercle of cerebellum; acute tubercular meningitis; chronic tuberculosis of bronchial glands.

44 (Johnston)—Female, 40. Aug. 7, 1893. Dr. Blackader.

FATTY AND CIRRHOTIC LIVER. Gallstones; acute necrosing mycotic enteritis; acute parenchymatous nephritis; jaundice.

45 (Johnston)—Male, 24. August 24, 1893. Dr. Armstrong.

CARIES OF LUMBAR AND SACRAL VERTEBRÆ. Sacro-iliac disease; retroperitoneal pelvic and psoas abscesses; acute miliary tuberculosis; tuberculous meningitis; chronic hyperplasia of spleen; chronic parenchymatous nephritis; nutmeg liver.

47 (Johnston)—Female, 20. August 28, 1893. Dr. Blackader.

OLD EMBOLIC SOFTENING OF CORPUS STRIATUM AND EXTERNAL CAPSULE. Recent embolism of left middle cerebral artery; chronic sclerotic and acute verrucose endocarditis of mitral valve with cyst in valve segment; brown induration of lungs; chronic hyperplasia of spleen with infarcts; cyanotic atrophy of liver; adhesive peritonitis; retroflexed uterus.

48 (Johnston)—Male, 41. Sept. 11, 1893.

PRIMARY CARCINOMA OF LEFT BRONCHUS. Secondary in bronchial glands, left lung and liver; subacute broncho-pneumonia; cancerous pleurisy and pericarditis; slight parenchymatous nephritis.

49 (Adami)—Female, 49. Sept. 15, 1893.

PHTHISIS. Chronic tuberculosis of right apex with cavity; indurative chronic tuberculosis of left apex; chronic interstitial nephritis diffuse; amyloid kidney; marantic thrombus renal veins; tuberculosis of intestines.

50 (Adami)—Female, 6. Sept. 27, 1893.

SCARLATINA. Enlarged glands of neck with purulent infiltration, extending round larynx and through connective tissue to skin; laryngeal ulcers; broncho-pneumonia with miliary abscesses of lung; parenchymatous nephritis; fatty degeneration of liver; enlarged mesenteric glands.

51 (Adami)—Male, 65. Oct. 5, 1893.

CARCINOMA OF LOWER END OF ŒSOPHAGUS AND CARDIAC END OF STOMACH. Ulceration and perforation; subdiaphragmatic abscess around œsophageal end of stomach; perforation and abscess between pericardium and diaphragm; acute pericarditis.

52 (Adami)—Female, 3. Oct. 9, 1893.

SCARLATINA. Granular pharyngitis; cervical lymphadinitis (non-suppurative); cloudy swelling of heart; clotting in cavæ and veins; slight bronchitis; acute parenchymatous nephritis; dura mater thick.

53 (Adami)—Male, 30. Oct. 18, 1893.

ACUTE MANIA. Lungs congested; bronchi full of mucus; no other anatomical changes.

54 (Adami)—Female, 31. Oct. 31, 1893.

ACUTE DIPHTHERITIC ENDOMETRITIS. Subinvolution of uterus; thrombo phlebitis of pelvic veins; pulmonary embolism with multiple pulmonary abscesses; tuberculous ulceration of larynx; tuberculosis of mesenteric glands; parenchymatous swelling of spleen, kidneys and liver.

55 (Adami)—Male, 62. Nov. 2, 1893.

PNEUMONIA. Gray hepatization right middle lobe; interstitial right upper lobe; œdema and congestion right lower lobe; chronic pleurisy with total right adhesions; extreme œdema left lung; hypertrophy and dilatation of heart; hydropericardium; left hydrothorax; chronic fibrinous perisplenitis; submucous rectal lipoma; cirrhosis of liver, early stage; atrophic cirrhosis of kidneys; old healed gastric ulcers; general arterial fibrosis.

56 (Adami)—Male, 35. Nov. 6, 1893.

ABSCESS OF FRONTAL SINUS; MILIARY TUBERCULOSIS. Acute rhinitis; congestion and œdema of lungs with old caseous tubercles and acute tubercles; acute parenchymatous nephritis; acute bronchitis; calcified mesenteric glands; slight tuberculosis of ntestines.

57 (Adami)—Male, 51. Nov. 15, 1893.

ACUTE PNEUMONIA. Creeping pneumonia approaching left upper lobe; gray hepatization spreading into left lower lobe; old adhesive left pleurisy, and recent acute double pleurisy; slight acute pericarditis; nephritis, mixed subacute, parenchymatous and interstitial.

58 (Adami)—Male, 47. Nov. 19, 1893. Dr. Stewart.

FIBRO SARCOMA lipomatodes of peritoneum and diaphragm; double sarcomatous pleurisy with effusion; interstitial myocarditis; chronic perihepatitis.

59 (Adami)—Male, 22. Nov. 20, 1893.

Dilatation of stomach; lymphatic enlargement of follicles of jejunum; cloudy swelling of kidneys; fatty liver. Death in convulsions, supposed cerebral syphilis.

60 (Adami)—Female, 30. Nov. 27, 1893.

SUBACUTE TUBERCULOSIS OF BOTH LUNGS. Ulceration of œsophagus (tuberculous); tuberculous ulcer of cæcum; amyloid kidneys and intestine; chronic adhesive pleurisy and peritonitis.

61 (Adami)—Female, 5. Nov. 28, 1893.

DIPHTHERIA of tonsils in stage of healing; acute glomerulo-nephritis; renal calculus; acute catarrhal gastro-enteritis; congestion of lungs; commencing broncho-pneumonia; abnormal lobulation of right lung.

62 (Adami)—Male, 45. Nov. 30, 1893.

CHRONIC INTERSTITIAL AND PARENCHYMATOUS NEPHRITIS. Acute pericarditis with effusion; hypertrophy and dilatation of heart; hydronephrosis and dilatation of ureters from the development of fibrous tissue outside base of bladder; cirrhotic and fatty liver.

63 (Adami)—Male, 33. Dec. 1, 1893.

CHRONIC PARENCHYMATOUS NEPHRITIS with extensive fatty degeneration; commencing interstitial nephritis; concentric hypertrophy of left ventricle; slight fatty liver; slight œdema of lungs; congestion of stomach.

64 (Adami)—Male, 47. Dec. 14, 1893.

ABSCESS OF OCCIPITAL LOBE. Cyst of left frontal lobe; Suppuration into lateral and third ventricles; purulent meningitis; calculi (multiple oxalic acid) in cyst of left kidney; Jacksonian epilepsy; trephining left parietal region; necrosing osteitis of parietal bone.

65 (Adami)—Female, 45. Dec. 16, 1893.

CHRONIC INTERSTITIAL NEPHRITIS. Chronic bronchitis with emphysema; hypertrophy of heart; chronic interstitial myocarditis; moveable left kidney with dilatation of ureter.

66 (Adami)—Male, 19. Dec. 27, 1893.

APPENDICITIS. Acute purulent peritonitis; fatty degeneration of heart; liver pale, mottled, fatty central degeneration; skin subicteroid; solitary ulcer of ileum; perforation and suture; amputation of appendix.

1894

1 (Adami)—Female, 11. Jan. 6, 1894. Dr. Finley.

LEUCHÆMIA; cirrhosis of liver; gastric and intestinal hæmorrhages; enlarged spleen; appendicitis (chronic catarrhal); leuchæmia of liver.

2 (Adami)—Male, 62. Jan. 17, 1894. Dr. Molson.

CEREBRAL SOFTENING. Cyst in right corpus striatum; intermeningeal hæmorrhage; chronic leptomeningitis; general arterio sclerosis; chronic interstitial nephritis; commencing red hepatization of left lung with œdema; acute bronchitis; dilatation and hypertrophy of heart; fibroid spleen.

3 (Martin)—Female, 31. Jan. 18, 1894.

LARYNGEAL DIPHTHERIA. Broncho-pneumonia; caseation of bronchial glands; caseous masses and tubercles in lungs.

4 (Adami)—Male, 19. Jan. 20, 1894. Dr. Bell.

TYPHOID FEVER WITH PERFORATION. Perforative peritonitis; purpuric hæmorrhages of skin; hæmorrhagic nephritis; hæmorrhage of retro-peritoneal glands; hæmorrhage of tongue; osteophytes along longitudinal sinus, inner surface of skull cap.

5 (Adami)—Male, 40. Jan. 26, 1894.

ACUTE MILIARY TUBERCULOSIS OF LEFT LUNG. Old apical tuberculosis calcified; apical calcified nodules of right lung; bronchitis; hæmorrhagic infarction of left lobe; submucous hæmorrhages; infarcts of kidneys; slight fatty degeneration and brown atrophy of heart; hypertrophy of ventricles; atheroma of aorta.

6 (Adami)—Male, 47. Jan. 30, 1894. Dr. Finley.

ACUTE FIBRINOUS PURULENT PERICARDITIS. Chronic tuberculosis of right upper lobe with cavity; putrid bronchitis; emphysema; broncho-pneumonia.

7 (Adami)—Male, 37. Feb. 18, 1894. Drs. Finley and Bell.

AMŒBIC ABSCESS OF LIVER. Cicatrix in colon; acute fibrino serous pericarditis; surgical operation opening of subdiaphragmatic abscess; chronic perisplenitis; old infarct; hogback kidney; acute parenchymatous nephritis; streptococcus infection.

8 (Adami)—Male, 65. Feb. 19, 1894. Dr. Finley.

HÆMORRHAGE OF RIGHT CORPUS STRIATUM. General arterio sclerosis and atheroma; hypertrophy and dilatation of heart; arterio sclerotic granular kidney; emphysema; bronchitis; broncho-pneumonia; cyst of right tunica vaginalis.

9 (Adami)—Male, 23. Feb. 19, 1894. Dr. Molson.

CHRONIC PULMONARY TUBERCULOSIS WITH CAVITIES OF BOTH LUNGS. Interstitial pneumonia; chronic pleurisy; caseation of bronchial glands; slight fatty liver; slight cloudy swelling of kidneys; accessory right supra renal.

10 (Martin)—Female, 19. Feb. 23, 1894. Maternity.

DIPHTHERITIC ENDOMETRITIS AND PHLEGMONOUS VAGINITIS. Hæmorrhagic infarction and miliary abscess of lung and liver; calcified nodules in liver; slight parenchymatous nephritis; slight fatty degeneration of heart; streptococcus infection.

11 (Martin)—Male, 34. Feb. 23, 1894. Dr. Finley.

CHRONIC PARENCHYMATOUS NEPHRITIS. Hydrothorax; hydropericardium; ascites and general anasarca; fatty liver; collapse and slight œdema of lung; thyroid inclusion; infarcts in spleen.

12 (Martin)—Male, 38. March 10, 1894. Dr. Finley.

GRAY HEPATIZATION OF RIGHT LUNG. Chronic parenchymatous nephritis.

13 (Adami)—Male, 61. March 15, 1894. Dr. Finley.

MULTIPLE TUBERCULOUS ABSCESSES OF BRAIN, in right ascending frontal region and ventricles; basilar meningitis; bronchitis; congestion and œdema of lungs; calcified tubercle at apex; suppurative tonsilitis; suppurative bronchial glands at root of right lung; atheroma; pneumococcus infection.

14 (Adami)—Female, 74. March 22, 1894. Dr. Finley.

HÆMORRHAGE IN RIGHT EXTERNAL CAPSULE. Atheroma of cerebral arteries; hypertrophy of heart; chronic interstitial myocarditis; chronic interstitial nephritis; chronic bronchitis and general emphysema.

15 (Adami)—Female, 26. May 3, 1894.

ACUTE SEPTICÆMIA; acute hæmorrhagic pleurisy and pericarditis; acute parenchymatous nephritis; acute myocarditis; cloudy swelling of liver.

16 (Adami)—Male, 64. April 4, 1894. Dr. Lafleur.

LARGE GRANULAR KIDNEYS; chronic tuberculosis, calcified apex; emphysema; hypertrophy and dilatation of heart; thickening of base of valves; fatty degeneration of heart muscle.

17 (Adami)—Male, 52. May 5, 1894.

TUBERCULOSIS. Caries of ribs.

18 (Adami)—Female, 75.

ATROPHIC CIRRHOSIS OF KIDNEYS. Thickening of heart valves; dilatation and brown atrophy; persistent foramen ovale; œdema of lungs; left hydrothorax; old intracapsular fracture of left femur.

19 (Adami)—Male, 68. May 30, 1894.

CIRRHOSIS OF LIVER. Icterus; enlarged spleen; chronic endarteritis; atheroma of coronaries, aorta and iliacs; moderate cirrhosis of kidneys; old apoplectic cysts in left external capsule and optic thalamus; atrophy of hemispheres; dilated ventricles.

20 (Adami)—Female, 34. June 1, 1894. Dr. Molson.

TUBERCULAR MENINGITIS. Tuberculosis left apex; tuberculosis of uterus; maceration of contained fœtus (7 mos.); tuberculous ulceration of ileum.

21 (Lafleur)—Male, 31. June, 6, 1894.

RECURRENT ENDOCARDITIS, mitral and tricuspid valves. Acute pericarditis; hypertrophy and dilatation of heart; atheroma of coronaries and middle sized arteries; tuberculous nodules at apices of lungs; tuberculosis of larynx; infarcts of spleen and kidneys.

21 a (Adami)—Female, 35. June 9, 1894. Dr. Lafleur.

FLEXURE OF LARGE INTESTINE with constriction and diverticula; old calcerous nodules at apices of lungs; ecchymoses of peritoneum; condylomata of vagina; chronic endometritis; fibro cystic ovary; patent foramen ovale.

22 (Adami)—Female, 39. June 12, 1894. Dr. Kirkpatrick.

STRICTURE OF RECTUM. Perforative peritonitis following operation.

23 (Williams)—Female, 35. June 18, 1894. Dr. Wilkins.

PULMONARY TURERCULOSIS WITH CAVITIES. Tuberculous ulceration of intestines; fatty liver with portal cirrhosis.

24 (Williams)—Female, 34. June 23, 1894. Dr. Alloway.

DOUBLE PYOSALPINX. Acute peritonitis; granular endometritis; small dermoid in right ovary.

25 (Williams)—Male, 23. July 3, 1894. Dr. Blackader.

ACUTE PERICARDITIS with effusion; caseous nodules in apex, calcified; mitral stenosis; chronic sclerotic endocarditis.

26 (Williams)—Male, 26. July 12, 1894. Dr. Wilkins.

HYPERTROPHY AND DILATATION OF HEART. Syphilitic endarteritis; bedsores; chronic perisplenitis with calcareous nodules.

27 (Williams)—Female, 56. July 26, 1894. Dr. Shepherd.

BILIARY CIRRHOSIS; nutmeg liver; cirrhosis of kidneys; aneurysm of cirrhosis splenic artery and calcified thrombus.

28 (Williams)—Female, 40. Aug. 7th, 1894.

TUBERCULOUS BASILAR MENINGITIS. Tuberculosis of lungs; general miliary tuberculosis.

29 (Williams)—Female, 38. Aug. 7, 1894. Dr. Wilkins.

LYMPHO-SARCOMA OF BRONCHIAL GLANDS. Compression of right bronchus and partial compression of trachea; acute right pleurisy.

30 (Adami)—Male, 32. Oct. 1894. Dr. Kirkpatrick.

SUPPURATIVE PACHY AND LEPTOMENINGITIS. Cellulitis of scalp and frontal region; enlarged spleen; fatty degeneration of liver and kidneys; cloudy swelling of muscles; suppurative ostitis and periostitis of frontal bone; no abscess of brain.

31 (Williams)—Female, 70. Oct. 21, 1894. Dr. Molson.

INTESTINAL OBSTRUCTION FROM CANCEROUS STENOSIS OF UPPER PART OF RECTUM. Submucous hæmorrhages of descending colon; chronic interstitial nephritis; arterio-sclerosis; brown atrophy of liver and heart; slight acute fibrinous peritonitis; retroperitoneal glands normal; preliminary operation, to lumbar cholotomy.

32 (Johnston)—Female, 38. Oct. 21, 1894. Dr. Finley.

ANEURYSM OF TRANSVERSE ARCH OF AORTA. Erosion of bodies of 4th and 5th dorsal vertebræ; compression of left bronchus and lung; ulcerative and bronchiectatic cavities of left lung, non-tuberculous with chronic adhesive pleurisy; chronic peritonitis and perihepatitis; chronic interstitial nephritis; chronic sclerotic mitral and aortic endocarditis; subperitoneal myoma of uterus.

33 (Williams)—Female, 17. Nov, 11, 1894. Dr. Armstrong.

APPENDICITIS. Laparotomy; acute septic peritonitis.

34 (Williams)—Male, 36. Nov. 14, 1894. Dr. Finley.

TUBERCULOSIS. Miliary tuberculosis and cheesy tubercles in both lungs; miliary tuberculosis of kidneys; cloudy swelling of kidneys; bronchopneumonia; old amputation of right leg, conical stump.

36 (Williams)—Female, 18. Nov. 18, 1894.

ACUTE LEFT PLEURISY; œdema of lungs; acute hyperplasia of spleen.

37 (Williams)—Male, 44. Nov. 23, 1894. Dr. Finley.

HYPERTROPHIC BILIARY CIRRHOSIS. Great enlargement of spleen; œdema of lungs; catarrhal gastritis; ascites; general anasarca; no obstruction of ducts.

38 (Williams)—Female, Nov. 24, 1894.

TYPHOID FEVER. Typhoid ulceration of ileum and colon; enlargement of spleen; hæmorrhagic infarction of lungs; small lymphomata in liver.

39 (Williams)—Female, 20. Nov. 26, 1894.

TYPHOID FEVER. Typhoid ulceration of intestines; acute enlargement of spleen; angioma of liver and calcified nodules.

40 (Williams)—Male, 32. Nov. 28, 1894. Dr. Molson.

CHRONIC ULCERATIVE PHTHISIS BOTH LUNGS. Chronic pleurisy tuberculous ulceration of ileum and colon.

41 (Williams)—Female, 19. Dec. 1, 1894. Drs. Finley and Armstrong.

WOUND OF CORONARY VEIN. Hæmorrhage (22 ℥) into pericardium; puncture of pericardium: acute pericarditis with effusion.

42 (Williams)—Male, 70. Dec. 10, 1894.

BRONCHO-PNEUMONIA. Putrid bronchitis and bronchiectasis; chronic interstitial nephritis; catarrhal enteritis; perforation of cavity into pleura with circumscribed adhesive pleurisy; no hydrothorax; anthracosis.

43 (Williams)—Female, 74. Dec. 16, 1894. Drs. Finley and Armstrong.

ERYSIPELAS. Abraison of chin; recent parenchymatous nephritis; fatty kidneys and liver; atheroma of heart valves and arteries with fatty degeneration.

44 (——) Female, 34. Dec. 18, 1894.

Report missing.

45 (Williams)—Female, 66. Dec. 19, 1894. Dr. Molson.

Fat infiltration of heart; moderate chronic interstitial nephritis; nutmeg liver, amyloid; large soft spleen; tuberculous ulceration? in colon; died comatose, brain not examined.

46 (Williams)—Female. Dec. 21, 1894. Dr. Armstrong.

Report missing.

1895

1 (Williams)—Male, 22. Jan. 14, 1895. Dr. Molson.

CHRONIC MITRAL ENDOCARDITIS WITH STENOSIS. Chronic interstitial nephritis; nutmeg liver; œdema of lungs.

2 (McKenzie)—Male, 50. Jan. 8, 1895. Dr. Finley.

CARCINOMA OF LIVER. Congestion and œdema of lungs; enlarged spleen; submucous hæmorrhages of bladder; chronic gastritis, with slaty pigmentation.

3 (Johnston)—Female, 26. Jan. 20, 1895. Dr. Molson.

HYPERTROPHY AND DILATATION of heart, with fatty degeneration; nutmeg liver; beefy kidneys; fæcal concretion in appendix.

4 (Williams)—Female, 33. Jan. 29, 1895. Dr. Alloway.

ACUTE SEPTIC PERITONITIS. Ruptured tubal pregnancy; laparotomy.

4 a (Johnston)—Male, 48. Feb. 18, 1895. Dr. Molson.

CHRONIC MYELITIS IN DORSAL REGION. Sloughing balanitis; bed sores.

5 (Johnston)—Female, 38. Feb. 19, 1895. Dr. Molson.

PUERPERAL SEPTICÆMIA? Papular dermatitis; old cicatrix of epiglottis; concentric hypertrophy of heart; acute hyperplasia of spleen; acute parenchymatous nephritis; chronic endometritis; uterine polyp; recent pregnancy; puerperal state; chronic gastritis; acute cloudy swelling of liver; acute bronchitis; pneumococcus infection; history of influenza.

(Johnston)—Male, 39. March 1, 1895. Dr. Finley.

ACUTE TUBERCULOUS MENINGITIS; enlarged heart; acute hæmorrhagic cystitis. No primary source of tubercle discovered.

7 (Johnston)—Female, 33. March 3, 1895. Dr. Armstrong.

ACUTE SEPTIC PERITONITIS. Pyosalpinx; laparatomy.

8 (Johnston)—Child, 2. March 2, 1895.

SEPTICÆMIA; purpura. Cause not discovered.

9 (Johnston)—Female, 27. March 8, 1895. Dr. Finley.

Syphilitic endarteritis of right sylvian artery, with thrombosis; softening of right internal capsule; slight cirrhotic kidney; brown atrophy of liver.

10 (Johnston)—Male, 12. March 9, 1895. Drs. Molson and Armstrong.

ACUTE SEPTIC MENINGITIS from otitis media. Purulent mastoiditis; trephining.

11 (Johnston)—Male, 74. March 18, 1895. Dr. Finley.

GLOBULAR HEART THROMBUS. Hypertrophy and dilatation of heart, with chronic interstitial myocarditis and parietal thrombi in left auricle; embolism of right popliteal artery; cirrhosis of liver; slight hypertrophy of prostate; embolism of mesenteric artery: gangrene of ileum.

12 (Johnston)—Male, 72. March 22, 1895. Dr. Armstrong.

ENLARGED PROSTATE, CASTRATION, PYÆMIA. Purulent infection of wound; operation—fistula of urethra in perineo; recto vesical fistula; (healed, leaving blind fistula of bladder and rectum); chronic purulent right vesiculitis; slight left hydronephrosis; hæmorrhoids; cloudy swelling of kidneys; induration of prostate with hypertrophy of middle lobe; chronic perihepatitis; enlarged spleen; obsolescent apical cavities; congestion and œdema of lower lobes of lungs.

13 (Johnston)—Female, 21. March 23, 1895. Dr. Finley.

VALVULAR HEART DISEASE. Hypertrophy and dilatation of heart; chronic stenosing mitral endocarditis; recurrent aortic endocarditis; parietal thrombosis in left ventricle; chronic enlargement of spleen; concretion in vermiform appendix.

14 (Johnston)—Female, 37. March 27, 1895. Dr. Finley.

SYPHILITIC ENDARTERITIS OF ANTERIOR AND MIDDLE CEREBRAL ARTERIES WITH THROMBOSIS. Diffuse sclerosis of brain; emphysema; brown atrophy of heart; elongated left lobe of liver.

15 (Johnston)—Female, 25. March 28, 1895. Dr. Finley.

ACUTE BRONCHO-PNEUMONIA. Acute fibrinous pleurisy; acute pericarditis with effusion; concentric hypertrophy of heart; acute mitral endocarditis; acute parenchymatous nephritis; cloudy swelling of liver.

16 (Johnston)—Male, 25. March 28, 1895. Dr. Finley.

ACUTE PLEURO PNEUMONIA, right upper and left lower lobes. Thickening and opacity of pia; sclerosis of brain; fibrous interstitial pneumonia and chronic pleuritis left upper lobe; concentric hypertrophy of left heart; chronic interstitial myocarditis; enlarged spleen; hogback kidney.

17 (Johnston)—Female, 50. March 29, 1895. Dr. Molson.

ACUTE GREY HEPATIZATION LEFT LOWER LOBE AND RIGHT MIDDLE LOBE. Chronic bronchitis and emphysema; recurrent mitral endocarditis, with stenosis; polypoid thrombus from mitral valve; atheroma of aorta; lemon colour of fat; anæmia; slight interstitial nephritis; subperitoneal myomata of uterus.

18 (Johnston)—Male, 60. March 29, 1895.

ACUTE PNEUMONIA. Acute pleurisy right lung; congestion and œdema left lung; acute sero-fibrinous pericarditis; hypertrophy of heart; chronic mitral and aortic endocarditis; p.m. gastro-malacia; hypertrophy of adrenal; slight double hydrocele; Liebermeister grooves of liver.

19 (Johnston)—Female. 31. April 4, 1895. Dr. Armstrong.

GENERAL SEPTICÆMIA. Extraction of teeth; gangrenous stomatitis; sub-cutaneous ecchymosis: streptococcus infection; anterior extremities of ribs twisted; acute hæmorrhagic gastritis; acute colitis; acute nephritis papillaris and acute double hæmorrhagic pyelitis; cloudy swelling of liver and kidneys; enlargement of uterus; lacing lobe of liver; acute hyperplasia of spleen; accessory spleen.

20 (Johnston)—Male, 61. April 7, 1895. Dr. Finley.

CHRONIC INTERSTITIAL NEPHRITIS. Anæmia; dropsy; double hydrothorax; hydropericardium; dilatation and hypertrophy of heart; slight interstitial myocarditis; brown atrophy of fibres; no fatty change in heart; dilatation of aorta; intense œdema of lungs; emphysema; old caseous mass at right apex; broncho-pneumonia; hypertrophy of right and middle lobes of prostate, with adenoma; lemon-yellow skin; hyperplasia of bone marrow.

21 (Johnston)—Female, 50. April 23, 1895. Dr. Blackader.

CHRONIC ULCERATIVE PHTHISIS right upper lobe. Ochre coloured fat; bifid ensiform; acute tuberculosis of lower and middle lobes; inflation of right lung; chlorosis of aorta; hyperplasia; brown atrophy of heart; nutmeg liver.

22 (Johnston)—Female, 24. April 26, 1895..

ACUTE SEPTIC PERITONITIS. Laparotomy; old caseous nodule in mesentery; menstrual state; cyst of right ovary.

23 (——)—Male, 27. April 26, 1895. Dr. Blackader.

Notes not entered.

24 (Williams)—Male, 41. April 30, 1895. Dr. Wilkins.

ACUTE CROUPOUS PLEURO-PNEUMONIA OF LEFT LUNG. Double left ureter; aneurysm of left renal artery; acute degenerative parenchymatous nephritis; pneumococcus infection.

25 (Williams)—Male, 60. May 18, 1895. Dr. Blackader.

Recurrent Endocarditis with Mitral Stenosis. Bedsore; œdema of feet; ascites; chronic fibrous perihepatitis; cirrhosis of liver; thrombosis of portal vein; left hydrothorax; brown induration and œdema of lungs; emphysema; hypertrophy of heart.

26 (Johnston)—Male, 53. May 19, 1895. Dr. Wilkins.

Dissecting Broncho-Pneumonia with Necrosing Pleurisy. Chronic putrid bronchitis and bronchiectasis; localized left empyæma gangrenosa; chronic interstitial nephritis; enlargement of prostate.

27 (Williams)—Male, 49. May 20, 1895. Dr. Wilkins.

Chronic Ulcerative Phthisis of Both Lungs. Universal pleuritic adhesions; chronic interstitial and acute parenchymatous nephritis; chronic interstitial hepatitis; fibrons perihepatitis; ascites.

28 (Johnston)—Male, 21. May 21, 1895.

Acute Tuberculous Meningitis. Tuberculosis of thoracic duct. caseous masses in bronchial glands; miliary tuberculosis of kidney; acute broncho pneumonia; recurrent endocarditis.

29 (Johnston)—Male, 2½. May 21, 1895. Dr. Wilkins.

Tuberculous Meningitis. Solitary tubercle of corpus collosum; miliary tuberculosis of lungs; caseous tubercles of bronchial and mesenteric glands.

30 (Johnston)—Female, 39. May 27, 1895. Dr. Blackader.

Acute Tuberculous Meningitis. Acute miliary tuberculosis of lungs; acute parenchymatious nephritis.

31 (Williams)—Female, 55. May 31, 1895. Dr. Wilkins.

Villous (Papillary) Cancer of Uterus, secondary in bladder; recto-vesico vaginal fistula; recurrent mitral endocarditis; hæmorrhagic infarction and brown induration of lungs; chronic interstitial nephritis; secondary cancer of inguinal glands.

32 (Johnston)—Female, 35. May 25, 1895.

Carbolic Acid Poisoning. Tanning of stomach and adjacent organs; sub-pleural ecchymosis; hæmatoma of right ovary; slight granular kidneys.

33 (Johnston)—Male, 40. May 24, 1895.

Laudanum Poisoning. Permanganate solution in lungs; œdema of glottis.

34 (Johnston)—Female, 2½. June 6, 1895. Dr. Hutchison.

Acute Tuberculous Meningitis. Tuberculous arthritis left hip joint with cold abscess; left hydronephrosis.

35 (Johnston)—Male, 19. June 10, 1895. Dr. Blackader.

CHRONIC PARENCHYMATOUS NEPHRITIS. General anasarca; old excision of elbow joint; caries of both elbow joints; atelectasis and compression of left lung; left hydrothorax; left acute pleuritis; diplococcus infection; obsolescent tubercle R apex; milk patch of heart; ascites and acute sero-fibrinous peritonitis; chronic catarrhal gastritis.

36 (McKenzie)—Female, 24. June 21, 1895. Dr. Shepherd.

FRACTURE OF SKULL, ribs and chavicle; rupture of spleen; hæmorrhage into peritoneum; gestation 5th month; calcified nodules of spleen and liver capsules.

37 (Johnston)—Female, 60. June 24, 1895. Dr. Hutchison.

SLIGHT BRONCHO-PNEUMONIA. Old fracture through head of tibia; old cyst in left frontal lobe of brain; senile granular kidneys; œdema of lungs; calcified plate in heart wall; chronic local adhesive peritonitis; old scar in upper part of rectum; obsolete apical cavities.

38 (Johnston)—Male, 30. July 4, 1895. Dr. Shepherd.

RUPTURE OF LIVER; LACERATION OF LEFT LUNG. Compound fracture of left leg; fracture of lower end of R. tibia; laceration of scalp; ecchymosis of serosa of ascending colon; fracture of ribs; pleuritic adhesions; hæmorrhage into lung tissue; laceration of R. kidney; fracture of transverse process of lumbar vertebra.

39 (Johnston)—Male, 65. July 7, 1895. Dr. Blackader.

ACUTE BRONCHO-PNEUMONIA. Acute pleurisy; hypertrophy of heart; atheroma of coronaries; recurrent aortic endocarditis; emphysema; cheesy nodule in spleen; chronic interstitial nephritis with retention cysts; enlarged prostate; cirrhotic and fatty liver; œdema of pia.

40 (Johnston)—Male, 53. July 8, 1895.

CRUSH OF CHEST. Multiple contusions; laceration of face; fracture of alveolar process; fracture of ribs; left hæmothorax; laceration of pericardium and heart; laceration of spleen.

41 (Johnston)—Male, 27. July 13th, 1895. Dr. Hutchison.

PERFORATED APPENDIX. Acute septic peritonitis (slight); septic perityphlitis; abscess of mesenteric glands; slaty pigmentation of Peyer's patches; acute septic pleurisy (slight); œdema and congestion of lungs; food in bronchi; cloudy swelling of spleen and kidneys.

42 (Johnston)—Male, 25. July 13th, 1895. Dr. Armstrong.

INTESTINAL OBSTRUCTION. Chronic adhesive fibrinous peritonitis; resection of intestines.

43 (Johnston)—Female, 65. July 15th, 1895. Drs. Wilkins and Kirkpatrick.

CHRONIC INTERSTITIAL AND ACUTE PARENCHYMATOUS NEPHRITIS. Chronic adhesive pericarditis; chronic mitral endocarditis with stenosis; fatty degeneration of heart.

44 (Williams)—Female, 44. July 17th, 1895. Dr. Blackader.

TUBERCULOSIS WITH CAVITIES IN BOTH LUNGS. Ochre coloured fat; chronic mitral endocarditis with stenosis; tuberculous mesenteric glands; chronic interstitial nephritis; nutmeg liver with tubercles; healed tuberculous ulcer of intestines; subperitoneal myomata uteri; amyloid kidney (slight).

45 (Williams)—Male, 25. July 17th, 1895. Dr. Blackader.

ACUTE LOBAR PNEUMONIA. Left lower, right lower and middle lobes; double acute fibrinous pleurisy; obsolete tubercles at apex; enlarged spleen; fatty liver.

46 (Williams)—Male, 45. July 18th, 1895. Dr. Wilkins.

PHLEGMONOUS ERYSIPELAS of right leg; bubo in right groin; cloudy swelling of heart and kidneys; old apical tuberculosis; nutmeg liver; atheroma of aortic valves and coronaries; streptococcus infection.

47 (Johnston)—Male, 20. July 19th, 1895. Dr. Kirkpatrick.

APPENDICITIS; ACUTE SEPTIC PERITONITIS. Mixed infection B. coli and streptococci; sinus from behind cæcum to left side of pelvis; brown atrophy of liver; cloudy swelling of kidney.

48 (Williams)—Female, 50. July 21st, 1895. Dr. Alloway.

TUBERCULOUS SALPYNGITIS; ACUTE MILIARY TUBERCULOSIS of lungs and spleen. Removal of appendages; hypertrophy of heart; slight fatty degeneration; chronic interstitial and parenchymatous nephritis; fatty liver and kidney.

49 (Johnston)—Male, 22. July 22nd, 1895. House of Refuge.

GENERAL TUBERCULOSIS. Emaciation; anasarca; large caseous tubercles; tuberculous periostitis of ribs; tuberculous caries of dorsal vertebræ; prevertebral abscess; acute pneumonia left upper lobe; œdema and slight pneumonia of right lung; fatty degeneration of heart; tubercles in spleen: acute parenchymatous nephritis; tuberculous infarct of kidney; œdema of rectum; caseous mass in pancreas; conglomerate tubercles of liver: tuberculous inguinal glands; old bullet wound right temple, perforating skull cap, bullet in situ; exostosis of right temporal bone; softening of right motor area of cortex and subjacent white substance.

50 (Johnston)—Female, 22. July 23rd, 1895. Dr. Alloway.

ADENOID CANCER OF SIGMOID FLEXURE. Secondary in omentum; intestinal obstruction and intestinal hæmorrhage; subacute adhesive peritonitis; distention of ileum; hæmorrhagic colitis; dysentery; P. M. gastro malacia; cloudy swelling of kidney.

51 (McKenzie)—Male, 68. July 25th, 1895. Dr. Blackader.

MELANOTIC SARCOMA left foot; secondary in inguinal glands; multiple cutaneous sarcoma; secondary growths in liver, heart, stomach and pancreas; jaundice; anasarca.

52 (John-ton)—Male, 23. August 14th, 1895. Dr. Wilkins.

Abscess and Phlegmon of Prostate. Acute septic broncho-pneumonia; phlegmon of wall of bladder; chronic enlargement of spleen; solitary tubercle of mesentery; staphylococcus infection.

53 (Williams)—Female, 27. August 18th, 1895. Dr. Blackader.

Typhoid. Obsolete tubercle of lungs; subinvolution of uterus; ulceration of intestines; enlarged spleen; cloudy swelling of kidneys.

54 (Johnston)—Female, 3. August 23rd, 1895. Dr. Armstrong.

Tuberculous Meningitis. Acute miliary tuberculosis; granulations of ependyma; chronic tuberculosis of liver, lungs and kidneys; cyst of broad ligament.

55 (Johnston)—Male, 23. August 30th, 1895. Dr. Shepherd.

Crush of Chest. Laceration of cheek; fracture of alveolar process of left superior maxilla; interstitial emphysema; separation of both sterno-clavicular joints and cartilages of both first ribs; left hæmothorax; laceration of lungs with congestion and œdema; ecchymoses of larnyx; chronic interstitial nephritis; enlarged intestinal follicles; hæmorrhage into right tympanic cavity; laceration of tympanum.

56 (Johnston)—Male, 28. Sept. 7th, 1895. Dr. Wilkins.

Hæmorrhage of Brain from Left Sylvian Artery. Internal hæmorrhagic pachymeningitis; hæmorrhage external ventricle; old spot of softening in right internal capsule; hyperæmia of lungs; large firm spleen; atheroma of aorta.

57 (Johnston)—Male, 70. Sept. 11, 1895. Dr. Hutchison.

Crush of Chest. Trolley car accident. Right hæmothorax and hæmopericardium; laceration of right lung; fracture of ribs; hæmorrhage of lesser omentum; gallstones; abnormal liver.

59 (Johnston)—Male, 88. Sept 15, 1895.

Fracture of Skull with Meningeal Hæmorrhage. Run over by butcher's cart. Contusion right side of head; hæmatoma beneath scalp; osteo sarcoma of orbital plate; contusion of brain; atheroma of cerebral arteries with aneurysms; fatty degeneration of vessels of brain; atheroma of aortic valves; hypertrophy and brown atrophy of heart; granular kidneys; brown atrophy of liver.

60 (Johnston)—Male, 9. Sept. 16, 1895.

Fracture of Left Femur. Multiple contusions; fat embolism of lungs.

61 (Johnston)—Male, 36. Sept. 16, 1895.

Plumbism. Chronic alcoholism; hypertrophy of heart; chronic interstitial nephritis; fatty and cirrhotic liver; general arterio-sclerosis; congestion and œdema of lungs; œdema of larynx.

62 (Johnston)—Male, 24. Sept. 16, 1895. Drs. Wilkins and Lafleur.
TYPHOID FEVER. Typhoid ulcers of appendix; congestion of brain; ochre coloured fat; chronic mitral endocarditis; congestion of lungs with apoplexy: acute parenchymatous nephritis and hepatitis; swelling of bronchial glands.

63 (Johnston)—Male, 71. Sept. 21, 1895. Dr. Shepherd.
REVOLVER WOUND OF RIGHT TEMPLE. Trephining; contusion of dura and adjacent part of brain; powder grains in brain; slight nutmeg liver; acute sclerotic kidney; anthracosis; acute broncho-pneumonia; milk patch of heart; atheroma of valves. Suicide.

64 (Johnston)—Female, 6. Sept. 23, 1895. Dr. Blackader.
TUBERCULOUS ENTEROCOLITIS AND PERFORATIVE PERITONITIS. Tuberculous mesenteric glands; tuberculous salpingitis; ulcerative tuberculosis both lungs; tuberculous bronchial glands; fatty liver.

65 (Johnston)—Male, 23. Sept. 24, 1895. Dr. Armstrong.
LARYNGEAL DIPHTHERIA. Phlegmon of neck: tracheotomy; diphtheritic bronchitis; acute parenchymatous nephritis and hepatitis; chronic mitral endocarditis; congestion and œdema of lungs (asphyxia); sub-endocardial ecchymoses.

66 (Johnston)—Male, 53. Sept. 26, 1895. Dr. Wilkins.
PERNICIOUS ANÆMIA. Dilatation and fatty degeneration of heart; chronic pericarditis; emanciation; sub-icterus; atheroma of coronaries; emphysema: œdema of lungs; enlargement of spleen; chronic proliferative gastritis with polypoid outgrowth; slight interstitial nephritis; brown atrophy of liver; iron reaction; fatty degeneration of the central zones; slight lymphoid hyperplasia of red marrow.

67 (Johnston)—New born child. September 26th, 1895.
MUCUS IN TRACHEA. Ecchymosis of pleura and thymus.

68a (Johnston)—Male. September 28th, 1895. Dr. Armstrong.
FRACTURE OF RIGHT CLAVICLE AND STERNUM. Epithelioma of penis and paraphimosis; ulcerative balanitis; double hæmothorax; milk patch; slight atheroma of aorta; chronic interstitial and parenchymatous nephritis.

68 (Johnston)—Male, 28. September 30th, 1895. Dr. Armstrong.
STRANGULATION OF INTESTINES. Chronic adhesive peritonitis; resection of intestines; hyperplasia of spleen; follicular ulceration of rectum; cryptorchidismus; congestion and apoplexy of lungs.

69 (Johnston)—Male, 27. October 4th, 1895. Dr. Armstrong.
TYPHOID FEVER. Perforation; suture of intestine; acute septic peritonitis; typhoid ulceration; follicular ulcers of colon with induration; acute hyperplasia of spleen; slaty pigmentation of rectum; fatty degeneration of intima of coronaries; œdema of lungs; subarachnoid œdema; fibrinous nodules in pia; chronic leptomeningitis; pure culture of typhoid bacilli from spleen.

70 (Johnston)—Male, 68. October 10th, 1895. Dr. Lafleur.

ACUTE BRONCHO PNEUMONIA. Chronic bronchitis and emphysema; interstitial nephritis; brown induration of lungs; slight cirrhosis of liver, with dilatation of central veins.

71 (Johnston)—Male, 33. October 15th, 1895. Dr. Finley.

SYPHILITIC GUMMA OF HEART WALL AND VALVES. Chronic sclerotic endocarditis; gumma of testis; hypertrophy and dilatation of heart; ascites; arteriosclerosis; brown induration of lungs; diffuse interstitial nephritis; acute and chronic gastritis; red atrophy of liver, with chronic diffuse interstitial hepatitis and gumma; sclerosis of pancreas.

92 (Johnston)—Male, 21.

TYPHOID FEVER. Supposed injury of abdomen. Typhoid ulceration of intestine, early stage; ascarides; enlargement of mesenteric glands and spleen; subpleural ecchymosis; cloudy swelling of kidneys and liver; slight atheroma of aorta; acute bronchitis; caseous and calcareous bronchial glands; typhoid culture from spleen and lungs.

73 (Johnston)—Male, 39. Dr. Armstrong.

APPENDICITIS—Operation. Septic thrombophlebitis of portal and mesenteric veins; multiple abscesses of liver; interstitial nephritis; round ulcer of stomach; healed typhoid ulcers of intestines; old tuberculosis of lungs; slaty pigmentation, congestion and œdema of lungs; mixed infection by intestinal bacteria.

74 (Johnston)—Male, 55. October 26th, 1895. Dr. Hutchison.

PRIMARY EPITHELIOMA OF BLADDER. Calculus of bladder; suprapubic cystotomy; secondary growth in vena cava, renal veins, lumbar vertebræ, heart, lungs, liver, pancreas and doudenum; cancerous thrombosis of vena cava inferior; chronic mitral and tricuspid endocarditis; round ulcer of duodenum; diphtheritic pyelonephrosis; scar of prepuce.

75 (Johnston)—Female, 49. October 29th, 1895. Dr. Finley.

PUERPERAL FEVER. Laceration of vagina; pelvic abscess; septic endometritis and phlebitis; subinvolution; omental hernia; chronic adhesive peritonitis; hydropericardium; hypertrophy and dilatation of heart; slight fibroid endocarditis; acute parenchymatous nephritis and hepatitis; thrombosis and abscess of left innominate and jugular veins; streptococcus infection.

76 (Johnston)—Male, 72. October 29th, 1895. Dr. Kirkpatrick.

FRACTURE OF RIGHT FEMUR. Multiple bruises; hypertrophy of heart slight fibroid palillary myocarditis; marginal emphysema; granular (contracted) kidneys; slight double hydrocele (senile); alcoholism gallstones; cause of death: shock following fracture.

77 (Johnston)—Male, 5. October 30th. Dr. Molson.

CARIES OF DORSAL VERTEBRÆ. Tuberculous peritonitis; prevertebral abscess and sinus into cavity of right lung; abscess of spinal dura; transverse compression myelitis with ascending and descending degeneration; thrombosis and caseous infection of cerebral sinuses and veins; dorsal kyphosis; bedsores; bronchitis; disseminated tubercles of pia; caseous mesenteric glands; right caseous pneumonia; caseous bronchial glands.

78 a (Johnston)—Male, 12. November 7th, 1895. Dr. Gardner.

SUPPURATIVE RIGHT OTITIS MEDIA. Right mastoiditis; septic thrombosis of right lateral sinus and jugular; right multiple necrosing pneumonia with putrid right empyæma; chalky mass in lower lobe of left lung; enlargement of spleen.

78 (Johnston)—Female, 28. November 4th, 1895. Dr. Lockhart.

PAPILLOMATOUS CYSTOMA OF OVARY; ACUTE PERITONITIS. Old retroperitoneal hematocele, calcified; laparotomy; slight cloudy swelling of kidneys; old partial obliterative appendicitis.

79 (Johnston)—Male, 54. Nov. 12, 1895. Dr. Finley.

ACUTE LOBAR PNEUMONIA. Aneurysm of ascending arch of aorta; apical scars and tubercles; atheroma of heart valves; cyanotic induration of kidney; nutmeg liver; chronic enlargement of spleen.

80 (Johnston)—Male, 20. Nov. 13, 1895. Dr. Molson.

PHTHISIS. Chronic ulcerative tuberculosis of lung and larynx; tuberculosis of appendix; mitral endocarditis and stenosis; atheroma of coronaries; acute pneumonia with multiple cavities; chronic bronchitis emphysema; acute tuberculous pleurisy; tuberculous perichondritis of larynx; incipient tuberculosis of colon; ascarides; brown atrophy of liver; thickening of pia.

81 (Johnston)—Male, 35. Nov. 14, 1895. Dr. Kirkpatrick.

CANCER OF PYLORUS (scirrhous); laparotomy; slight mitral sclerosis; slight fibrous myocarditis; hyperæmia and œdema of lungs; adenoma of left adrenal; sclerosis of pia.

82 (Johnston)—Male, 50. Nov. 18, 1895. Dr. Kirkpatrick.

CRUSH OF CHEST. Elevator accident; rupture of diaphragm and hernia of stomach and spleen; no hæmorrhage; arteriosclerosis; atheroma of pulmonary artery; œdema of lungs; fracture of ribs; interstitial nephritis.

83 (Johnston)—Male, 22. Nov. 18, 1895. Dr. Molson.

HÆMORRHAGE INTO RIGHT INTERNAL CAPSULE. Chronic interstitial nephritis; hypertrophy and dilatation of heart; atheroma of arteries; arterio sclerosis; ecchymosis of stomach; sclerosis of pulmonary arteries; granular pharyngitis; pulmonary apoplexy.

84 (Johnston)—Male, 16. Nov. 20, 1895. Dr. Armstrong.

CRUSH OF CHEST AND ABDOMEN. Rupture of spleen and displacement of fragments; fracture of left femur; separation of epiphysis of left tibia; greenstick fracture of left ulna; multiple contusions; hæmorrhage into peritoneum; two accessory spleens; functional hypertrophy of heart; ecchymosis of pelvis of kidneys; congestion and ecchymosis of lungs.

85 (Johnston)—Male, 50. Nov. 22, 1895. Dr. Molson.

PARIS GREEN POISONING. Sesquioxide of iron in bronchi; sclerosis of tricuspid and mitral valves; brown atrophy of heart; arterio sclerosis; brown ecchymosis beneath pleura; ecchymosis of spleen; acute catarrhal gastritis with œdema and ecchymosis; œdema of intestinal mucosa; absence of fœtor; Paris green in stomach and intestines; chronic interstitial nephritis with cyst; slight fibrous epididymitis; hypertrophy of lateral lobes of prostate: slight fatty and cirrhotic liver.

86 (Johnston)—Female, 72. Nov. 25, 1895. Dr. Finley.

THROMBOSIS OF LEFT SYLVIAN ARTERY. White softening of brain; arterio sclerosis; anasarca; chondromalacia; hypertrophy, dilatation and brown atrophy of heart; sclerosis of endocardium and coronaries; subendocardial lipoma; chronic bronchitis; indurative peribronchitis; acute capillary bronchitis and broncho-pneumonia; chronic interstitial nephritis; pneumococcus infection.

87 (Johnston)—Male, 21. November 28th, 1895. Dr. Finley.

CHRONIC TUBERCULOUS PERITONITIS. PERFORATIVE APPENDICITIS. Tuberculous meningitis; left sero-fibrinous pleurisy; right dry tuberculous pleurisy; atrophy of heart; miliary tuberculosis of spleen, liver and kidneys; chronic catarrhal gastritis; recent softening of fornix; intestine free from tuberculosis. Mania.

88 (Johnston)—Male, 17. November 30th, 1895. Dr. Kirkpatrick.

RUPTURE OF LIVER. Hæmorrhage into peritoneum from branch of portal vein; acute emphysema and pulmonary apoplexy; food in bronchi; persistence of thymus gland.

89 (Jamieson)—Male, 25. December 1st, 1895. Dr. Molson.

CHRONIC ULCERATIVE PHTHISIS. Milk patch of heart; hypertrophy of heart; arteriosclerosis; aortic incompetence; aortic and mitral sclerotic endocarditis; nutmeg liver.

90 (Johnston)—Female, 4. December 3rd, 1895. Dr. Kirkpatrick.

BURN OF SKIN. Retro peritoneal hernia.

91 (Johnston)—Male, 33. December 3rd, 1895. Dr. Finley.

HYPERTROPHIC BILIARY CIRRHOSIS OF LIVER. Jaundice ; enlargement of spleen, 1930 grams ; anasarca ; chronic adhesive peritonitis ; dilatation of collateral veins ; slight thickening of heart valves ; subacute parenchymatous nephritis ; slaty pigmentation of rectum ; corpora amylacea of prostate ; follicular ulceration of ileum ; slaty pigmentation of solitary follicles.

92 (Johnston)—Male, 30. December 30th, 1895. Dr. Shepherd.

TUBERCULOUS PYELONEPHROSIS. Tuberculosis R. ureter. Nephrotomy Caseous tuberculosis of R. testicle, and miliary tuberculosis of L. testicle ; general acute miliary tuberculosis ; tuberculous meningitis ; bony anchylosis of both knees and right hip ; tuberculous sinus in right flank following nephrotomy localized miliary tuberculosis of peritoneum ; false diverticula coli ; slight mitral sclerosis ; brown atrophy of heart ; subpleural ecchymosis ; miliary tubercles in spleen, liver and kidneys.

93 (Johnston)--Male, 22. Dec. 7, 1895. Dr. Finley.

TYPHOID FEVER. Ecchymosis of mucosa of gall bladder ; local acute simple peritonitis ; staphylococcus aureus infection ; slight atheroma of mitral and aortic valves ; slight atheroma of coronaries ; acute bronchitis ; acute hyperplasia of spleen ; cloudy swelling of kidneys and liver ; typhoid ulceration of ileum and colon with adherent sloughs ; acute hyperplasia of mesenteric glands. Typhoid culture from spleen.

94 (Johnston)—Male, 28. Dec. 11, 1895. Dr. Molson.

CHRONIC SUPPURATIVE PYELONEPHRITIS. Calcified thrombus in pulmonary artery ; abscess of pancreas ; suppurative perinephritis ; psoas abscess ; general amyloid ; bifid cartilage of 3rd rib ; bedsores ; left pyelonephrosis (early) ; amyloid of kidneys, spleen, liver, adrenals, intestines, bladder, stomach and gallbladder; lacteal cysts in wall of intestines; amyloid of pancreas ; calcification of mesenteric arteries ; brown atrophy of heart ; black pigmentation of pia ; B. coli infection.

95 (Johnston)—Female, 14. Dec. 11, 1895. Dr. Kirkpatrick.

ACUTE SEPTIC FIBRINO-PURULENT PERITONITIS. Menstruation ; laparotomy ; corpus luteum ; hyperæmia of lung ; streptococcus infection ; (history of sore throat) ; no local cause.

96 (Johnston)—Male, 63. Dec. 16, 1895. Drs. Molson and Wilkins.

CHRONIC TUBERCULOUS FIBRO-CASEOUS PERICARDITIS. Chronic tuberculous peritonitis ; tuberculous pleurisy with effusion ; chronic bronchitis ; collapse induration of lung ; chronic catarrhal gastritis ; villous papilloma of stomach ; caseous tubercle mesenteric glands ; tubercles in capsule of spleen ; slight chronic interstitial nephritis ; enlarged prostate ; corpora amylacea ; nutmeg liver ; accessory thyroid calcareous plates in dura.

97 (Jamieson)—Female, 60. Dec. 19, 1895. Dr. Alloway.

COLLOID ADENOCARCINOMA OF SIGMOID FLEXURE. Acute septic peritonitis; secondary colloid cancer of omentum and peritoneum; laparotomy; chronic glomerulo-nephritis; myomata and secondary cancer of uterus; cystic ovaritis with hæmatoma.

98 (Jamieson)—Male, 23. Dec. 19, 1895. Dr. Molson.

CHRONIC PHTHISIS WITH CAVITIES. Hydropericardium; milk patches.

99 (Jamieson)—Male, 36. Dec. 22, 1895. Dr. Armstrong.

CRUSH OF R. LEG. Railway accident. Hæmorrhage; amputation; obsolete tubercles in lungs; slight emphysema; thickening of edges of aortic and mitral valves; atheroma of aorta.

NUMBER OF AUTOPSIES RECORDED.	1883	1884	1885	1886	1887	1888	1889	1890	1891	1892	1893	1894	1895	Average.
Total in each year	80	47	67	74	96	90	75	69	49	61	66	46	101	71·7
Private cases	9	0	3	10	9	4	8	10	0	6	0	0	0	....
Hospital cases...	71	47	64	77	87	86	67	59	49	55	66	64	101	67·3
Total deaths in hospitals	144	143	162	154	185	196	183	169	159	189	213	191	174	174
Percentage autopsies to death..	49·3	32·8	38·8	50·0	47·5	43·8	36·6	34·9	30·8	29·1	30·9	33 5	58·0	39·6

Number recorded by Osler, 87; Sutherland, 45; Howard, 32; Jamieson, 4; Johnston, 457; Lafleur, 87; Finley, 107; Adami, 73; Martin, 4; Williams, 35 others, 3.

SPECIAL INDEX.

GENERAL LESIONS AND DISTURBANCES.

Tuberculosis.

Tuberculosis.

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SKIN AND SUBCUTANEOUS TISSUE.

[Reprinted from the Boston Medical and Surgical Journal of December 22, 1898.]

THE MEDICO-LEGAL SIGNIFICANCE OF THE PRESENCE OF SUGAR AND GLYCOGEN IN THE LIVER POST MORTEM.[1]

BY W. K. BROWN, M.D., AND WYATT JOHNSTON, M.D., MONTREAL.

In 1897 Lacassagne and Martin, of Lyons,[2] reported the results of two hundred cases which were tested as to the presence of sugar and glycogen in the liver at the autopsy. Their observations were made with a view of investigating the statement of the late Claude Bernard,[3] that the livers of persons dying sudden or violent deaths contained sugar or glycogen, and usually both, while these substances were said to be absent from the livers of persons dying of disease.

Until the subject has recently been re-studied and a practical method of performing the test was discovered by Lacassagne there were practically no observations, except some made in his laboratory by his pupils Colrat and Fochier in 1888, and ~~Colomb~~ in 1893. The technique recommended is to make an emulsion of the liver-substance (usually 100 grammes), by rubbing it up with water, boiling this in a porcelain capsule, filtering with animal charcoal, and testing the filtrate by Fehling's solution. If glycogen was present the fluid was opalescent. We find that smaller amounts of liver-substance suffice, and that the use of charcoal is hardly necessary.

Colomb

[1] Read at a meeting of the Massachusetts Medico-Legal Society, October 5, 1898.
[2] Archives d'Anthropol. Criminelle, 1897, p. 446.
[3] Thesis de Faculté des Sciences, 1853.

Lacassagne and Martin recognized the medico-legal value of a test of this kind in deciding such difficult points as whether the injuries received by a person suffering from some serious disease are the immediate cause of death or not. They state that, in the main, their results are strongly confirmatory of the statements of Bernard, though they reserve for the present their opinion as to its significance in poisoning cases. They find that dead-born syphilitic children give a negative reaction, and children live-born or dying during parturition positive ones. On these points we have confirmed their observations.

As the matter seemed to us of practical importance we have made observations on a series of one hundred unselected cases, obtained from the coroners' court and hospital service, and about equally divided between violent and natural deaths. We wish here only to summarize the results obtained, and it is the intention of one of us [4] to report the observations more fully in a subsequent paper. The number of cases studied we do not regard as sufficiently large to serve as a basis for exact statistics. Our results were typical in eighty-eight cases and atypical in twelve.

(*a*) RESULTS IN CASES DYING FROM DISEASE.

Sugar and glycogen were found absent from the livers of those dying from disease except in two cases, in one of which cerebral hemorrhage and in the other brain abscess suddenly terminated the lives of persons apparently in fair health. One case of fatal gastric hemorrhage in hepatic cirrhosis gave a negative test, however. In cases of sepsis, of even a few hours' duration, the result of the reaction was always negative. In an elderly person dying of exhaustion ten days after the receipt of severe burns sugar and glycogen were

[4] W. K. B.

also absent. A negative result was obtained in a case of puerperal eclampsia fatal in twenty-four hours. A negative result was obtained in a case of diabetes dying from septic infection following gangrene.

(*b*) RESULTS IN CASES DYING FROM VIOLENCE.

Where the death was sudden and affected persons in vigorous health, sugar and glycogen were always present, provided that the processes of alimentation were proceeding in the ordinary manner. On the other hand, in three cases where death resulted within forty-eight hours of injuries (cut throat, fracture of vertebræ, fracture of the base of skull) the result was negative; in the last-named case there was a moderate catarrhal jaundice, from which the patient was convalescing, and the lungs showed traces of commencing aspiration pneumonia. In two cases where death was immediate and due to fracture of the skull, the liver showed neither sugar nor glycogen. In both of these cases there was evidence, both post-mortem and clinical, of acute alcoholism, while there appeared to be no food in the stomach or upper intestine, and in one of them scarcely any feces was found in any part of the intestines. Here it appeared that the individuals had been indulging in a long spree, and, as is common in such cases, had taken little or no food. In another case, where death occurred in the course of an attack of delirium tremens, a negative test was obtained. We think that this point is one on which further information is important, in view of the frequency with which an excessive use of alcohol forms an incident in the circumstances in deaths calling for medico-legal examination.

A case of suicide by carbolic acid also gave negative results to the test. Here, as the man was found dead locked in his room, we had no clear account of the

conditions affecting alimentation, but it was stated that his habits were intemperate and that he had recently been drinking heavily. The autopsy did not show that food had recently been ingested.

In two cases of drowning, where the bodies were in an advanced state of decomposition, the test gave negative results; in two others the test was positive.

CONCLUSIONS.

We can confirm the results of previous investigators as regards the result of the test in healthy persons living under normal conditions of nutrition. From our experience so far we are inclined to regard the test as one of very considerable value where the conditions relative to alimentation previous to death can be established through the history or by the results of the post-mortem. It should be borne in mind that the information as regards the cause of death is indirect and that the direct information relates only to the glycogenic function of the liver. We wish to emphasize the important influence of sepsis and of alcoholism with abstention from food; when these are excluded we find the results very constant.

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February 5th, 1898.

THE CONDITION OF TEST CULTURES ESPECIALLY AS REGARDS TITRATION FAVOURABLE TO CLEAR SERUM REACTIONS BY THE DRIED BLOOD METHOD.

BY

WYATT JOHNSTON, M.D., and D. D. McTAGGART.

[From the Laboratory of the Board of Health of the Province of Quebec.]

THIS matter was referred to in the discussion at Philadelphia on June 3rd, 1897, before the American Medical Association, by Bates Block and one of us. We found that by varying the composition of the medium and the conditions under which the culture is kept, it is possible to obtain a culture which will yield excellent results or the reverse. We can now at will obtain a bouillon culture which can be left in contact with solutions of non-typhoid blood for 24 to 48 hours without giving any reaction, and will react promptly with typhoid blood. On the other hand, we can by neglecting certain details, get a test culture which will give one of two pseudo-reactions with almost any blood solution or serum. Pseudo-reaction A is a decided clumping of over 95 per cent. of all the bacteria, but without loss of motion. Pseudo-reaction B culture with most blood solutions will show cessation of motion, with defective clumping. The former (A) we found to occur with cultures made very active by daily transplantations, and the latter (B) to growth in rather too alkaline bouillon.

Our earliest observations happened to be made with bouillon giving satisfactory results. These became unsatisfactory for a few weeks under daily transplantations and good again on returning to monthly changes of stock cultures. After a few months a change in the laboratory *personnel* with different assignment of duties led to the second class of pseudo-reactions troubling us for a few weeks. Attention to the reaction of the culture medium remedied this difficulty, which seemed entirely due to a trifling increase in the alkalinity of the laboratory bouillon. At another laboratory in charge of one of us there was still a supply of bouillon remaining which gave satisfactory results. This reacted 3.5 per cent. acid to phenolpthalein and just verged on the acid point with litmus. The adoption of bouillons having a reaction requiring 3.5 per cent. of normal alkali to restore the tint of phenol-phthalein led to satisfactory results once more. The reading of the end point varies somewhat with the observer and the optimum reaction varies slightly with the particular culture and the composition of the medium, but always lies between 3 and 4 per cent.

The pseudo-reactions here described occur in bouillons giving a heavy growth and some sediment at the end of twenty-four hours at 37° C. They show a tendency to the spontaneous clumping mentioned by Widal, but this is greatly exaggerated by adding blood solution. Serum does not have the same tendency to cause pseudo-reaction A. Dilution alone did not prove in our hands a satisfactory remedy.

We believe that these anomalies explain the statements of some writers that all serums tend to cause clumping sooner or later. The optimum reaction depends somewhat on the temperature of the thermostat and the age of the culture. Satisfactory cultures show gentle clouding only at twenty-four hours, and have no sediment or serum. If the culture medium is made too acid it becomes insensitive, loses motility, and does not react promptly, but gives no pseudo-reaction.

We note for the first time in the BRITISH MEDICAL JOURNAL of December 4th, 1897, that Delépine had already in the JOURNAL on April 17th, 1897, called attention to the danger of pseudo-reactions if the bouillon is alkaline. He recommends neutral bouillon, whereas we found acid bouillon preferable. The question is one of intensity of growth rather than of pure alkalinity alone, since the pseudo-reactions can also be avoided by raising the alkalinity till the growth becomes scanty. Personally, our interest lay more in deciding the most suitable reaction for dried blood work.

Reprinted for the Authors from the BRITISH MEDICAL JOURNAL,
February 5th, 1898.

A QUANTITATIVE METHOD OF SERUM DIAGNOSIS BY MEANS OF DRIED BLOOD.

BY
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Montreal.
[From the Laboratory of the Montreal General Hospital.]

WIDAL suggested[1] the plan of simply dissolving the dried blood drop in a fixed number of drops of water. Dacosta advocates the same plan. The objection to this is that the previous volume of the blood drop cannot be determined after it has been dried, and they have not attempted to estimate the limit of possible dilution.

The plan adopted by us is that of taking the sample by means of a loop of standard size, made of No. 20 gauge copper wire, 2 mm. inside diameter. This gives a considerable quantity of blood which, when dried and spread on a glass slide, can afterwards be dissolved by means of a definite number of loopfuls of water. The solution thus obtained is abundant enough to yield much more blood solution than is necessary for quantitative measurement of the reaction. To ensure greater accuracy a small outfit was used in which the original loop used in taking the blood is returned, and employed in making the dilutions.

The dried blood drop is dissolved in five loopfuls of water used *seriatim*, the solution from each drop being sucked up by a capillary tube, and the whole mixed. From this stock solution standard sub-solutions are made in a straight capillary tube bound by a rubber band to a thermometer scale. The advantage of this plan is that the exact quantity of blood taken is immaterial. The tube is easily washed. The method of placing the loopfuls separately on the glass, as recommended by Delépine, was found very reliable.

In testing, a small platinum loop of the blood solutions is mixed with as many loops of water and bouillon culture as may be needed to give the required dilution, and make the volume of the diluted blood as nearly as possible equal to that of the culture. For convenience a table of the formulæ for dilutions was made, and is useful when one is ascertaining the extreme limit of dilution possible, as this often reaches into the hundreds.

For an ordinary test—to learn, for instance, if the blood reacts at 1 to 10—all that is necessary is to mix a loop of the stock 1 to 5 solution with one of the culture. By mixing it with four loops of water and five of culture, a dilution of 1 to 50 is readily obtained. The time required when this simple test only is needed does not exceed five minutes.

We have made duplicate observations to ascertain the average limit of error, and find this to be about 10 per cent. after some familiarity has been obtained with the method. It is much more accurate than a plan previously suggested by one of us of taking the standard of colour as a guide.

Comparative tests, when the duplicate samples consisted either of serum or of watery solutions of fresh blood taken by a Zeiss-Thoma barometer pipette, gave us results practically identical with those by the dry blood method, the latter being slightly lower, however, as a rule. So that if the dry blood showed a given intensity, the serum would show at least that, and perhaps 10 per cent. more.

Drying for a few days did not make any great difference in the intensity, though it slightly lessened the reaction. We defer the publication in detail of our results until our observations are more numerous.

The result of the quantitative dry method so far has convinced us that it is delicate and accurate enough to give results sufficiently close for practical purposes or for comparison with other methods of testing. It has not yet been found necessary to use the quantitative test for the ordinary routine diagnostic work; but where the results are to be used for recording doubtful or exceptional cases for scientific publication, we think quantitative data preferable.

It does not appear necessary, however, to use quantitative tests in ordinary routine diagnostic work. Our experience has been that in no case hitherto examined by us have we been able to obtain a decisive reaction by the use of quantitative methods of fluid blood or serum, when it could not also be obtained by the dried blood test without quantitative complications.

At the same time, we fully agree with the views expressed by Professor Welch on a recent occasion, that for scientific observations it was very desirable that the dried blood method should be also made a quantitative one, and our work in that direction was prompted by a friendly suggestion coming from him, for which we wish here to express our thanks.

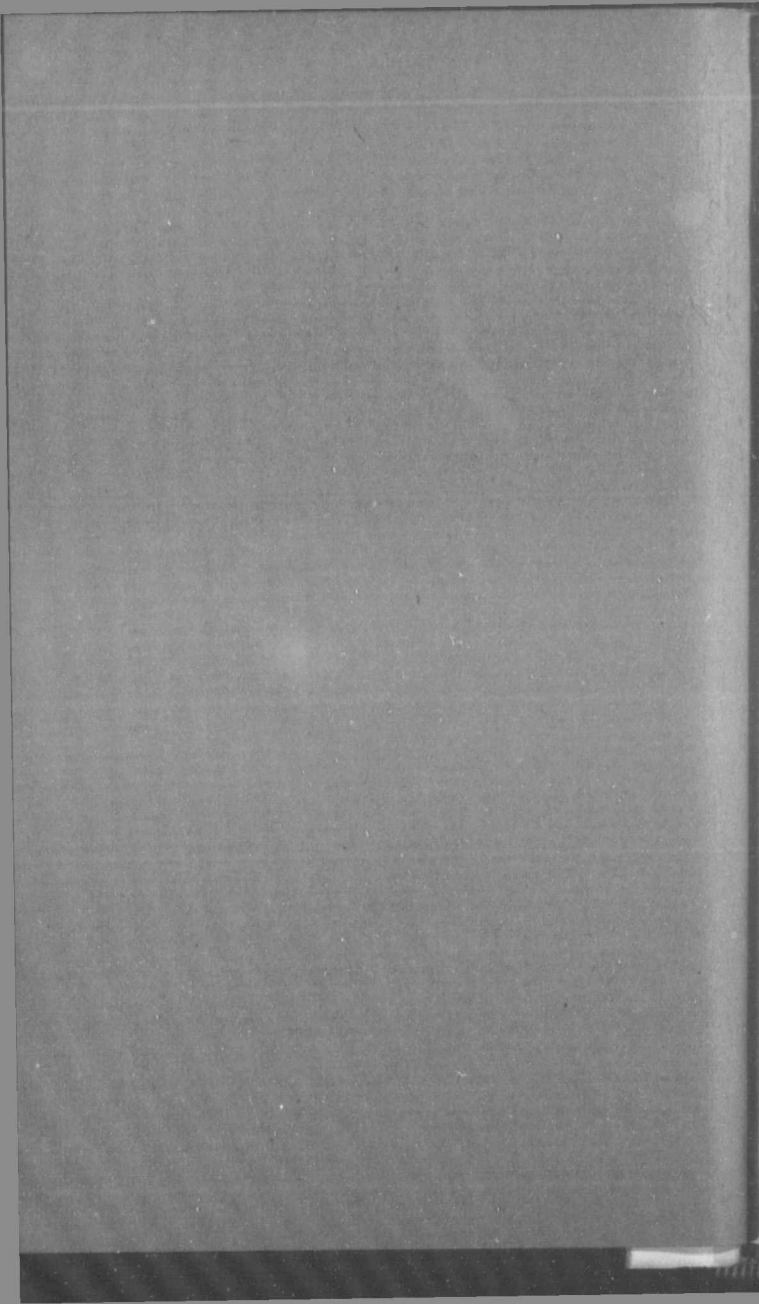
REFERENCE.

1 Soc. de Biol., January, 1897.

NOTES ON THE PROGRESS OF LEGAL MEDICINE:— THE MEDICOLEGAL STUDY OF INJURIES.

BY
WYATT JOHNSTON, M.D.,
OF MONTREAL, CANADA.

FROM
THE PHILADELPHIA MEDICAL JOURNAL,
1898.



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NOTES ON THE PROGRESS OF LEGAL MEDICINE:— THE MEDICOLEGAL STUDY OF INJURIES.

BY WYATT JOHNSTON, M.D.,
of Montreal, Canada.

FEW medicolegal textbooks deal at all thoroughly with wounds and injuries except with a view to the diagnosis of crime. We find that while relatively full consideration is given to wounds proper, this is by no means the case with other injuries, and only such accidents receive attention as are liable to be connected or confused with homicide or suicide. The one exception to this rule is made in the case of traumatic neuroses.

Thus, the criminal aspects of legal medicine tend to overshadow the civil ones in our textbooks, although the civil courts have to deal with ten medicolegal matters for one that comes before a criminal court. The civil cases, too, are more likely to devolve upon the practising physician than upon the medicolegal specialist. It is very desirable that the writers of some of our textbooks and works of reference should deal with the subject-matter in its non-criminal aspect.

Our journals, too, do not give much prominence to this line of work, such articles as appear being generally given not as original communications but merely as news-items, usually meager in detail and insufficiently vouched for from a scientific point of view. Even in special journals, such as the *Railway Surgeon* and the *Medico-Legal Journal*, the aim is rather to record forensic precedents than the scientific aspects of the question, and to be of assistance more to lawyers or ex-

ecutive officers than to physicians. Owing to the scarcity of detailed case-reports, new casuistic work and compilation of the scattered observation are especially needed as a basis for further progress, and one would prefer to see the careful study of cases take the place of the well-worn platitudes and ex-cathedra statements in which the addresses and communications made before medico-legal societies so abound. Of recent American literature on this subject, the excellent monograph by C. Phelps on *Gunshot Injuries of the Brain*,[1] that by Pearce Bailey on *Accident and Injury in Relation to the Nervous System*,[2] and the important clinical study by W. B. Coley of *The Relation Between Injury and Sarcoma*,[3] are examples of the highest type of literature upon the subject, and it is work of this kind that is most needed at present.

The barrenness of our own literature makes it necessary to be familiar with foreign sources of information. One is struck by the fact that the literature in this branch of legal medicine is almost exclusively German. Besides several periodicals[4] devoted exclusively to it, numerous articles appear in the general medical journals as well as the medicolegal ones, such as *Friedreich's Blätter*, and the *Vierteljahresschrift f. gerichtliche Medicin*. In the last-named journal an important series of monographs have recently appeared, dealing with the medico-legal relation of injury of the various internal organs and cavities, and especially with such remote and indirect aspects of trauma as tuberculosis, tumors, etc. A special department for "Unfallheilkunde" has been included for the past years in the Virchow-Hirsch *Jahresbericht*, while a number of useful larger works, hand-

[1] D. Appleton & Co., 1897.
[2] D. Appleton & Co., 1898.
[3] *Annals of Surgery*, March, 1898.
[4] *Monatschrift für Unfallheilkunde, Aerztliche Sachverständigen Zeitung, Archiv f. Unfallheilkunde.*

books, monographs, and collections of case-reports and important decisions have also been published. Of these I would specially mention Constantin Kaufmann's *Handbuch der Unfallverletzung*, 2d Ed., 1897; L. Beeker, *Lehrbuch der Sachverständigen*, 1895; E. Golebiewski, *Handbuch der Unfallsversicherung*, 2d Ed., 1897; P. Blasius, *Unfallversicherungsgesetz und Arzt*, 1892. Very full official reports, with details of cases and decisions, are also published by the German Imperial Accident Insurance Bureau, and collections of illustrative cases have been issued by R. Kaan, F. Ritter, and others. R. Stern's monograph on *Traumatische Entstehung innerer Krankheiten*, as well as his article *Trauma als Krankheitsursache*, in Lubarsch and Ostertag's *Ergebnisse*, deserve special mention; as does also the recent article by Thoinet, *La Pneumonie Traumatique* in *Annales d'Hygiène publique*, July, 1898.

Much of the best German scientific work in this department is by men who are proprietors of private sanitariums, hospitals, institutions for mechanical therapy, massage, etc. The leaven of science does not appear to work to the same extent upon the proprietors of our own numerous institutions and sanitariums, for, with certain noteworthy exceptions, of which that at Saranac Lake is the best known, the publications from our "institutes" suggest other motives than the mere advancement of science. On studying the matter, it appears that the cause of such conspicuously rapid and satisfactory progress in Germany may be summarized as follows:

1. The fact of a large proportion of the population being in Government employ has led to State-control of accident-insurance and benefit-societies, so that accident-insurance loses in large part the character of a private business-enterprise, and the medical men employed in it become placed in a position where they can view

matters with more independence than if employed by private corporations.

2. The Government regulations are to a certain extent to be followed in preparing reports of examinations, but these are not mere schedules of questions and answers.

3. In case of lawsuits for damages being made, the district government-physician practically acts as a judge in the medical aspects of the case. In the event of appeal, the medical questions are referred to an official committee of physicians; and if again appealed, to a higher court (super-arbitrium) of physicians having jurisdiction over the whole State. In each case a full report has to be made in writing, reasons being given for the views adopted. In this way an appeal exists from medical as well as legal opinion as regards the question whether the scientific facts in the evidence have been properly interpreted. Errors are investigated and often publicly criticised. The fact that every report is sharply scrutinized and annotated by higher medical authority leads to more careful preparation than would otherwise be the case, and in this way the progress in this branch of medical work has been directed and controlled by the highest medical talent in the country. We find reports by men of such standing as Virchow, and Bergmann, and others, dealing jointly with such minor matters as to whether a box on the ear of a schoolboy was really the cause of unusual and distressing symptoms that followed.

4. The medicolegal questions in civil as well as in criminal matters are referred to official physicians, and those in civil matters permit of a more refined analysis and delicately balanced judgment than in criminal. When the prisoner must have the benefit of any doubt, the reasoning must be on very broad lines in order to be safe. Another factor of importance is that, in Ger-

many, all such work is done by government-officials, who, before responsible duties are entrusted to them, must pass the *Physicats Examen*, a most severe test of efficiency in the requirements for sanitary and medicolegal work. The conditions that have so favored the study of the medicolegal relations of traumatism in Germany do not exist with us at present, and it does not seem likely that a general system of medical officialism could be practicable in the absence of a strongly centralized government. Better opportunities for the medicolegal and clinical study of traumatism exist in connection with the smaller benefit and accident-insurance societies, where matters are left to the decision of the physician, than in the very large insurance-corporations whose wholesale business-methods of settlement do not conduce to the scientific consideration of the medicolegal points involved in individual cases. Unfortunately the medical officers of our benefit-societies do not realize the scientific potentialities of their position and regard the opportunities afforded them for scientific medicolegal study with anything but enthusiasm.

In France no special examination exists, but by a regulation no one can act as expert before the courts unless five years have elapsed since receiving his diploma.

In England and America it is customary to entrust a large proportion of this work to recently graduated hospital-internes, who naturally regard it more as a perquisite by which they are kept supplied with a certain amount of pocket-money than as a subject worthy of thorough scientific study.

Recently the London County Council recommended to the Home Office that medicolegal expert duties should be assigned as far as possible to the pathologists of the public hospitals, though, so far, no official regulations to that effect have been issued.

In England and America there is no system of practical medicolegal instruction compulsory in any of the universities. A very full account of the provisions for practical medicolegal instruction in European countries is given in the article by Dr. P. Loye.[5]

When we consider that in almost every medicolegal case the essential problem as to whether a certain effect is due to a given cause, is in the majority of cases one in pathologic etiology, it is remarkable how comparatively little sound pathology one finds in the records of medicolegal cases and how very little the subject of pathology has been enriched from medicolegal sources. From reading a large number of records it would seem as if accurate work in this direction were greatly needed at present.

Careful examination into the facts will convince most persons that when the entire medicolegal material furnished by almost any of the general hospitals in our large cities can be placed at the disposal of some member of the staff, specially interested in this special line of work, it would enable instruction in legal medicine to be given the same thorough clinical and practical attention as is now the case with the other subjects of the curriculum.

A provisional arrangement of this kind was recently made at my request by the staff of the Montreal General Hospital. It is yet too early to say in how far we will be successful in placing the teaching of the subject on a practical clinical basis. At the outset I have met with a certain coyness on the part of the surgical staff in allowing their cases to be studied, which tended to limit the amount of available material, whereas the cordial and thorough cooperation of the staff of a hospital is necessary for the full success of the plan. I must mention that Dr. F. W. Draper, of Boston, has

[5] *Annales d'Hygiène publique*, 1890.

already followed for some years the system of having occasional clinics on living medicolegal cases given by one of his assistants.

It seemed advisable to explain in a general way the extent and character of the literature available before going into the details of the subject and to leave these for consideration in subsequent reviews.

Recent Work Bearing Upon the Pathology and Morbid Anatomy of Shock.

BY

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Montreal, Canada.

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RECENT WORK BEARING UPON THE PATHOLOGY AND MORBID ANATOMY OF SHOCK. *

By Wyatt G. Johnston, M. D., of Montreal, Can.,

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I recollect one of my teachers in Pathology quoting in full Johannes Müller's lecture upon the functions of the spleen, the full text of which is alleged to be as follows: "Gentlemen: We will consider to-day the functions of the spleen. As nothing at all is known about the functions of the spleen we will therefore pass on to the next subject." The pathology and anatomy of shock appear to be treated in much the same summary manner by those who write on the subject. Pearce Bailey, "Accident and Injury in Relation to Diseases of the Nervous System" (p. 202), writes as follows:

"The belief which was at first systematically formulated by Page has now become general, that in by far the larger number of litigated

* Read in the Symposium on Shock, Toronto, 1898.

cases of nervous disorders which follow railway and allied accidents there is no gross injury to the nervous tissue. Psychical concussion may be a cause of subjective nervous symptoms, and if severe may indirectly cause organic injury. But the conception as a cause for general nervous symptoms and as a pathological condition is without foundation and has been almost entirely abandoned. By far the larger number of cases of the traumatic neuroses may be explained by the theory that the symptoms are those of hysteria or neuræsthenia, functional disorders of which the pathology is unknown. There are, however, a few cases in which the symptoms are somewhat different from those of hysteria or neuræsthenia, and which may depend upon the structural lesions of the brain and cord though our knowledge of their pathology is largely speculative."

Again the same author (Ibid p. 233) says: "Little is known of the pathology of neuræsthenia. It rarely kills, and there are no recorded autopsies in which the lesions found were sufficiently adequate to account for the neuræsthenic symptoms. In the autopsies which have been made of persons who died during the course of the disorder the nervous system has not been examined with sufficient care to discover any morbid appearances in the ganglion cells. Hodge, and, more recently, Lugaro

have shown, however, that visible alterations in form and structure occur in the ganglion cells of animals as a result of fatigue. And since fatigue is the most prominent feature in the clinical picture of neuræsthenia it is to be inferred that the pathology of the disorder is to be sought for in the nutritional disturbances of the ganglion cells. It would be useless to speculate here as to how these changes are brought about or what their essential characteristics are. It is enough to say that it seems probable that to explain the disturbances of function there are structural changes which may eventually be seen and to a certain extent understood. But until our knowledge regarding the pathology of neuræsthenia is more exact and full, it must continue to be classed with the functional diseases."

I have selected this author as being the most recent American writer on the subject and because his statements represent correctly the general consensus of opinion on this subject at present. While the remarks above quoted refer more particularly to the traumatic neuroses which are chronic in character and whose relations to shock are remote they also apply to the acute and immediate effects of shock, such as appear after severe injuries from crushing or other causes. The essential feature which all forms have in common is that the discoverable nerve lesions are far

from characteristic and are not extensive enough to explain the profound and often very complicated disturbances of the nervous function found clinically. The fact that in one case a profound condition of shock may accompany a relatively slight injury while in a severer injury little or no shock may exist, also that in some cases the shock may be late in appearing points on the whole to the central rather than the peripheral nervous system being chiefly affected. It is in the ganglion cells of the brain cortex and the spinal cord, but especially the former, that changes have been sought for.

Virchow found nearly thirty years ago, in examining bodies where severe cerebral concussion had occurred years before, that the same ganglion cells, or as they are now called, neurons, were frequently found to be calcified, and hence it appeared probable that anatomical changes had existed in them at the time of the accident. This point was not established experimentally, however, and for many years no further anatomical observations of importance were made on the subject. In 1890, Schmaus of Munich made a number of observations and experiments as to the connection between myelitis and compression or other injuries of the cord. By striking upon a board placed along the back of a rabbit he was able to produce a series of paralytic and paretic

symptoms with almost an absence of gross lesions, capillary hemorrhages being almost the only anatomical change. Schmaus' studies were made more with reference to the fibers than the nerve cells and did not establish the occurrence of changes in these, but the more recent investigations in the same direction by Kirschgässer showed alterations in the cells also.

Within the past five years, the study of the neuron or ganglion cell has attracted much attention from neuro-pathologists. The various modifications of the Golgi method have enabled the changes in the fibers to be studied with precision, while still more recently a number of methods, notably those of Nissl, Van Gieson, and Barker, have enabled the minute changes in the ganglion cells to be studied out. The result of these studies has been to show that a number of important changes can be made out in the arrangement of the chromatin and the cell substance. This is shown in some cases by the abnormal distribution of the chromatin particles, in others by their almost complete disappearance. As the authors by no means agree yet among themselves as to the exact character of the lesions or their significance they need not be discussed at length here.

In addition we must bear in mind the occurrence of artefact effects due to the methods employed and of post-mortem changes previous

to the autopsy. Further the particular method employed and especially the personal equation where different observers employ the same method seem to cause differences in the result. This will explain in large measure the numerous minor discrepancies and diversities of opinion in the literature of the subject.

Within the last few years however, the advances made in neuro-pathology and in microscopical technique, valuable for the study of conditions, is such as, I think, to necessitate revision of this too agnostic standpoint. In the many articles published on the subject, the tendency is to show that changes of some sort in the ganglion cells are a fairly constant accompaniment of injury to the nerve centers which apparently leave no gross changes. The procedures employed by Van Gehuchten, Goldscheider and Flatau, Hodge, Levi, Mann, Lugaro and others agree in the main as to the fact of such changes being demonstratable, although there is a want of uniformity as to their exact nature. Ewing and Van Gieson have rendered important service in pointing out clearly the distinctions between artefact changes, due to decomposition, and those really representing marked alteration in the nerve tissues. We have the latter again occurring under two sets of conditions: one where the nervous tissue alone is especially involved, and

the other where it forms part of a general disturbance as in the case of toxæmia, etc.

The studies of Kempner and Pollock, that a number of definite changes in the nerve cells occur after infection or intoxication with the products of the sausage poison bacillus (B. botulinus) further afford the important evidence that when injected with protective serum these changes remain absent. It is noticeable that some of the most striking changes which attract the eye of the observer, such as the vacuolation of the nerve cells, etc., are now known to be merely post-mortem in origin.

With regard to the psychical element upon which so much stress has been laid, this perhaps has been unduly exaggerated. Morton Prince's observations on this point are very striking; he investigated a large number of football players of American colleges without finding a single instance of the conditions analogous to "Railway Spine," as a result of the game. Now short of actual warfare and railway collisions, there is probably no condition which would seem at first sight more conducive to spinal and nervous concussions than football as played in American colleges. He explains the absence of the neuræsthenic symptoms by the mental state of preparedness and the concussions being far from unexpected. Similarly, the fact that railway employes are more or less in a state of expectantness, and this ex-

plains their greater immunity than passengers, and of course the various emotions, fright, etc., are less liable to affect them than the latter.

The studies of the nerve cells hitherto have given much information as to changes in structure but little as to changes in function, although Hodge found that in stimulating the nervous cells in guinea pigs he produced a state of exhaustion with a lessened amount of chromatin; this change has not been quite satisfactorily corroborated by others. In any case, attention has been fixed, perhaps too much, on the chromatin elements, which have possibly only a minor part in exhibiting the functional activity of the nerve cells. It is obvious that some procedure in demonstrating the chemical and metabolic changes which take place would be capable of throwing great light upon this matter. This want to some extent has been supplied, at least as regards phosphorus, by the method of Lilienfeld and Monti (Ztsch. f. Physiolog. Chemie., 1893), who used the phospho-reaction brought out by means of molybdate of ammonia reduced by pyrogallol as an evidence of the relative abundance or lessened quantity of phosphorus compounds in the cells, which, as regards function, is perhaps a better index than the chromatin. The reaction has been used by a number of others, including Sherrington and Helde. It was

found somewhat uncertain in regard to tissues though useful in test tube experiments.

A decided improvement in technique has been found by A. B. Macallum of Toronto, who uses an aqueous solution of 1—4 per cent phenyl hydrosine hydrochloride, which must not be more than 2 or 3 days old. It distinguishes sharply between the molybdate compounds and the phospho-molybdate compounds, the former appearing brown, the latter dark green, at first almost black; the reaction is certain in the absence of an alcohol or a caustic alkali. On adding an acid solution of molybdate ammonium and nitric acid, a deep blue violet color appears.

Details of methods are very fully given in Dr. Macallum's paper (Proc. Royal Soc., Vol. 63, p. 467, 1898). Mr. P. H. Scott, working under his directions, found that the Nissl's granules in the nerve cells gave a distinct phosphorus reaction which was also present in the nerve elements of the retina in an eye which had been exhausted by prolonged exposure to light, and the corresponding eye blindfolded, in experiments on rabbits. Dr. Macallum has kindly written to me concerning the necessity of great care in the preparation of the reagents, especially in the keeping of the acid ammonium molybdate solution tightly stoppered so as to prevent evaporation. The nitrate molybdate reagent is improved by dissolving one part of

pure molybdic acid in four parts of strong ammonia and adding thereto, slowly, 15 parts of acid, nitrate, sp. g. 1. 2, proportions indicate weight. Solution is faintly yellow with decantation, remaining free from precipitate.

It seems desirable that an examination should be made of the condition of the nerve elements with regard to the presence or absence of such changes in cases of shock. There is a good deal of clinical evidence to show that in the absence of gross alteration in the functions some condition is present where the vital powers are greatly lessened, and a slight additional disturbance of this vital force will produce unexpectedly fatal results. For instance, it is common to find cases transported long distances and apparently suffering from a mild degree of shock for a trifling operation, perhaps the removal of a nearly detached limb seems expedient, and a whiff of chloroform or ether is given and the patient never rallies from this short and trivial anæsthesia; this occurs most commonly in alcoholic subjects.

In reference to this point I wish to call attention to the recent studies of H. K. Wright, formerly of Montreal, who has shown that organic changes of the nature of granular degeneration in the nerve cells process occurs as the result of overdoses of bromide of potassium, either in man or animals. He has also shown that a similar condition is produced by

chloroform and ether anæsthesia, and this may explain in part why the accidents occurring as above described so frequently follow the administration of an anæsthetic. At the same time we must not too hastily conclude that the neuron which has become so prominent lately is to be made the base for everything in neuro-pathology, as apart from the previous explanation of late death after anæsthetics in epilepsy probably, and in Graves' disease certainly, the disturbances of the internal secretions, owing to the irregular and abnormal condition of the thyroid gland, appear to be a true explanation of the difficulty. As regards epilepsy, Ohlmacher has recently published observations showing that in a number of cases at all events enlargement of the lymph glands exists (the so-called status lymphaticus) and often there is persistence of the thymus gland. That these conditions exist in otherwise inexplicable sudden death has long been known and the relation seems, if not proven, at least possible. What seems to be needed at present is the careful working over of pathological material and particularly a careful study along the lines laid down by Nissl, Macallum, and others, of the chemico-pathological changes which are associated with the conditions of nervous disturbance.

In my own laboratory, although a number of observations have been made in this direc-

tion, we do not so far feel prepared to communicate the results, as we do not feel that we have yet gotten at the bottom of the personal equation. It seems in this line of work in particular very difficult to compare the results of one observer with those of another. We have been able to confirm in a general way the views of Ewing and Goldscheider as to which changes are due to artefact conditions and which ones indicate general systemic disturbance. Our opinion as to the conditions of the nerve cells in shock must be reserved for further consideration.

On the whole it may be stated with considerable confidence that minute anatomical intracellular changes of some sort underlie the conditions of shock encountered in railway surgery. It is probable, too, that limited lesions of minute dimensions due to hæmorrhage, etc., are more common than has hitherto been supposed and the marked increase in the number of cases of syringomyelia which have come to light since attention has been directed to their occurrence makes the assumption of purely functional explanations less and less tenable for the majority of cases.

I may add a word or two as to the pathological conditions which justify us in assuming shock as the cause of death. We should insist upon all the causes including hæmorrhage being rigorously excluded, and establish a com-

plete and thorough autopsy, noting the absence of gross anatomical lesions of vital organs as well as the absence of conditions such as fat embolism or fatty degeneration recognizable only by misroscopic examination.

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ON THE PRACTICAL CLINICAL TEACHING OF STATE MEDICINE.

BY WYATT JOHNSTON, M.D.,

of Montreal,

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By instruction in State medicine, I refer to that beyond what is required to qualify the average medical student for his degree, up to what is required to qualify him as a medicolegal or sanitary official. Though the term State medicine has by usage come to signify, in this country and England, merely public hygiene, it should properly include legal medicine as well, and it is in this wider sense that I employ it.

On comparing American facilities for instruction in State medicine with those existing in Europe, we find ourselves handicapped by the absence of official connection between the medical schools and the State. Professors of hygiene and legal medicine do not with us, as they do in Europe, become as such entitled to official positions which give them opportunities to study and practice the specialties which they teach; hence, except in the case of a comparatively few State universities, it is evident that our schools must select their teachers in these branches from among those persons holding State positions, or that the latter must obtain teaching positions in order to make practical teaching possible. Evidently, active cooperation between teaching bodies and State or municipal authorities is highly desirable.

The largely increased number of teachers who now occupy State positions, as compared with that of a few years ago, naturally leads one to inquire to what extent the teaching had become more practical. We find, however, that beyond a decided augmentation in the amount of laboratory work, the State medicine courses are still largely modeled on the old plan, and little or no teaching analogous to clinical instruction is given.

Professor F. W. Draper, of Harvard Medical School, who has for some years made systematic clinical instruction in legal medicine a feature of his course, is almost the only teacher who has done so. In hygiene very little has been attempted anywhere in the way of practical instruction in sanitation, and while courses have been begun in several institutions, they have as a rule been discontinued. I would not advocate any great increase in the compulsory work in these subjects for all students, since hygiene and legal medicine are subjects in which, while every student must know the minimum, advanced work is better left optional. This rule, which applies to all specialties, has been well pointed out by Dr. F. P. Mall in a recent article in the PHILADELPHIA MEDICAL JOURNAL.

I thought it might be of interest to record some attempts which I have made to place instruction in State medicine on a practical clinical basis in Montreal.

1. *Legal Medicine.*—A special advanced optional course was given in this subject for students who had already fulfilled the University requirements. I had, as coroner's physician and as pathologist to the Montreal General Hospital, postmortem material making with some outside cases a total of about 300 autopsies yearly. By being officially placed in charge, through the kindness of my colleagues, of all the medicolegal cases and damage claims averaging, arising in connection with cases treated in the Montreal General Hospital, a very interesting medicolegal material was obtained which could be readily utilized for instruction. This was given in the form of demonstrations and a weekly clinic, together with a system of medicolegal case reporting by students, similar to that followed with our medical and surgical cases. In this way, each student attending the course has occasion to report a number of cases and receives a fairly thorough training in the scientific estimation of disability, based upon the methods of the German authorities, who have made the subject a special study. This training in estimating the disability after injury or deciding whether or not a disease is really due to traumatism, appears likely to be of more real service to the majority of physicians than training in criminal medicolegal work. I pre-

pared a class syllabus giving the principal data on the subject of disability according to standard authorities, which has been published in the *Montreal Medical Journal* for April, 1900.

For training in the criminal side of medicolegal work, besides the examination of stains, etc., each student was required to do autopsies on bodies upon which typical injuries in the way of cuts, stabs, shots, and corrosive poisoning had been previously inflicted (post-mortem). The extent to which the written report, prepared in each case was accurate, clear, and to the point, showed in how far the technic was correct and the appearances accurately observed and properly interpreted. The practice was followed throughout the course of detailing two students to each case, one representing the plaintiff or State, and the other the defense. The latter was required to comment upon the report of his colleague and criticise it. (A plan which incidentally saved the instructor a good deal of labor.)

The largest amount of material available on any one class day occurred in the spring of 1899, and was as follows: (1) A case of infanticide; (2) a case of sudden death; (3) a case of attempted murder with inquiry into mental condition of accused; (4) a case of alleged rape with examination of stains; (5) a damage suit for injury to nerves of arms; (6) an accident-insurance case of fracture of the leg; (7) an employer's liability case of loss of an eye. Such a variety of material was, however, quite exceptional, and is by no means necessary.

During the course, one evening a week was set apart for a conference at the teacher's house (after a method followed by Professor Wm. Osler) at which the work of the week was discussed. I had also in my class the students of the McGill law faculty, to whom I give a short annual course of lectures. We tried the experiment at these conferences of detailing two law students, who had also usually seen the cases at the clinic, to act as prosecuting and defending attorneys. The evidence, of course, was confined mainly to the medical points, essential outside facts taken as being in evidence when necessary. The medical witnesses were then examined and cross-examined, and after the judge (a law student)

had given his decision, I was enabled to make a few remarks as referee.

The study of the cases in this way appeared to be more thorough and real than when the cases were simply demonstrated, and the students appeared to digest and apply their information very well.

A set of syllabus forms for medicolegal investigation, some of which were adapted from those of Professor Lacassagne, proved an invaluable practical guide in studying and reporting cases.

The course was followed by 20 students, of whom 12 obtained certificates.

2. *Hygiene.*—The class in this subject was a postgraduate one and the standard adopted that of the English *Diploma in Public Health.* This calls for six months of practical outdoor sanitary work and six months of bacteriological and chemical laboratory work.[1] What I wish to say concerns chiefly the plan of instruction in outdoor sanitary work, which is a follows:

A practical course in sanitation was given in connection with the Montreal Health Department jointly with Dr. L. Laberge, medical officer of health for Montreal, without whose kind cooperation the work would have been impossible.

Each member of the class was given a syllabus or prepared one for himself, detailing the points to be observed in making some special sanitary inspection, for instance, of a dairy, a vaccine institute, a water-supply, a factory, etc. Each one had thus personally to study the objects most important from a sanitary standpoint, doing also any necessary laboratory work and preparing (1) a full report; (2) a summary of the objectionable features found, and (3) recommendations for improvement. The reports made during the week were discussed at the weekly conferences of the class and criticised by the members. In their spare time, the members also accompanied the inspectors and officials in the discharge of their routine duties, though this was less insisted upon. The candidates were given opportunities of clinical study at the civic infectious hospital, though this part of the work was not organized.

[1] This standard was adopted officially by McGill University and a diploma provided for, this being arranged by Dr. F. R. Ruttan, Registrar of the McGill Medical Faculty, to whose prompt action in the matter the successful issue of the course is largely due.

By the plan followed, a maximum amount of independent work is secured, with a minimum demand on the instructor's time, and the nature of the work obliges the men to study the subject thoroughly and draw their own conclusions unaided.

Another means by which we propose to utilize the opportunities for study in sanitation without too much loss of time is a series of clinics in connection with the local health board in which the various questions of interest requiring to be dealt with during the week can be discussed before the class and the reasons explained for the course adopted. This is a departing from the stereotyped lectures with diagrams and models of drains, etc., which often form the stock in trade of the teacher in hygiene. Set demonstrations and inspections of typical sanitary objects of interest are better, but still lack variety and do not sufficiently confront the student with the actual problems which present themselves for solution.

The laboratory instruction consisted in a practical course in sanitary chemistry by Professor R. F. Ruttan, and one in sanitary bacteriology by myself. Some lectures on sanitary law were given by Mr. C. M. Holt. Some instruction in sanitary engineering had been arranged for, but we found no time to give it. This was also the case with meteorologic work.

In recording the methods I have personally followed, I have to plead guilty of ignorance as to the extent to which they may have been already used by others, never having personally had the benefit of any systematic instruction in hygiene or legal medicine. I can only say that being struck by the fact that little was apparently being done in these branches upon the well-tried lines of clinical instruction which has been found to answer so well in other subjects, I tried to adapt them to State medicine. Although no doubt others are doing this also, they are still too commonly neglected. While, as far as I can judge, the result has been decidedly encouraging, the ultimate result of the plan may be success or failure. The experience of other American teachers who have tried to establish advanced teaching in hygiene have shown that the matter is not very easy; in fact, one reason for reporting my experience now is

a fear that later on I may be unwilling to do so, and may have unrecorded experiences which, if not fortunate in my hands, might still be helpful to others.[2]

For the benefit of those working along similar lines, I may mention that in beginning work of this kind, one must secure beforehand the cooperation of teaching, licensing, and sanitary bodies to prevent a clashing; one must be prepared to give personally any part of the whole course if necessary; and one must find before beginning some means by which those attending will derive a material advantage from it.

A cooperation of sanitarians, teachers, and legislators, effected through some central sanitary body like the American Public Health Association, might afford a valuable means of securing uniformity in the matter of qualifications and diploma. I think that a diploma should imply at least several months of instruction; for shorter courses certificates might be given.

I think it important that in connection with medical schools facilities should be provided for advanced and practical teaching in State medicine, and that the State authorities should aid in the matter. Elaborate laboratories are less needed than practical and systematic utilization of the ordinary sanitary material on the same basis as clinical teaching. I have tried to show how facilities such as exist in every city in America may be readily made use of to place the teaching of State medicine upon the basis which it occupies in Europe.

Another direction in which teaching bodies might render more assistance to the health boards is in the systematic training and certification of sanitary inspectors and the lay officials of health departments. Universities proper having the teaching-staff equipment are in a better position to do this than health boards. I am at present giving a course of systematic instruction to the sanitary inspectors in connection with the Montreal Health Department at the request of Mr. H. B. Ames, chairman of the Health Committee, as a return for the opportunities afforded for teaching my own classes.

[2] Since writing the above we have completed the course and 5 candidates, 3 of whom are health officials, have earned diplomas; 8 others, including 3 health officials, have partially filled the requirements and will complete them later on.

The courses of the British Sanitary Institute have been selected as the best model to follow in courses of this sort. It is difficult to see what body should hold the examinations and issue certificates such as are given by the British Sanitary Institute.

We have secured informal pledges from the public authorities that a qualification in public health will be made in the near future a prerequisite to the holding of certain positions, and in the meantime they have at my suggestion adopted the plan of giving a cash bonus to such officials already in office as may secure the qualification, on the assumption that their services would thereby become more valuable.

ON THE ESTIMATION OF DISABILITY AND DISEASE DUE TO INJURY.*

BY

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The literature in regard to the medico-legal aspect of disability and disease resulting from injury is at present almost exclusively German, I know of no work in English which deals with the subject in a general way, though several of those referring to special parts of it are among the most valuable we possess. The reason for the preponderance of German literature in this field lies in the very extensive system of government insurance against industrial accidents, introduced into Germany in 1884 and controlled by the German Imperial Insurance Bureau. This has resulted in the training of a class of medical officials whose unbiased and thoroughly scientific study of the questions involved cannot be placed too high.

The principle of an equitable compensation of labour accidents has been enforced by law in Switzerland (1881), Austria (1887), Italy (1898), Belgium (1894), Norway (1894), Denmark (1898), Finland (1897), England (1898) and France (1898), and will before long probably be adopted by all civilized countries. Russia and Sweden have similar laws already drafted. A bill aiming at this was introduced recently in the Ontario Legislature, but has not yet become law. I understand that similar legislation is contemplated for the Province of Quebec.

The above legislation is in all cases based on the principle that in the case of personal injury received through accident, occurring in connection with occupation and not intentionally caused by the employee or specific negligence on the part of the employer, the loss should be shared between employer and employee under conditions fixed by the government, which also guarantees the payment of such indemnity as may be decided upon, if the employer becomes financially unable to do so. The amount of such indemnity paid by the employer varies from 50 per cent. in England and France to 60 per cent. in Austria, and 66 2-3 per cent. in Germany and Switzerland. The non-compensated part of the loss represents the workman's share. As a rule the loss is equalized by systems of liability insurance or by mutual benefit societies guaranteed by government, the assessment varying with the localities

* Read before the Montreal Medico-Chirurgical Society, Jan. 26, 1900.

It is purposed to supplement the present article by subsequent ones dealing with the topics of "Methods of Examination for Disability," "Trauma in Relation to Disease," and "Disability Due to Sickness."

and the particular trades insured. Payment by annuity is mostly followed, though in England this may be commuted by a lump sum calculated on a basis of three years full wages.

The limitation of liability does not apply to cases in which specific negligence of the employer is proved, in which case the full amount of damages may be recovered under the common law; but in England the injured person must decide at the outset whether he will accept the partial indemnity or proceed in the courts. A very clear summary of the law on the subject has been recently published by Professor F. P. Walton, (*La Revue Legale, Feb., 1900.*)

As the assumption is that the employer is liable for a certain proportion of the loss in all cases unless exceptional negligence is shown, the legal questions under the government insurance system, are relatively scanty; and the assessment of the amount of disability incurred, which is essentially a medical matter, is the chief problem. The question of what constitutes sufficient ground for assuming a certain medical fact to be proved, is of course a matter of judicial decision. In Germany, Austria and Switzerland, there are over 20,000,000 persons insured under the laws, and the claims from over half a million accidental injuries are annually adjusted by the officials. In Germany, the hospital or home treatment is free during the first three months following an injury, but compensation only begins at the termination of three months.

The conditions under which we have to do with the estimation of disability are:—

(1) Employer's liability.
(2) Accident insurance and benefit societies.
(3) Medico-legal damage claims.
(4) Pensions, etc.

In employer's liability, the nature of the medical work depends largely upon whether special legislation exists concerning responsibility in ordinary cases, or whether the responsibility is left an open matter to be settled by litigation in each case. In the former, the medical study of the case is the chief factor; in the latter, the legal element dominates from the outset, and the medical problems are of secondary importance. In accident insurance, the liability is limited by contract, the amount, rates and compensation being specified, and a proviso made excluding all effects of illness or constitutional conditions, so that the medical aspect of the case is considerably narrowed. Hence, comparatively few accident insurance claims, unless grossly unreasonable, are contested, apart from the fact that from business reasons a reputation for paying claims is generally sought.

In medico-legal damage claims, one of the chief hindrances to rational adjustment is the circumstance that the facts are often only known to

one side and the amount demanded is usually much in excess of fair compensation. It is especially in these cases that trivial proofs of injuries, which are often non-existent, are supported by expert testimony of a kind which has obtained for medico-legal experts as a class a more than doubtful reputation.

The above is mentioned to indicate how one set of conditions, which rarely calls for serious medical consideration under certain circumstances, may form the bulk of medico-legal work in others. The amount of trouble and expense caused to railroad companies, for instance, by cases where the injury is ill defined, non-existent, or exaggerated or misrepresented, far exceeds that caused by definite severe and well authenticated injuries. It must be remembered, however, that the element of prognosis, with all its attendant uncertainty, enters largely into this branch of medico-legal work, whereas the criminal branch of legal medicine deals mainly with what is past.

The closely allied subject of sickness insurance, which has been thoroughly studied in Germany and to which our insurance companies are now beginning to devote attention, presents many points of interest which have not received the scientific study they deserve from the medical officers connected with benefit societies.

In the present article the subject matter is divided as follows :—

(1) Permanent disability from injury.

(2) Temporary disability from injury.

In giving practical instruction on the estimation of disability due to injury in connection with my medico-legal course, I found it impossible to obtain adequate information on the subject from any of our English text-books.* I had, therefore, prepared, in the form of synopses, a concise summary of the statements of the leading German authorities (especially C. Kaufmann and Ch. Thiem) for the use of my students. These are reproduced together with an abstract of some of the comments made about the more important topics.

It has since occurred to me that this might also be of service to physicians concerned in accident insurance work or who have to give evidence about damage cases in court, as well as insurance officials, judges, claims agents and those having to do with pensions of any kind. It is remarkable how little use has been made hitherto of the very convenient continental method of expressing the disability in terms of the percentage loss of earning power, and how little it has been made with us a subject of medical study.

The best known of several tabulations of the loss of earning power in

* The appearance of Pearce Bailey's English translation of E. Soliebiewski's "Hand Atlas of Diseases due to Accident" (W. B. Saunders, Philadelphia,) will materially improve matters.

regard to the leading forms of permanent disability, is that known as the Vienna Schedule, which has served as a basis for most other similar tables. It must be noted that in certain points minor differences exist among the standard authorities, and that the values given are only intended to be approximative and to serve as a point of departure in deciding individual cases. It must be remembered that the Vienna schedule is expressed, so to speak, in terms of unskilled workman. From this relatively simple problem, the variations called for by special forms of occupation can be determined. From the point of view of disability workmen are divided into four classes :—

(1) The unskilled laborer.

(2) The laborer whose work requires skill as well as strength, such as the bricklayer, mason, etc.

(3) The handicraftsman : as carpenter, joiner.

(4) The higher grades of skill, as mechanics.

It will be seen that the same injury might produce different results in each class. For instance, anything which impairs the finer movements of the fingers or wrists would represent a great loss to an engraver, whereas a laborer would be relatively little impeded by a partially anchylosed wrist, which was not painful and permitted of heavy work being done. On the other hand, a sensitive scar of the hand, which would incapacitate the laborer completely, might not interfere at all with the finer movements of the engraver. Accidents lessening the flexibility and free motion of the feet without impairing their firmness as a base of support and rendering them painful give relatively slight impairment to laboring men as compared with that caused in the case of roofers or sailors, etc. Injuries to the lower extremities cause much less inconvenience to those whose work can be done in a sitting posture than to others. Certain callings require unusual acuteness of sight and hearing as compared with others.

The following factors also come into account :—Can the condition be rectified by mechanical appliances if it cannot be improved by treatment ? Is it likely to get better or worse; is it temporary or permanent ? Can the person without difficulty adapt himself to another occupation ? Does the condition, besides incapacitating him from work, cause him an actual increase of expense for nursing, attendance, etc.? Are his chances of securing other employment diminished ? Does he suffer from pain ? Has the injury made him liable to any special disease ? Is the condition in part due to disease existing before the accident, or to some complication set up or predisposed to by it ? Can operative treatment be undertaken ? (The patient is under no obligation to submit himself to any operation which may be dangerous, all involving general anæsthesia coming under this category.) Was the

condition due, not to accident, but to occupation disease? Did it arise from causes unconnected with his work?

Medical men as a class tend to underestimate the injury to laboring men, and especially to reckon too short a time as the limit of disability after injury of the bones or of parts (hands and feet) used in rough work. The date at which a patient can be released from hospital treatment or when medical supervision becomes unnecessary, is often only one-half of the time required to put him in condition to renew his work. The schedule policies adopted by many of the insurance companies are not well adapted for the insurance of working men, as they are compiled on tables prepared for classes whose work is largely clerical or sedentary.

The frequency of actual simulation is much smaller than one would gather from medico-legal literature, and the cases, as a rule, are very easy of detection. On the other hand, more or less tendency to exaggeration is found in the majority of cases. Attributing to a recent injury conditions which pre-existed is perhaps the most common form of simulation: a decision on the matter may be difficult when the case is not seen soon after the alleged injury.

Just as in bacteriology we have certain postulates necessary to constitute proof of injury due to accident we require here:—

(1) There must be proof of the occurrence of an accident or injury.

(2) The accident and its effects must have occurred suddenly.

(3) The part affected must be located in the region injured.

It is astonishing to find how often these obviously essential data are unproven in cases of alleged injury.

Age. Injuries of young persons heal more rapidly than those of the old, and adaption to altered conditions is more complete and rapid. The immediate effect of injuries on the very young and very old is more marked than in adults. The predisposition to special diseases is greater at certain times of life, for instance, the liability to hernia in cases with advancing age.

Sex. Females need higher compensation for disfigurement than males. Slight disfigurement may be compensated only in case of females.

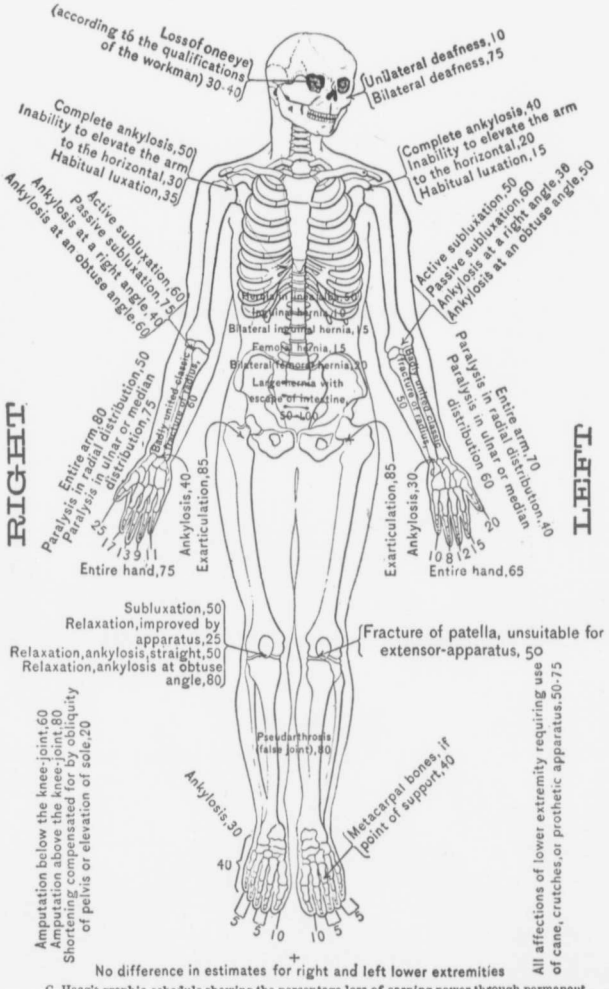
Previous Disease. The occurrence of an injury may leave a liability to the same injury. This is especially noticeable in dislocations, in abnormal conditions of the skin predisposing to erysipelas from trifling injury, or from exposure. Fragility of the bones from rickets or osteoporosis renders fractures more probable. The enlarged spleen in malaria is subject to injury. The existence of a latent or partly cured infectious disease, such as tuberculosis, may lead to unexpectedly bad results when persons are injured in the chest or subjected to a prolonged confinement. Disease of the ear greatly increases the danger of infection and meningitis in fractures of the base of the skull. Chronic heart diseases and

chronic lung diseases lessen the chances of recovery from a severe injury or shock, and influence unfavourably injuries of the chest wall. The enlarged (or pregnant) uterus is specially subject to injury from falls or other external causes. A latent appendicitis may be made acute by very moderate injuries of the abdomen. Rheumatic conditions may prolong the disability from injuries of the bones and joints. A disease may be the direct cause of the injury, as in epilepsy. The occurrence of disease as a consequence of injury is treated of more fully in another part of this article.

Alcoholism is one of the most important factors in regard to injury. Besides being a frequent cause of accident or neglect it may effect very unfavourably the chances of recovery. There is a great tendency amongst heavy drinkers, apparently in good health, to be seriously affected by relatively trifling injuries. The mere fact of confinement to bed through fracture of a bone is very liable, in a drinking man, to lead to an attack of delirium tremens, often followed by pneumonia. Hence, whenever practicable, methods of treatment which permit such patients to be up and about, are preferable. The grave effects of chronic alcoholism, such as ascites, renal cirrhosis, etc., lessen the chances of recovery and predispose to sudden death.

Occupation. Certain accidents are specially liable to occur as a direct result of the occupation ; toxic effects from inhalation of poisonous fumes, effects of changes of temperature and absorption by the skin of poisonous substances, apart from the direct danger of mechanical injuries from falling bodies, defective scaffolding, or other support, moving machinery, or electric currents, etc.

Over-exertion in connection with employment may be brought about by accident and is a frequent cause of sprains, ruptured muscles or tendons, and of hernia. On the other hand, many conditions ascribed to simulating accident may be really gradual in onset and due to unhealthy occupations. These should be carefully excluded and hence suddenness of onset in accidental conditions is an important point to establish. Predisposing conditions due to occupation may aggravate the effect of accident. The occupation may be such as to render it temporarily unsuitable for persons who have been injured. A tendency to neuralgia, left after injuries, makes exposure to draughts or changes of temperature injurious. Conditions leading to a defective closure of the eyelids or to conjunctivitis excludes from occupations carried on in dusty places. A tendency to giddiness, partial deafness, much loss of vision, or inability to move promptly, makes it dangerous to continue an occupation which necessitates being in the presence of moving machinery or involves the perception of signals. Callings which bring the person much in contact with the public, are more or less debarred to persons having



G. Haag's graphic schedule showing the percentage-loss of earning-power through permanent partial disability. (Compensation allowed equal to 60% of disability under the state system of compulsory insurance of workmen.)

Reproduced from Gould's American Yea Book of Medicine and Surgery. (By permission of W. B. Saunders, Philadelphia.)

mutilating or disfiguring injuries; and those which necessitate shouting out orders, to persons whose vocal organs have been permanently damaged. The percentage frequency of the common forms of accident vary greatly in rural and urban districts.

In persons who have been injured, one has to determine if the disabling effects are transient or permanent, and if such permanent conditions will improve, remain stationery, or get worse. When an annuity is paid, this may be increased or decreased according to the course of the case. The chances of ultimate recovery are often greatly enhanced by such measures as may relieve the person from the necessity of attempting heavy work before he is fit for it. On the other hand, the definite and final settlement of a claim one way or another, often has a wonderfully beneficial effect upon cases represented as being quite hopeless, and it is certain that the annuity system by no means tends to bring about the cures and lends itself to grave abuses.

The relative frequency of percentage compensation was found in Germany to be in the following order: 10, 20, 15, 50, 33, 25, 100, 30, 40, 75, 60, 66, 80, 90, 70. Thus 10 per cent. was the most and 70 the least frequent of the allowances made; the average was 30 per cent., disability below 10 per cent. not being compensated.

It is a matter both of common law and of regulation, that persons receiving such indemnity would take every reasonable means to favour the cure. It is quite common everywhere for the interested party to pay for the medical treatment. The occupation followed should be one which will favour recovery. The employment of artificial limbs and supports may be a reason for reducing the indemnity.

I. SCHEDULE OF PERMANENT DISABILITY.

The following table shows the percentage of loss of earning power, in the case of unskilled laborers.

I. Head.	Disability per cent.
Limitation of movement	8-16
Bone defect with epileptic attacks	50-70
" " " paralysis right arm	80
" " " " " and leg	100
Scar and bone defect of cranium attacks of pain	33
Hemiplegia following apoplexy	80-100
Headaches and weakness of right arm following depressed fracture.	56-60
Incurable neuralgia	15
Persistent headache, dizziness, nausea following concussion of brain	80
After injury headache, dizziness, epistaxis " "	50
Incurable despondency, irritability and headache	50
Incurable epilepsy only result	30-50
Incurable insanity	100
Permanent weakening of mental faculties, loss of memory	40-50
Weakness, unsteady gait, lessened mental capacity, confusion of ideas following fracture of skull	70-80

	Per cent.
II. Face.	
Disfigurement from scars of cranium (more in case of females)	8-16
Loss of nose do	10-15
Loss of incisor teeth (females only)	5
Defective closure of one eyelid,	8
" " both	16
Incurable trismus	33
III. Eyes.	
Loss or blinding of one (according to employment)	25-33
Loss of both eyes	100
Defective sight one eye	25
Trachoma extreme	100
Chr. conjunctivitis	15
Obstruction of lachrymal duct	8
Nystagmus	16

Josten's table for estimating loss of vision, partial in both eyes.

Rule.—Estimate loss of central vision in each eye if over 50 per cent. (amounts below 50 per cent. disregarded).

Follow column giving the vision of one eye below V. and of the other eye to the right of V, the intersection of the column will be at the percentage of disability for both eyes.

V.	0.50	0.40	0.30	0.20	0.10	0.00
0.50	0	6.5	13.5	20.0	26.5	33.5
0.40	6.5	14.5	22.0	30.0	38.0	46.0
0.30	13.5	22.0	31.5	41.0	50.0	60.0
0.20	20.0	30.0	41.0	52.0	62.5	73.5
0.10	26.5	38.0	50.0	62.5	75.0	87.0
0.00	33.5	46.0	60.0	73.5	80.0	100.0

	Per cent.
IV. Ears.	
Loss of one ear (or complete deafness one ear.)	8
Moderate deafness one ear	0
" " both ears unless occupation needs acute hearing)	0-8
Slight deafness both ears	0
Chr. discharge from ear	15-30
V. Neck and Voice.	
Permanent wearing of tracheotomy tube	50
Inability to read (alexia)	75
Inability to speak (aphonia)	40
Hoarseness or whispering voice	8
Dyspnœa from stenosis of larynx	33-40
VI. Chest.	
Restricted movement from distortion of chest wall, extreme	60
" " " " " moderate	33
" " " " " slight	8
Restriction of movement from badly healed fracture of ribs, extreme	40
" " " " " medium	25
" " " " " slight	8
Intercostal neuralgia	25
Lung disease, severe	100
" medium	40
" slight	0-8
Traumatic heart disease	0-100

VII. Abdomen.

Pain in abdominal wall and inability to carry heavy burdens, due to rupture or sprain of muscles	25
Prolapse of uterus	25
Disturbances of digestion, extreme	80
" " medium	25
" " slight	0
Liver diseases, extreme	80
" " medium	25
" " slight	0

VIII. Hernia.

Hernia in laparatomy scar	25
Umbilical hernia	15
Ventral hernia	25
Inguinal hernia	8
Omental hernia	8
Femoral hernia	15
Hydrocele of cord	15

IX. Genito-Urinary.

Inability to retain urine	15-20
Difficult micturition	15
Loss of penis	8
Loss of testes	15
Urinary fistula	50
Painful enlargement of testes and spermatic cord	15
Rupture or loss of kidney	20

X. Trunk and Vertebral Column.

Impaired mobility, extreme	50
" " medium	25
" " slight	8
Rupture of lumbar muscle, severe	50
" " " medium	25
" " " slight	8
Disease of spinal cord, severe	100
" " medium	66
" " slight	40

XI. Upper Extremity.

Loss of both hands or arms	100
Upper Arm.	
Loss of arm above elbow	66-75
Anchylosis at shoulder	50-60
False joint at shoulder	50-60
Wasting of muscle	25-60
Chronic arthritis of shoulder	16-66
Badly set fracture of clavicle	16-50
Badly healed dislocation, blade injuries	8-50
Forearm.	
Loss at wrist or elbow	66-75
Anchylosis of elbow, extended	40-50
" " semi-flexed	25-33
" " flexed	33-40
False joint	50-60
Diminished flexion or rotation	25-60
Hand or all Fingers.	
Loss at wrist	66-75
Anchylosis of wrist	25-33
Loss of all fingers but not metacarpals	66-75
Anchylosis of all fingers	60-66
Distortion of all fingers	60-66

Single Digits.

Loss of single thumb, loss of metacarpal	25-33
Loss of both phalanges	25-33
Terminal and ½ proximal	16-25
Terminal phalanx	5-10
" thumb tip (special trades only)	0-5
Stiffness 1st and 2nd joint	25-33
" 1st joint	15
" 2nd "	8
Distorsion and fixation in flexed position, extreme	25-33
" " " medium	16-25
" " " slight	8-16

Forefinger.		*Middle finger.*	*Ring finger.*	*Little finger.*
Loss with metacarpal	16-25	8-16	8-16	0-8
Loss of all 3 phalanges	16-25	8-16	8-16	0-8
Loss of 2nd and 3rd phalanges	8-16	8	8	0
Loss of terminal	0	0	0	0
Loss of finger tip	0	0	0	0
Anchylosis of all three joints	16-25	8-16	8-16	0-8
" proximal and middle	16-25	8-16	8-16	0-8
" proximal	8-15	8	8	0
" middle	8	8	8	0
" proximal and terminal	15	8	8	0
" middle and terminal	8-16	8	8	0
" terminal alone	0	0	0	0
Fixation in flexed position, extreme	16-25			
" " medium	8-16			
" " slight	0-8			
Chronic arthritis of one finger	8-33			

Thumb.

Thumb and fore	40-50
" middle	33-46
" ring	33-40
" little	25-33
Thumb, fore and middle	50-60
" " ring	50-60
" " little	40-50
Thumb, fore, middle and ring	60-66
" " " little	60-66
Thumb, middle and ring	50-60
" " little	33-40
" ring and little	33-40
Fore and middle	25-33
" ring	25
" little	16
" middle and ring	40
" " little	33
Middle and ring	16
" little	16
Middle, ring and little	25
Ring and little	16

Combined loss of fingers of both hands.

Loss of all fingers on both hands except one finger on each	100
Loss of both thumbs	50
Thumb and forefinger of one hand, and opposite thumb	55-60
Thumb, forefinger and middle or ring finger of one hand with loss of other thumb	66-70
Loss of all fingers of one hand except forefinger and loss of opposite thumb	75
Loss of both fingers and forefinger	80
Chronic arthritis of several or all joints of both hands	30

XII. Lower Extremity.

Loss of both legs	100
One thigh and one leg	100
Complete paralysis both legs	100
Chronic arthritis	16-66
Injuries to pelvis	16-80
" " fibrous tissues	60
Synovitis	8-33
Tenosynovitis	8-33
Thigh.	
Loss of thigh	75
Anchylosis of hip, extended	50
" " flexed	60
Wasting muscles of one thigh	33
Healing of fracture with diminished motion of knee or ankle or both.	25-66

For neuritis, or injury of nerves 25 per cent. more than for loss of limb.

Leg.	
Loss below knee	66
False joint at ankle	40-66
Anchylosis of knee, extended	40
" " flexed or over extended	50
False joint at knee	60
Foot.	
Loss at or below ankle	66
Injury to ankle bones	16-40
Anchylosis of ankles with flat foot	40
" " with	50
Chr. arthritis of one or more joints	15-50
Toes.	
Lower extremity foot	8
Loss of great toe	10
" any other toe	6
" all toes of one foot	50

With 5-10 per cent. more for loss of metatarsal bones.

II SCHEDULE OF TEMPORARY DISABILITY.

The following table compiled from the statements of the various standard authorities, notably C. Kauffman, shows the duration of temporary disability and some of the commoner complications and consequences of frequent forms of injury.

Explanation of abbreviations :

Numbers indicate duration of disability in weeks ; unless otherwise stated.
p.p.d., permanent partial disability.
per cent., percentage loss of earning power.
p.t.d., permanent total disability.
B., confinement to bed necessary.
H., treatment in hospital preferable.
F., likely to be fatal.
Complications are enclosed in brackets.

HEAD.

Scalp : *Contusions :* Slight, 1—2 ; severe, 2—6 ; (blood cysts, neuralgia, varicosities.)

Wounds ; 1—2, B. ; *Lacerations :* 2—6, B. ; (4—8, F. ; erysipelas, 4—12, H. F. ; wandering erysipelas, 8—26 ; loss of hair ; sensitive to heat and cold ; neuralgia ; insanity ; sensitive scar ; epilepsy, aura from scar).

CRANIUM : *Contusions :* Same duration as scalp wounds ; (osteoma, osteosarcoma).

Fracture : Vault or base, 1—6 months, H. ; often p.t.d. or p.p.d ; (meningitis, F, ; encephalitis, F. ; abscess, F. ; thrombosis and pyæmia, F).

BRAIN : *Concussion :* H. ; immediate unconsciousness lasting hours or days, vomiting.

Compression : H. ; slow, hard, irregular, pulse.

Contusion : H. ; cramps, spasms or paralysis immediately after injury. In compression, contusion and concussion, (mental disturbances ; paralysis ; tinnitus ; headache ; impaired vision, hearing and speech ; tuberculous meningitis ; diabetes ; polyuria ; white softening ; chronic brain abscess—headache and dizziness, exclude ear disease ; brain tumours ; epilepsy ; insanity—connection recognised if early after accident).

FACE : *Cuts, Lacerations and Contusions :* 1—4 ; heal rapidly ; (salivary fistula, 4—8 ; erysipelas, 3—4 ; relapses frequent).

Burns : if superficial, 2—4 ; from boiling liquids and explosions ; deep, or corrosions ; (scarring and disfiguring require plastic operations—important in young women ; obstruction of orifices ; ectropion ; danger of foreign bodies ; paralysis of facial).

Fractures : of nose, 2—4 ; (lachrymal fistula, traumatic ozæna, malposition in setting) ; of malar bone, (rare) 3—6 ; of superior maxilla : 4—10 (necrosis) ; of inferior maxilla, 4—10, (necrosis, aspiration pneumonia).

Loss of Teeth : disfigurement ; (10 per cent. p.p.d to young women for loss of incisors).

EYE.

EYELIDS : *Contusions :* 1—3. *Lacerations :* 2—4 H. ; *Burns :* 3—10 H. ; *Stabs and Cuts :* 1—2 ; (ectropion, entropion, ptosis, 2—4, H. ; operation).

CONJUNCTIVA : *Foreign Bodies :* 1—3. *Lacerations :* 1—3. *Burns :* caustics (keratitis, synblepharon. Trachoma—when case infection occurs accidentally in course of treatment).

CORNEA : *Foreign Bodies :* not infected, 3—7 days ; infected, 1—4 ; (loss of sight).

Contusions : 4—8, (hypopyon, loss of vision, phthisis bulbi, liable to take unfavourable course later).

Wounds : non-penetrating, seen early, 1—2 ; if neglected, 2—6, B. ; (hypopyon). Penetrating cuts and stabs, 1—2 ; not infected.

Lacerations : 6—12, B. ; (iridectomy, cataract later, opacity of cornea—central or peripheral).

SCLERA : *Wounds :* superficial, 1—2 ; perforating,—involving ciliary body, choroid, retina, or with foreign bodies, non-suppurating, 6—12 ; often loss of vision later. Suppurating, 6—26 ; loss of eye, (sympathetic ophthalmia).

IRIS : *Contusions :* paralysis of accomodation, traumatic mydriasis, 6—10. Separation of iris, no operation, 2—4, B. ; operation, 4—12, B. ; (p.p.d., loss of vision).

Foreign Bodies : non-septic, seen early, 2—4 B. ; septic, (panophthalmitis), 4—6 B. ; loss of eye.

Wounds : usually severe iritis, prolapse of iris and dislocation of lens, 4—12 ; (often panophthalmitis).

LENS : *Rupture of capsule :* resorption, 6—8 ; (in young persons). Inflammation, 4—16, H.

Foreign Bodies : opacity and suppuration requiring extraction, and perhaps secondary operation, 2—15 months, H.

Wounds : operation usually required, 2—6 months, H. Note : Resulting

aphakia cannot usually be corrected by glasses during work if other eye sound. Eyes operated on for cataract require almost same compensation as for loss—30 per cent. Loss of eye with cataract suitable for operation, 20 per cent. Blindness of one eye when the other has a cataract, 70 per cent. if successfully operated.

CHOROID, RETINA AND VITREOUS : *Contusion and Hæmorrhage*, 4—8, B.

Retinal Hæmorrhage : slight, 2—6, severe, 4—12 ; (severest forms near macula, good prognosis only if prompt recovery ; detachments usually incurable).

Foreign Bodies : good results in 6 per cent. ; 6—12, H. ; (usually panophthalmitis and sympathetic ophthalmia).

Wounds : non-suppurating, common, 6—10. Suppurating call for enucleation ; 6—12, H.

ORBIT : *Foreign Bodies :* small, not dangerous ; large, dangerous from suppuration ; (meningitis).

Wounds : stabs and cuts from instruments or splinters ; if suppuration, 4—20 H. ; (meningitis, F).

EAR.

LOBE : *Contusions and Abrasions :* 1—2.

Lacerations and Cuts : 1—4.

Burns and Corrosions : 2—6, (deformity or defect ; hæmatoma ; stenosis of meatus).

MEATUS : *Foreign Bodies and Injuries :* 1—2 ; (entrance of water ; blow on jaw ; fracturing skull).

TYMPANUM : *Rupture :* foreign bodies usually in upper part ; (separation of ossicles ; fracture of malleus or stapes) ; indirect injury from blow, fall, alteration of air pressure or explosion ; 1—8, B. ; (purulent otitis prevented by aseptic measures ; curable ; subjective noises may persist for months ; persistence of perforation or recurrence of ear disease).

TYMPANIC CAVITY : *Foreign Bodies :* cause suppuration (meningitis) ; hæmorrhage, 6—15 ; (deafness may persist ; polypus ; bone necrosis ; complete healing necessary before beginning work).

LABYRINTH AND NERVE : *Concussions :* blows and falls, striking head or any part of body ; explosions and noises ; (dizziness, loss of co-ordination, tinnitus) ; 8—12 ; often incurable.

HEARING : Slight or medium deafness of one ear, 0 ; severe, one ear, 10 to 30 per cent. ; deafness of both ears, 30 per cent. ; (chronic ear disease shortens expectation of life ; abscess ; mastoiditis, pyæmia, F).

NECK.

Corrosions and Burns : (scarring and fixation). Injury to deeper tissues from explosions and lacerations ; (larynx, hyoid, trachea, vessels and nerves exposed) ; injury of carotid or jugular, usually F. ; after ligature, paralysis or aphonia in 30 per cent. of cases.

HYOID BONE : *Fracture ;* 3—6 ; (dysphagia).

LARYNX : *Contusions :* may be fatal from shock.

Fractures : dangerous ; also dangerous after tracheotomy from obstruction of tube (disturbance of speech ; difficulty of changing canula ; supervision required).

LOSS OF SPEECH : aphonia and hoarseness ; (asymbolia ; asphasia ; alexia from central lesions).

CHEST.

THORAX : *Concussion :* (shock fatal (?) ; loss of consciousness ; syncope ; heart and lung diseases).

Contusion : slight from blows or falls ; ecchymosis of skin and muscles ; 1—2. Severe, from crushing ; may have internal injuries with little injury of chest wall ; (lesions of heart, diaphragm, and vessels) ; often fatal ; may heal in 1—4 ; or lead to chronic disease.

Fractures sternum, simple, 4—10 ; compound, usually fatal ; (cough, palpitation of the heart, dyspnœa, suppuration and abscess) ; of ribs simple, not dangerous, 5—12 B. ; (danger in old persons of lung disease). Compound fracture, (hæmothorax ; heals after aspiration, rarely suppurates ; pleurisy, heals readily with adhesions, may suppurate, 2—4 months, H. ; (pneumonia, œdema of the lungs, neuralgia at site of injury, fistula, caries of rib,—tuberculous but curable, 2—4 months,—lung tuberculosis most fatal).

Wounds : burns, (scarring and contraction) stabs and cuts are rare in industrial accidents. Wounds of chest and lung generally curable unless involving large vessels, but suppuration common. Heart wounds : not always rapidly fatal, unless in auricles, sometimes curable. Rupture of diaphragm. from falls and run over accidents, usually associated with fatal injuries.

Heart Disease : Traumatic forms include (1) acute endocarditis, (2) chronic endocarditis, (3) rupture of valve, (4) nervous heart disturbance, (5) pericarditis, (6) aggravation of existing disease ; after injury, psychic shock or over exertion. Heart dilatation a prominent symptom in serious cases.

Lung Disease : Traumatic forms include (1) acute traumatic lobar pneumonia, (2) localized traumatic pneumonia, (3) bronchopneumonia, (4) secondary pneumonia, (5) laceration of lung, (6) gangrene, (7) traumatic tuberculosis of lung, (previous condition of lung important, also previous health). Main diagnostic symptom of traumatic pneumonia, early appearance of bloody expectoration.

Traumatic Pleurisy : 50—60 per cent. recoveries.

ABDOMEN.

ABDOMINAL WALLS : *Contusions :* Ruptured muscles from direct violence, overstretching and heavy lifting, usually in recti below umbilicus, 4—6.

Perforating Wounds : without injury of organs, usually heal readily, 2—8 B. ; (prolapse of omentum or intestine).

STOMACH : *Contusions :* from compression against vertebræ, 1—3 months ; (gastric ulcer may follow, 4 per cent. due to trauma, hæmatemesis leading symptom).

Stabs and Cuts : operation immediate ; 2—3 months.

INTESTINE : *Wounds :* same as stomach. *Contusions and Lacerations,* operation imperative ; unless early operation, are fatal from peritonitis, gangrene or hæmorrhage.

LIVER : *Contusions :* occasionally occur. *Lacerations :* common ; result good by early laparatomy, otherwise fatal.

SPLEEN : same as liver ; often no injury to abdominal wall ; (constitutional disturbances after removal).

HERNIA : *Inguinal :* predisposition exists in most cases ; sudden onset necessary to show traumatic origin ; immediate pain and inability to work ; enlargement of ring or enlarged veins point to pre-existing hernia ; causes, direct violence, lifting or heaving, in heavy work ; early examination needed, improbable when simultaneous double hernia, or unilateral hernia with en-

larged ring on opposite side, or inguinal testis exists. Old hernia may be demonstrated: (1) by records of examination for military service, or accident or life insurance; (2) signs of truss; (3) size larger than lemon; (4) irreducible but not strangulated; (5) inguinal canal short and wide. Hernia can rarely be stated to be quite recent; burden of proof rests with claimant. Indications for gradual onset: (1) continuous heavy work; (2) advanced age; (3) statements that a moderate load was found too heavy. Compensation based on 10 per cent. disability, less in females, double hernia same compensation as single as same truss suffices; increase compensation when truss is worn with difficulty or causes inflammation, or if hernia suddenly increases while wearing a proper truss.

Strangulation is to be compensated for if due to injury or over-exertion. Strangulation of a hernia already compensated for may be admitted if a good truss is worn, but not unless worn at time of accident. Always examine both sides to see if recent or old; always test efficacy of truss after application.

Femoral, umbilical and ventral: same as inguinal, but may require more compensation as truss is less easily applied.

KIDNEY: *Concussion and contusion:* haemorrhage and traumatic nephritis; (casts and blood after a few hours, albuminuria); may be fatal.

Lacerations: may be free from symptoms (blood) in a few days, 4—10, H.; hydronephrosis: *floating kidney* requires bandage or operation.

BLADDER: *Rupture:* from direct violence or lifting, one-third of operated cases recover; 4—12, H.

URETHRA; *Lacerations:* in pelvic fractures, 40 per cent. fatal; from straddling falls, 14 per cent. fatal; 6—12, H.; may leave stricture; liable to relapse.

PENIS: *Contusions and crushing:* 2—4; *lacerations:* 3—8 B.; 2—3 months (deformity).

TESTIS: *Contusion and Concussion:* 1—2; liability to sudden death from shock; haematocele, 3—4; hydrocele, 4—6; purulent inflammation, 4—8, (spermatocele and varicocele); loss of testis, 10—15 per cent. if double, or much more if followed by hypochondriasis.

FEMALE GENITALS: *Abortion* from injury of pregnant uterus, *prolapse* from over-exertion; signs of recent origin, pain and tenderness, acute inflammation, absence of chronic inflammation, ulcers, thickening and attrition.

TRUNK AND SPINAL CORD: *Rupture of muscles:* 3—10, B.; lumbago, usually rheumatic in origin, chief difficulty of diagnosis.

Contusions: 1—3 months; contusions of vertebræ, slight, without injury of cord, 1—4 months, B.; severe, may last months or years or give p.t.d.

Fractured vertebræ: 6—12 months. *Dislocation:* same as fracture; (inflammation of spinal membranes, meningocele, meningeal haemorrhage, myelitis or sclerosis of cord, paralysis, bed sores, cystitis, often fatal).

UPPER EXTREMITY.

CLAVICLE: *Fracture:* 5—10; sometimes bilateral; in women greater need to prevent deformity by B. and traction; (injury to nerves and vessels, overgrowth of callus, shortening, disfigurement, false joint may require suture, effect on movement, atrophy of deltoid).

Dislocations: 4—12, H.

Fracture: of blade or acromion, 6—8; usually no permanent disability, but may prevent full motion of arm.

Of neck, 6—12; injury of axillary nerve and paralysis of deltoid; danger of stiffness of shoulder joint and difficulty in raising arm.

SHOULDER : *Contusions :* great functional disturbance at first ; rapidly relieved by treatment ; 4—8.

Sprain : swelling and tenderness in anterior part of capsule ; healing prompt ; 4—8.

Dislocation : if promptly recognized and reduced, 4—8, with no further results ; (separation of great tuberosity and fracture of head of humerus, 6—10 ; if dislocation reduced, may have complete cure ; otherwise, pressure on vessels and nerves require subsequent operation ; primary injury to to nerves or compound wounds ; paralysis of circumflex nerve and atrophy of deltoid ; recurrent dislocation from trifling causes happens when arm is used a few days after reduction ; old dislocations occur through non-recognition in early stage, usually in cases not seen at first, attempts at reduction may cause injury to nerves or vessels or fracture of humerus).

HUMERUS : *Fracture :* separation of great tuberosity often confused with sprain, 8—12 ; old cases good objects for mechanical treatment, 2—4 months, may have p.p.d, from limited mobility in raising arm or chronic arthritis. Anatomical neck or epiphysis, 8—12, best results from extension.

Compound Fracture : 2—4 months ; (injury to radial nerve, operation ; injury of axillary artery, operation, 8—10).

AXILLARY VESSELS : injured by external causes or in reducing old dislocations ; may be fatal ; usually p.p.d. from weakness of arm and disuse of shoulder.

AXILLARY NERVES : *Injury and contusions of shoulder or crushing :* if severe, complete and incurable paralysis of arm ; in slight cases, neuralgia ; (neuritis from crutches).

SYNOVITIS OF SHOULDER : liable to occur in persons carrying burdens or from injury ; 3—6 ; paralysis of deltoid from prolonged rest and fixation, besides causes given above.

SHAFT OF HUMERUS : *Fractures :* 8—12 ; if transverse, extension and B., if fragments override ; compound, non-infected, the same as simple ; if infected, may need amputation ; (nerve injuries, usually to radial ; malposition requires operation ; false joints).

ARM MUSCLES : *Laceration :* of muscles and tendons, common ; in biceps, separation of scapular origin in heavy lifting ; (atrophy and weakness of flexors).

NERVES OF ARM : *Crushing or section :* (neuralgia, suture, stretching, paralysis and trophic injuries, blebs and ulcerations of hands and fingers after injury of median and ulnar). Note : Examine condition of nerves, test sensation, etc., before applying splint, to demonstrate primary injury.

ELBOW : *Laceration or burns of skin :* scarring, 8—12 ; may require plastic operation.

SYNOVIA : over olecranon, injured by falls ; may suppurate if neglected, 4—6, B.

BICEPS TENDON : *Section or rupture :* suture.

ULNAR NERVE : injured in dislocations.

ELBOW JOINT : *Sprains :* usually associated with hæmorrhage ; 4—6 ; stiffness cured by mechanical treatment.

Contusions : posterior and inner surface ; inflammation of bone ; 3—8.

Dislocation : backward most common ; 1—2 weeks after reduction may begin passive motion ; 4—8 ; often limitation of movement.

Fracture through elbow : stiffness is now less frequent owing to mechanical treatment ; compound, good results if properly treated ; (infection ; nerve injuries, p.p.d. mostly from interference with nerves of hand).

Fracture through condyles. 8—12, B. ; deformity, operation.

FOREARM : *Contusions :* usually heal well, even with much swelling ; 4—8;

Wounds : often complicated with injury of vessels, nerves, and tendons ; suture beneficial even years later.

Crushing : causes extensive separation of skin ; 4—12 H. ; often p.p.d.

Fractures : of both bones, 8—12 ; (ischæmic paresis of muscles from tight bandaging ; at first easy to treat ; if only noticed after removal of splints, leaves permanent effects ; interference with pronation and supination from bony adhesions, callus, or malposition, require operation ; false joint, may not cause disability ; in other cases, operation and fixation needed ;) treatment by extension in supine position.

Fractures of Ulna : in upper third, often dislocation ; 8—12 ; old neglected cases cause functional disturbance, operation and resection of head of radius or ulna.

In middle or lower third, 8—10 ; (pseudarthrosis or impaired rotation).

Fractures of Radius : in upper and middle thirds, pseudarthrosis if fragments not opposed but one supine and the other prone ; in lower thirds, Colles' Fracture forms 10 per cent of all fractures, often called fracture of the forearm ; or treated as sprained wrist ; 3 weeks fixed and 4 weeks gymnastics ; massage good, even in worst cases, but may take one to two years. (fracture of lower end of ulna may leave pain and disturbed function ; comminution of lower fragment, or fractures of carpal bones ; compound fractures, results bad ; worst results due to paralysis from tight plaster bandage).

WRIST : *Sprain :* massage, 2—4 ; with rest treatment, function disturbed for months ; heavy work might be better done than delicate hand movements.

Dislocation : rare ; usually means fractured radius.

Tenosynovitis : suppuration, 2—4 ; often relapses ; common about thumb in certain occupations, smith, carpenter, joiner, farm labourers, washerwomen.

HAND AND FINGERS : *Contusion and crushing :* from severe injuries, hence often protracted ; in crushing of ungual phalanx, remove nail to lesson risk of infection ; 2—4.

Sprains : 2—6 ; often lead to stiff joint with thickening ; benefited by massage.

Dislocations : rare ; Röntgen ray examination important ; 3—6 ; operation gives good results.

Fractures : bony union, 3—8 ; if soft parts are much injured and inflamed, 4—12 B.

Wounds : early treatment important ; first aid should be simple, water dressings, or iodoform gauze ; unskillful use of carbolic acid or perchloride of iron liable to produce gangrene ; infection of wounds most important, and phlegmon may occur through infection by callosities or small foreign bodies.

General considerations for assessing cases of hand injury.—The younger the person the greater the chances of adaptation ; new conditions or change of employment and ultimate improvement of condition ; heavier compensation needed for old persons. Sex : Men are better able to find work with injured hand than women, as the latter do chiefly fine hand work ; common labourers do not use fine finger movement ; skilled labour needs especially high compensation, if the injured finger is used in special occupation ; women require special compensation for deformity ; previous injury, if not already compensated for, should increase the amount of disability. Estimation of the loss of power of hand or arm to be tested quantitatively, the angle to which flexion is possible and the force in various parts to be tested and compared with the opposite hand ; the special effects of injury to nerves, as seen in the claw hand from beginning of the ulna nerve with the loss of

apposition of thumb, in which the hand is quite useless. In the radial nerve, absence of extension and abduction ; in median nerve, loss of apposition, separation of fingers, loss of power of flexion, Dupuytren's contracture and ulcers also cause disability. Degeneration of muscles, electrical tests.

LOWER EXTREMITY.

PELVIS : *Contusions :* extensive ecchymosis, removable by puncture ; separation of tissues (infection) ; 4—8, B.

Sciatic Nerve : Contusions : by falls or tumbling over when kneeling or squatting ; 4—12, B. ; (cramps and prolonged sciatica, nerve stretching, or section and suture).

Fracture : often multiple, always severe, gravity depends on implication of pelvic organs especially urinary tract ; 2—4 months ; (injury of urethra, 3—6 months, H. ; often p.p.d. ; fracture through acetabulum may affect hip joint).

HIP : *Contusions :* often present extensive separation of the skin and extravasation of blood or lymph ; 4—8, B. ; (contusion of groin, 1—2 ; often infected from injury of glands ; rupture of psoas muscle, after severe exertion, pushing or lifting, 4—10, B. ; injury of great vessels, danger of immediate bleeding, or gangrene of whole or part of leg).

HIP JOINT : *Sprains :* rare.

Contusions : falls on trochanter ; 3—6, B. ; if simple contusion healing good.

Dislocations : reduced when recent, 6—12, B. ; fracture of acetabulum may make reduction harder, extension apparatus, 8—12, B. ; in fracture of neck, dislocation unreduced ; with union in good position, the gait is less disturbed than in simple unreduced dislocation ; injury of great vessels may cause death from bleeding and gangrene ; old unreduced dislocation may be reduced without operation, but latter is preferable ; in unreduced dislocation, first, crutch used, then stick ; if paralysis and pain remain from head of femur, it should be resected.

THIGH : *Contusions :* extensive and severe functional disturbance ; 4—10, B.

Laceration of muscles : adductors or quadriceps ; in tendons, suture required ; results good ; 4—8.

Wounds : complicated by infection, dangerous ; after injury of large vessels, gangrene ; crushing commonly from run-over accidents.

FEMUR : *Fracture :* of neck ; intracapsular, rarely gives bony union in old people, 2—6 months ; always have partial or total stiffness of hip-joint and shortening with a limp ; (in old people, often bed sores and hypostatic pneumonia ; extension and long splint) ; usual cause, external violence in long axis or axis of trochanter ; rarely spontaneous ; in impacted fractures, may walk with stick ; often only sprain diagnosed and short rest in bed ordered ; these cases later have profuse callus and anchylosis of joint.

Of shaft, simple, 3—4 months, H. ; extensive twisting or separation of fragments, common ; treatment by plaster ; shortening usually considerable ; malposition may require osteotomy ; compound fractures heal well with good treatment, but if thigh is crushed, amputation indicated ; (malposition ; shortening ; false joint. requiring fixation apparatus and walking out ; stiffness of knee joint from inaction, requiring gymnastic treatment ; relaxation of ligament, needing apparatus ; atrophy of quadriceps ; paralysis of peroneal nerve, from over extension of knee).

KNEE : *Wounds :* from falls, corrosions, cuts and bites ; danger to popliteal vessels ; in neglected cases, purulent arthritis,

Contusions: with bloody effusions, 4—8, B. ; on knee cap, bleeding into bursa.

Sprains: slight, 1—2 ; severe, 4—12, B. ; (relapsing synovitis with effusion ; uncertain gait ; fatigue and tendency to fresh sprain ; may cause fixation ; stiffness in joint with exostoses, p.p.d. ; muscle atrophy ; rupture of internal lateral ligament, 5—10, B., and apparatus, 1—2 years.

Dislocations: from severe violence ; good results if seen early ; 2—4 months, H. ; anterior and posterior dislocations often complicated by injury to vessels.

PATELLA : *Dislocation:* 3—12, B. ; readily healed if replaced but liable to recur ; if unreduced, motion is impaired.

SEMILUNAR CARTILAGES : *Rupture:* impaired motion requiring operation ; floating cartilage.

KNEE JOINT : *Fracture through:* 8—12, B. ; if comminuted, 8—16 ; if transverse ; (fibrous union ; with stiffness, mechanical treatment, 1—2 years ; weakness of quadriceps) ; fracture through condyles, 8—16 ; fracture through upper end of tibia, 12—24 ; (stiffness often results).

LEG : *Wounds: contusions: abrasions:* periostitis often diagnosed when merely bandaging bad ; varicose veins, special care necessary ; also if scars or ulcers are injured ; varicosity aggravated by accident and may lead to ulcer ; to be compensated if the direct result of accident ; *thrombosis* common, apart from varices ; pain felt in leg with swelling following ; patient may work one or two weeks with increasing pain before disabled ; if the accident can be proved, thrombosis may be regarded as due to it even if work is continued during interval.

Laceration: of muscles and sinews in calf, often tendo achilles ; 8—12 ; suture.

Fracture: shaft ; usually of both bones ; 4—6 months ; (stiffness of joints from disuse, massage beneficial ; malposition often leaves an angle ; pain from pressure on nerves ; eversion of foot, osteotomy ; X ray diagnosis important, callus at first transparent ; may have delayed union and false joint ; if treated by plaster splint or allowed to walk with apparatus, operation rarely necessary ; swelling of foot and ankle from interference with vessels or thrombosis relieved by massage to restore muscular tone, or by passive motion of joint ; thrombosis likely in advanced age ; compound fractures, 4—8 months, H. ; often leave necrosis of bone, fistulæ or ulcers).

ANKLE : *Sprain:* usually from falling or jumping ; best results from massage ; 2—6 ; (swelling and radiating pain ; uncertain gait and tendency to sprains ; stiffness, if kept at rest during the cure ; good results by massage and mechanical treatment ; persons with varicose veins suffer most).

Dislocation: anterior or posterior ; 8—12, B. ; less disability from badly healed anterior than posterior : calcaneus position than equinus ; operation with good result ; subastraguloid, prognosis good ; in neglected cases, only hope of improvement is operation ; dislocation with fracture of astragalus, good if replaced, otherwise pain persists.

Fractures: through malleolus of fibula ; 2—4 months ; position of foot most important ; danger of subsequent stiffness.

Fractures compounded from injury by bone fragments ; (pressure necrosis ; swollen foot and leg ; thrombosis ; embolism ; and stiff joint, permanent if from callus, often in equinus position ; *flat foot* if fibular fracture set without correction of position ; prevents climbing and standing long, never perfectly healed benefitted by plate).

Fracture of malleoli, either the inner or outer may break ; the commonest form, Pott's fracture of fibula and inner malleolus. Fractures above malleoli often comminuted, may need resection if compound, 3—6 months, H.

Fracture at tibio-fibular ligament may occur with sprain; in neglected cases, often leave flat foot.

Fracture of astragalus from falling or jumping often complicated with fractured malleoli and leaving club foot deformity ; may leave severe results, especially anchylosis ; X ray diagnosis important in all injuries of ankle.

Foot and Toes : *Wounds :* neglected, dangerous from sepsis and phlegmon ; scars on soles, bad and may need operation ; special liability to tetanus ; infection may follow trivial wounds.

Contusions : 4—12 ; walking painful ; badly fitting shoes aggravate the condition ; bed and warm applications necessary.

Sprains : in cases of flat foot, very serious ; may take years to heal.

Lacerations : with rupture of tendon and fascia ; the plantar fascia may be tender for months.

Fracture . of os calcis, pain in standing and walking may persist from 2—4 years ; of metatarsal, often interferes with function ; pain after removal of bandages may last for months causing limping and limited use of foot.

Rules for assessing injuries of lower extremity.

(1) Certain occupations especially require steady footing ; if climbing and good balancing necessary, motion must be free.

(2) In other occupations much walking is needed.

(3) In others, prolonged standing. For all these, good restoration of function needed, not merely cure of the injury. It should be noted whether the person can acquire a sedentary occupation and do his work sitting, or if he can only do light indoor work. The p.p.d. in lower extremity is 40—75 per cent. after serious injuries. The actual motion of the joint is to be accurately noted.

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ON THE ESTABLISHMENT OF MEDICO-LEGAL DIPLOMAS.[1]

BY WYATT JOHNSTON, M.D., MONTREAL, CAN.

THE points I wish to discuss are:
Is there a need for such diplomas?
What should be the standard adopted?
How can the teaching be provided for?
How is the diploma to be recognized?

I. NECESSITY FOR MEDICO-LEGAL DIPLOMAS.

The selection of medico-legal experts may be arrived at in three ways: First, *partisan selection*, or leaving the choice to the litigants. This has the inevitable result of a conflict of opinion, and has led, in America and elsewhere, to the development of a class of men whose ingenious, though perverted, ideas of medico-legal matters have had a maximum tendency to bring the subject of medico-legal expert work into general disrepute, with a minimum amount of compensating benefits in the way of addition to our scientific knowledge.

The second plan, or *arbitrary selection* by the judicial or state authorities, which has been followed until recently in France, and is being constantly advocated with us as a panacea for the existing evils in connection with medico-legal testimony. This plan has the great advan-

whom as a class we owe most of our recent prog-

[1] Read before the Massachusetts Medico-Legal Society, June 11, 1901.

tage of developing a number of skilled officials, to ress in medico-legal knowledge. The drawbacks are: That it is only applicable under specially favorable conditions, as regards the constitution of courts in regard to stability and freedom from bias; also it has a tendency to limit the rights of litigants, beyond what is consistent with our legal traditions and usage. In France this method has proved so defective, owing to the egregious blunders of individual experts, that its abandonment has been recently decreed by the Cruppi Law, regulating the appointment of official experts for the defense in criminal cases.[2]

The third plan, that of *selection by special qualification*, has been successfully followed for the last fifty years in Germany and Austria, and has now been introduced into France by the establishment of a post-graduate diploma course in legal medicine. It is difficult to see how any system of selection of medico-legal experts can be successful, unless some adequate, defined and reasonable standard of technical training be established. This has the advantage (1) of ensuring that all who receive appointment as experts will have had suitable preparation; (2) of permitting the individual selection by state and judicial authorities from among these; (3) of affording a means whereby any one desiring to take up such work can show that he possesses the necessary training; and (4) of protecting properly qualified persons from the competition and rivalry of the unqualified.

Thus the establishment of medico-legal diplomas would appear to be the first step in any scheme for reforming expert testimony, whatever the particular methods followed in making appointments.

[2] Semaine Méd., July 5, 1899.

II. STANDARD OF MEDICO-LEGAL DIPLOMAS.

Under the German system, the required qualification for official medico-legal work is the passing of the *physikats-examen* subsequent to graduation and licensing. The amount of special study required is about six months, and the standard demands fitness for the performance of official public health, as well as medico-legal work, the appointees, as official government physicians in small centres of population, being entrusted with both kinds. This is inapplicable where (as with us) the public sanitation and medico-legal matters are under separate and independent control, and it has not been found in any case that the exaction of the double qualification in State Medicine has much in its favor, as the sanitary districts are as a rule much smaller than the medico-legal ones.

The best standard for general adoption in medico-legal diplomas is that recommended by Professor Brouardel, in 1884, for adoption in France, but only accepted in 1900, and which is fully explained in the published report by Professor Brouardel.[3]

The qualification demands a full academic year (nine months) post-graduate study in legal medicine, including practical autopsy work, elementary toxicology, study of nervous diseases, medico-legal methods in examination of stains, questions of sex, etc., and a knowledge of medical law and responsibility. Courses in legal medicine are not compulsory for medical students in France or Germany, whereas most of our teaching bodies go to the opposite extreme — of supposing that all medical students are qualified for the work on

[3] Bulletin of the Paris Faculty of Medicine, February, 1900; and Annales d'Hygiene Publique, Nov. 11, 1884.

graduation, by the too often very inadequate courses in legal medicine provided for in the ordinary medical curriculum.

In arranging (jointly with Prof. George Wilkins of Montreal) the schedule for the medico-legal diploma[4] which was adopted by the Medical Faculty of McGill University in November, 1900, we followed the requirements of the Paris diploma as regards the scope of the course and length of study required. We selected, however, the model followed in the British diploma of public health, of demanding at least six months' practical training as assistant in medico-legal work, of all candidates, a precaution which seems very necessary. We also included among the requirements a practical training in the medico-legal study of disability after injury. This branch of legal medicine I consider to be really more necessary to the ordinary medical student than the medico-legal

[4] Candidates for the diploma must possess a degree in medicine, or other qualification for practice, and present certificates of having attended the following courses: (1) A course of six months' scientific study in legal medicine, consisting of systematic lectures and practical medico-legal instruction in laboratories and elsewhere. (2) A course of six months' training as assistant in medico-legal practice. The candidate shall produce a certificate, or certificates, satisfactory to the faculty, that he has continuously and actively assisted in the regular duties of some medico-legal expert recognized by the faculty. Of the two six-months' courses referred to in this and the preceding paragraph, not more than three months shall be concurrent. (3) A special course, or courses, of lectures in legal medicine and mental diseases. (4) A practical (laboratory) course in toxicology. (5) A course of instruction in the law relating to medicine, and to the status, rights and responsibilities of the physician. (6) A series of short courses, with demonstrations upon the following subjects: (*a*) methods of conducting medico-legal autopsies; (*b*) the methods of medico-legal microscopy; procedure in the examination of blood stains, etc.; (*c*) methods of skiagraphy; (*d*) methods of procedure in cases of wounds and injuries; (*e*) method of procedure in cases of assaults upon women and children; (*f*) methods of procedure in cases of abortion and infanticide; (*g*) methods of procedure in cases of the determination of sex and paternity; (*h*) methods of procedure in cases of offence against morals; (*j*) methods of procedure in the study of mental conditions; (*k*) methods of procedure in examination for life and accident assurance; (*l*) estimation of compensation for injury. No candidate shall be admitted to any portion of the examination for this diploma until at least one full academic year has elapsed since his graduation in medicine.

autopsy training which he usually receives, since the majority of practitioners will rarely, possibly never, have to make medico-legal autopsies in their practice, whereas in each year they will almost certainly have on several occasions to examine or testify in reference to personal injury cases.[5]

The amount of instruction provided for on toxicology and mental diseases is intended rather to qualify the expert to co-operate intelligently with a skilled analyst or alienist, than to undertake the work independently. For independent work in either of these branches, at least a year's additional special study and practice as assistant would be needed.

One of the difficult problems to settle is that of establishing a standard of special training in pathology, to qualify as a specialist in medico-legal autopsy work. It would seem that we have the choice of training either a type of medico-legal specialist in one line of work, or a type of all-round medico-legal practitioner, and the question of deciding what options of study are permissible, after the general work is covered, is a new one. The exaction of a year's additional study for the medico-legal specialist seems to be a reasonable minimum.

III. ARRANGEMENTS FOR GIVING COURSE.

Most medical schools have the means of giving the necessary instructions, provided that, by co-operation with the courts, sufficient access to material can be had. It will be found as a rule that an offer to give free instruction to official nominees, in return for the privilege of utilizing the material for instruction, will be effective. It is

[5] Prof. F. W. Draper of Harvard has for years included instruction in this branch in his course to medical students, the work being in charge of Dr. Dwight.

very desirable that the practice followed in Germany, by which those who have received official appointments return at intervals of three years for a three-months' supplementary course of instruction at government expense, should be introduced. The plan of giving medico-legal clinics on groups of selected cases makes an interesting method of teaching, and the pupils have an opportunity of studying the cases independently. Enough suitable cases generally occur in the routine material of any large hospital.

It is a relatively simple matter to secure among the teaching staff some who are specially interested in legal medicine, and who have special knowledge of neurology, chemistry, obstetrics and other topics, on which strict practical courses have to be given. One often wonders why our numerous post-graduate schools and polyclinics have not established a high standard of post-graduate instruction with rigorous examinations. They would probably find the hospital and other authorities ready to support them by exacting such qualifications in making appointments.[6] Our post-graduate instructors, being free from the burden of under-graduate teaching, might be fairly expected to make a better showing in this direction than is actually the case. The time must be past when a simple degree or license adequately guarantees fitness for all medical positions.

IV. RECOGNITION OF THE DIPLOMA.

While it is hardly to be expected that the State authorities would be at once disposed to recognize officially the possession of a diploma as indispensable for all medico-legal experts, yet the influence of recognition by medical societies, medico-legal societies, bar associations and the bench, would in itself probably be sufficient to

make the qualification worth obtaining by those engaged in medico-legal work. The want of any such qualification would also tell somewhat against the amateur expert.

It would thus appear that, pending general official recognition, those engaged in medico-legal work would be aided by the establishment of medico-legal diplomas.

A SIMPLE METHOD FOR BACTERIOLOGICAL EXAMINATION OF MILK SAMPLES.

(From Hygienic Laboratories of the Provincial Board of Health and McGill University, Montreal.)

BY

WYATT JOHNSTON, M.D. AND F. B. JONES, M.D.

Reprinted from the Montreal Medical Journal, February, 1902.

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BY

WYATT JOHNSTON, M.D., AND F. B. JONES, M.D.,

(From Hygienic Laboratories of the Provincial Board of Health and McGill University, Montreal.)

We desire to report briefly some observations we have made with a view to simplifying the technique for the bacteriological examination of milk so as to permit of this very useful and important test being more generally used than is the case at present.

The method commonly recommended for the bacteriological examination of milk is to estimate the number of bacteria growing on gelatine plates. This gives information of great practical value, for one can readily recognize milk in good condition from that which is bad. We know that a rigid observance of the most scrupulous care in the care of the animals, in milking, storing, airing, cooling and shipping the milk as well as proper precautions as to cleanliness of the utensils and the hands of the milkers are all necessary to keep the number of bacteria in fresh milk below 10,000 per cubic centimeter at the time of delivery.

The importance of this is only now beginning to be realized by the health boards and the medical profession. We have, in fact a ready means of controlling the milk supply, sufficiently simple to be carried out by a medical man, or by the head of the family, or the milkman himself, and capable of being popularized, as in the case of the taking of samples of blood, sputum or throat exudate.

The method of making cultures for this purpose as usually carried out is by diluting 1 cc., or a fraction of 1 cc., of milk taken in a sterilized pipette with 500 or 1,000 times its volume of sterilized water in order to obtain a convenient number of colonies, for counting, in the plate culture. This can only be conveniently carried out in a laboratory, and, as the number of bacteria in milk tends to increase rapidly, delay in transmitting to a laboratory is a serious source of error.

It has been found by those occupied with the bacteriological examination of milk that the use of a wire loop gives sufficiently ac-

* Read before the Montreal Medico-Chirurgical Society, January 7, 1902.

curate results to enable the dilution to be dispensed with. This plan appeared to us to be the one most suitable.

A series of comparative tests with the loop and dilution methods gave the following results:

Loop method.	Dilution method.
27,000	33,000
37,000	36,000
139,000	160,000
187,000	194,000
48,000	54,000
480,000	460,000
160,000	172,000
546,000	530,000
83,000	64,000
134,000	169,000

It will be seen that while the dilution method is somewhat more uniform the difference is slight, and the loop method gives results near enough for the work in question.

We have found that a wire of No. 25 gauge when formed into a loop which fits closely over a knitting needle, size No. 18, furnishes a loop which takes up with great constancy 1-1,000 cc. of milk, which we have found the most convenient unit to work with. This may only apply to the materials we have personally tested, as the gauge both of wire and needles varies somewhat, and it is necessary to calibrate them.

The outfit used is extremely simple. The following is taken from the card giving instructions to those making use of the test.

BACTERIOLOGICAL DIAGNOSIS OF MILK.

Directions for Use.

1. Melt the gelatine by warming it just enough to make quite fluid; do not heat it strongly.

2. Inoculate the melted gelatine with one loopful of milk by means of the wire loop. Take care to avoid contaminating the loop by contact. Shake up the vessel containing the sample. Avoid taking the sample from the surface cream or from the sediment.

3. Distribute the milk uniformly throughout the gelatine by sharply rotating the bottle without actually shaking it. Let the bottle lie on its side till the gelatine is quite firm. Take care that no gelatine reaches the cotton wool stopper. Cooling may be hastened by laying on any cold surface.

4. Fill the stoppered tube with a sample of the milk for control. Return the wire loop with the outfit.

5. Fill out the card and transmit the outfit to the Laboratory of the Board of Health, Province of Quebec, 76 St. Gabriel Street, Mont-

real. Take care that the outfit is not exposed to a temperature above 70° F. or below 35° F. so as not to melt the gelatine or destroy the bacteria by freezing. A report will be made as a rule on the second or third day.

N.B.—In case of pasteurized milk 10 loopfuls to be taken.

The loop of aluminum wire (suggested by Dr. L. B. Wilson as a substitute for copper wire which we first used) is furnished sterilized in a corked test tube, also sterilized. This is placed in a small mailing

case together with a flat sided Blake vial containing nutrient gelatine ready for use, a tag to serve as a label and a slip of printed directions for use. For ordinary milk one loopful suffices for the inoculation. In the case of pasteurized milk or milk likely to contain less than 5 colonies per cc. ten loopfuls are preferable. The development of exclusively spore-bearing bacilli is evidence that the milk has been sterilized.

In order to make sure that the result of a low count of bacteria is

not due to the addition of preservatives these can be tested for, if the sterilized tube containing the wire loop is filled with milk and returned with the sample. An amount of 20 cc. suffices not only for this, but also for testing the fat and total solids if desired, or for making a microscopic examination of the sediment obtained by centrifuging. In the case of milk containing large numbers of bacteria the detection of preservatives is less important.

Arrangements have been made by which these outfits are supplied at the request of physicians or milk dealers by the Board of Health of the Province of Quebec. A source of error of which little is generally known is the reduction of bacteria which occurs when milk is frozen, so that this must be guarded against in rigorous weather.

The question of adopting fixed standards for bacteriological purity of milk is one which has recently attracted some attention. It may be conceded that 500 colonies per cc. is about the maximum permissible for pasteurized milk. The explanation which we have had printed on the back of our report is the following:

EXPLANATION OF REPORT.

Class I.—First class milk should not contain more than 10,000 bacteria per cubic centimeter.

Class I.—With 10,000 to 100,000, the milk, while not first-class, may be still usable.

Class III.—With 100,000 to 1,000,000, it is in a decidedly poor condition and had better be pasteurized before use.

Class IV.—Milk having over 1,000,000 has deteriorated to an extent which makes it objectionable as a food.

Milk containing any chemical preservatives is unfit for use. Good pasteurized milk should not contain more than 100 to 200 bacteria per cubic centimeter. In order to keep the number of bacteria below 10,000 limit at the time of delivery, great care must be exercised in milking, stirring, cooling and shipping the milk and failure to secure this result will be found due to the neglect of some essential precaution. At temperatures below 40° or 50° F., very little increase of the bacteria occurs.

This agrees in the main with the classification adopted by the milk commissions of this society: "Milk not pasteurized and not delivered in the city within four hours after milking must not contain more than 30,000 bacteria per cc. at any time of the year. Fresh milk with less than 5,000 colonies per cc. to be classed A.A.; with 5,000 to 15,000 A.; with 15,000 to 30,000, a; above 30,000, B.; above 100,000, C. Milk

not to be considered as germ free unless pasteurized twice. Acidity of the milk not to exceed 0.2 per cent."

This standard has been finally adopted by the city health department for over a year, but it will be noticed that not one of the samples given in the table comes under the heading Class A, or even "a" and that two-thirds of them fall in Class C, although taken in December, at the most favourable time of the year.

We think that the method recommended is so simple that any milk inspector could reasonably be expected to make a bacteriological examination of all samples collected as a matter of routine, and at the same time to give any persons interested in the purity of the milk supply an opportunity of obtaining direct information about any particular milk without expense or unnecessary delay.

A NEW MORGUE FOR THE CITY OF MONTREAL.

A NEW MORGUE FOR THE CITY OF MONTREAL

To the Editors of THE MONTREAL MEDICAL JOURNAL.

DEAR SIRS,—We have taken the liberty of suggesting the arrangements necessary, from a medical point of view, in connection with a morgue for the city of Montreal, as the matter keeps cropping up from time to time in the City Council, and no very definite idea appears to exist as to what is needed.

One of us recently had an opportunity of visiting the morgues of the following cities: Boston, New York, Philadelphia, Baltimore, Cincinnati, St. Louis and Chicago, for the purpose of studying the arrangement and construction of buildings of this description, and the other has taken occasion to do the same with the morgue at Paris.

The general arrangement, apart from medical matters, is a matter into which it would be out of place for us to enter fully here, as it does not come within our province as physicians. We will therefore simply state that it should be centrally placed and be in direct communication with, or form part of a police station, in order that all matters concerning the guarding and identification of bodies shall be in the hands of the police.

In Montreal the morgue would naturally have to contain a coroner's office and a court for holding inquests, with a separate room for witnesses. Some provision for the safe keeping of the records is also necessary. There should be an apartment for the janitor or guardian of the building.

MEDICAL ARRANGEMENT.—The essential requirements are (1) the mortuary, (2) the room for autopsies, (3) the laboratory.

1. *Mortuary.*—A mortuary is required for keeping bodies

during the pending of a medical examination or inquest and for keeping unknown bodies as long as may be necessary for identification. As this must be done without danger to health and without creating a nuisance, some means must be employed to prevent the decomposition of the bodies.

The system of cold storage in a dry atmosphere at about the freezing point is the only means which has been found suitable for this purpose elsewhere and has the advantage of costing very little, after the apparatus has been constructed.

A cold storage chamber suitable for the requirements of Montreal could be made at a cost of about $1,200 by building a chamber with double wooden walls, the ceiling consisting of a galvanized iron tank filled either with brine cooled artificially, or simply with a mixture of ice and salt. By this means the temperature in the air space below can be maintained at the freezing point for an indefinite time and all danger of the nuisance and danger to health arising from putrefaction avoided.

In Montreal, where ice is cheap and plentiful, its use would be probably found more economical than that of artificial means of obtaining cold, especially if the ice was stored on the premises. In any case, if it was subsequently found advisable to substitute some artificial process, such as the ammonia distillation or expansion, no further change would be required than simply fitting the expansion tubes into the tank. By either artificial or natural cold the cost ought not to exceed a couple of hundred dollars per year. The use of cold would only be necessary during the period from May to November; during the remainder of the year sufficient cold can be obtained from the outside air by having a flue opening outside.

Some provision for refrigerating is absolutely essential for medico-legal purposes in Montreal, since during the summer, especially in the case of drowned bodies, the effects of putrefaction make identification uncertain and the results of autopsy unreliable, under ordinary condition s

in the course of a few hours. In addition, where it is necessary to preserve organs for a time, pending inquiries as to the necessity of a chemical analysis, putrefactive alkaloids are produced in them which tend to confuse the results of the chemist and at the same time the poisonous alkaloids tend to become destroyed.

Whether it is desirable to make a place where the bodies of unknown persons are kept, a place of public show, which any person shall be allowed to visit through morbid curiosity, or whether the view of such bodies should be restricted to those who are professionally or personally interested in their identification, is a question which can best be decided by the police. There is much to be said on both sides, but certainly the less the idea of a morgue is made repugnant to the general public the better. In any case means should be taken to secure that bodies which have been identified shall not be needlessly exposed, and that as far as possible inquirers shall only be obliged to see such bodies as correspond in sex, age, etc., with the individual whose identity it is sought to establish.

By keeping a description of the body, clothing, etc., together with a photograph, in the coroner's office, it could be at once stated whether any of the bodies in the morgue corresponded with those sought. Photographs suitable for this purpose can readily be taken with an ordinary Kodak camera and can be printed at a cost of only a few cents each. Such photographs would, in addition, form a permanent proof of the appearance of the body if it finally had to be buried unidentified.

The chamber for viewing these bodies should be so placed as to be accessible without interfering with the other parts of the morgue. It could be illuminated either by daylight or by electricity, in which latter case the consumption of ice would be decidedly less.

In connection with the mortuary there should be a room in which undertakers could lay the bodies in coffins and in which the bodies could be received. This should have a

separate entrance apart from that used by the public and situated in the rear of the building.

The mortuary should communicate directly with the post-mortem room. The jury could view the body, if necessary, in the mortuary.

2. *Room for Autopsies.*—The autopsies should be performed in a large, well-ventilated room lighted partly from above. This should be furnished with a suitable dissecting table and instruments, weights and measures. Hot and cold water should be provided and there should also be a good artificial light, in case examinations have to be made at night.

It is very essential that accommodation should be provided for physicians, medical students, law students and other persons interested in medico-legal investigations, so that they may have the benefit of seeing autopsies performed. The absence of some such arrangement at present is a serious drawback to the proper education of physicians in medico-legal duties, the result of which is only too apparent throughout the country.

Provision should of course be made by which, when it is considered necessary by the judicial and police officials, an autopsy may be perfectly private and only witnessed by such persons as are officially necessary. This restriction might seldom require to be enforced, but should be left under the control of the coroner.

For autopsies which have to be held privately, or where more than one autopsy has to be held at the same time, a smaller post-mortem room is required, connecting directly with the mortuary and placed in a part of the building not accessible to the public. By this means, when it is necessary to order the removal of a body from a private house in order to secure a more thorough medical examination, the friends and relatives could be assured that the body would not be exposed to the public view.

3. *Laboratory.*—In connection with the autopsies microscopical examination is often necessary before an opinion

can be given, and the examination of blood-stains, hairs and other substances is often necessary. For this reason a small laboratory, provided with the necessary apparatus and fittings, should adjoin the post-mortem room.

Part of this labratory should be arranged for making simple chemical examinations, especially of the blood, urine and the contents of the stomach. It would probably not be expedient to provide a fully equipped chemical laboratory for elaborate chemical analysis, as this work is better entrusted to some regular chemical laboratory in important cases. In poisoning by certain volatile substances, notably prussic acid, it may be necessary to have the reagents for a chemical test immediately at hand. In connection with the laboratory a supply of jars suitable for preserving organs for analysis should of course be always available.

In poisoning cases it is often necessary to perform experiments in order to establish the poisonous nature of the substances isolated by the chemists. As such work alone, should be made jointly with the physicians of the morgue, a room for keeping animals under observation should be available somewhere in or near the building.

In connection with the autopsies it is often necessary to consult books of reference in regard to a number of special anatomical and other details which it is impossible to retain in the memory. A collection of standard books on legal medicine should therefore be available.

It is often necessary while studying an injury to compare it with other similar injuries, and for this purpose a collection of medico-legal specimens should be available. A small museum would not necessarily involve an increase in the size of the morgue, as the rooms containing it could be also employed for other purposes.

The maceration of skeletons is from time to time necessary in order to determine questions of identity or to study the nature of injuries. Specimens exhibited in court are often of great value in explaining the true nature of injuries to the jury.

In concluding, we wish to state that there are two classes of morgues: The one, are those employed simply as places of deposit for dead bodies; the other those which form recognized centres of medico-legal investigation and teaching, and have raised legal medicine to its present important position. As both classes of morgue cost practically the same sum to build and equip, the future success or failure of a morgue depends upon how it is designed and above all how it is conducted. It is to be hoped that in providing a morgue those in authority will furnish one in every way worthy of this city.

A building such as we have suggested could, as has already been pointed out by Coroner McMahon, be constructed by capitalizing the sum now annually paid by the coroner's court in connection with the transport and care of bodies, rooms for juries, and so forth. If built in connection with the police station the annual cost of heat, lighting and attendance would form but a trifling amount.

In the American cities mentioned above, and in most European cities, the construction and running expenses of morgues are met by the municipalities. In the case of Montreal it appears as if expenses in connection with transport, preservation, guarding and burial of bodies, as well as arrangements to prevent nuisance or danger to health, should legally belong to the city, while those in connection with judicial or medical study of the cases should be borne by the Province.

No doubt an amicable arrangement could be readily arrived at by which a satisfactory service could be obtained at an outlay not exceeding that required for our present very primitive arrangements.

(Signed) WYATT JOHNSTON.
GEORGE VILLENEUVE.

REPORT OF COMMITTEE ON TEACHING OF HYGIENE AND GRANTING OF DIPLOMA OF DOCTOR OF PUBLIC HEALTH.

Your committee during the past year has studied the question of what may be considered the normal educational standards of hygiene, and wishes here to acknowledge the valuable assistance received from persons not on the committee, in this matter. It was considered that the subject should be classified under the following divisions:

1. Hygiene instruction in connection with medical schools and universities.

2. The higher hygiene instruction for diplomas or certificates of hygiene, such as is required by health officers, sanitary engineers, and other sanitary experts as chemists and bacteriologists.

3. Hygiene instruction to school teachers and school children.

4. Hygiene instruction in veterinary schools.

5. Hygiene instruction to the sanitary inspectors, inspectors of food, etc.

6. Regulations of hygiene instruction by boards of regents.

The reports received from different members of the committee have been submitted and are appended hereto, and the following is a summary of the conclusions arrived at, based upon these as well as other correspondence arising from them:

HYGIENE INSTRUCTION IN MEDICAL SCHOOLS AND UNIVERSITIES.

The average medical student requires sufficient compulsory instruction to fit him for such hygienic work as arises directly out of his practice and which will enable him to co-operate intelligently with health boards. This calls for a general theoretical grounding in all branches of hygiene such as might be covered in a course of forty or fifty lectures and demonstrations, together with some elementary laboratory work in hygiene.

1. It would be advisable that some practical training in sanitation such as disinfection, house inspection, etc., be given either as an optional or compulsory course. 2. A more advanced optional course should be given, optional to students who wish to study the subject more deeply. 3. A diploma course, open to post-graduates, should be available for those who wish to become specialists in hygiene (see also under Section 2 of this report). 4. In addition to the above, a short series of elementary lectures on personal hygiene, forming a

course in sanitary science, and methods of preventing, such as come within the scope of the public, should be available for all students in the various faculties. 5. In connection with the course in sanitary engineering, architecture, etc., adequate arrangement should be made for instruction in hygiene and proper examination of the candidate.

DIPLOMAS IN PUBLIC HEALTH FOR MEDICAL HEALTH OFFICERS.

These require a special course and special examination. On the whole your committee considers that the English diploma of public health, is one suitable for adoption as a standard. This requires six months' scientific training in hygiene, including laboratory instruction in sanitary chemistry, bacteriology and the study of preventable diseases, and six months' practical training as assistant in the regular duties of medical health officer. Of these two courses only three months may be concurrent, and the candidate cannot pass any part of the examination until one full year has elapsed since his admission to the practice of medicine. The instruction in addition to the ordinary sanitary matters must comprise sanitary law, sanitary administration, and the study of hospital treatment of infectious diseases.

It will be noticed that the above qualifications are limited to medical men, and especially restricting the minimum requirements of the medical health officer. It is thus advisable that the sanitary bodies should make such arrangements as will enable persons holding these diplomas to have some advantage in securing or holding appointments. Possibly the holding of an appointment could be made conditional upon securing the diploma within a specified time.

It would seem advisable to have a somewhat higher qualification than the D. P. H., which might be obtained by further special study. This would correspond to the D. S. C. in hygiene, or to the doctor degree in hygiene, as given by the Scotch and some of the English universities. A course of this kind would call for at least two years post-graduate study, with special proficiency in some branch of labatory work in addition to the general knowledge acquired by the D. P. H. and, at least for the doctor's degree, the presentation of a thesis representing original research.

Those parts of the course dealing with practical sanitation are, of course, not called for in the case of those holding appointments as medical health officers. Arrangements of the course should be such as to enable persons engaged in sanitary work to take them piecemeal who are unable to devote their entire time to the work.

It would seem advisable that similar provision be made for diplomas in veterinary hygiene and sanitary engineering, as far as possible, and the work of the laboratory experts in chemistry and bacteriology. In

the case of these special experts, however, it would seem more appropriate to require that the standard be particularly higher in the special branch in question than that the standard should be raised diffusely in all the branches of hygiene. The D. P. H. standard would seem to form a good groundwork from which specialization would be carried on in any branch.

A sanitary expert engineer should have same general knowledge of sanitary chemistry and bacteriology that would be required of the medical health officer, but would not require special instruction in preventive medicine and infectious disease. The sanitary chemist or bacteriologist would require at least two years' special training in his line of work with a certificate of a year's service as assistant in the special line of work and the passing of a special examination.

The examinations for the D. P. H., as specified, must cover at least four days and be written and practical, including both laboratory and outdoor sanitary work. The requirements of a sanitary analyst would be those in the main which are now required by the food and drug experts; it would be necessary that a knowledge of bacteriology as well as chemistry be required in each case. In several states these qualifications already exist. The highest standard is that of Austria and France, where four years' special chemical training as students, and one year as assistant, is demanded before a certificate can be given. Part of this course is included in the ordinary college curriculum.

SCHOOL TEACHERS AND SCHOOL CHILDREN.

The qualifications established by the English school board for the teachers by which a certificate is conferred as a result of attending a course of about thirty or forty lectures on physiology and hygiene, especially adapted to their requirements, is one which seems fairly satisfactory. Your committee considers that such a course should either be made compulsory for all teachers or that special certificates be furnished those who take it. We cannot but strongly condemn the present prejudice which has led to the exaggerated exaltation of the study of the effects of alcohol and narcotics in this course, as displacing subjects which are of more importance. We consider that this topic in a forty lecture course could be dealt with amply in one or at most two of the lectures, and would strongly urge that the course in hygiene for school teachers should be directed rather to disseminating the knowledge of the laws of health than to the spread of temperance propaganda and statistics which can better be dealt with independently. A teacher who has obtained a certificate in the above mentioned course might be considered as competent to give elemen-

tary instruction in hygiene to school children where no better arrangement can be made, and we think that hygiene and elementary physiology should be made part of the knowledge of every child. It is also recommended that a more advanced course in hygiene should be given to all students in universities and colleges, to include not only personal hygiene, but such instruction in Municipal, State and National Sanitation as would give them an intelligent and sympathetic interest in all measures relating to public health.

We would emphasize the view that the main object of instruction to teachers is to help them in carrying out intelligently such measures as may be necessary for the pupils under their charge, but that the supervision of the health of schools is much better entrusted to the school physician or medical school inspector who should be attached to all large schools. The qualifications for a medical school officer has not so far been formulated, but a special knowledge of school hygiene and the diagnosis of infectious diseases should be made one of the conditions for this position where the holder does not possess some other sanitary certificate covering the ground. We think it would be preferable, if feasible, that persons occupying positions of this kind have a D. P. H. certificate. The medical officer should be specially trained in making examinations as to the sight and physical condition of the pupils and regulate such gymnastic exercises appropriate to their case.

THE REQUIREMENTS FOR VETERINARY SCHOOLS.

These should be analogous to those of medical schools, and in our opinion a diploma course in veterinary hygiene should be adopted. The special requirements indicated in Dr. Ravenel's report appear to be sound. The matter of regulation of veterinary instruction is one which is closely allied to that of the hygiene of domestic animals and should be dealt with thoroughly in agricultural colleges.

EXAMINATIONS AND CERTIFICATES FOR SANITARY INSPECTORS AND INSPECTORS OF FOOD, ETC.

For this the requirements of the British Sanitary Institute appear to be specially well adapted and are quite feasible. These require a sufficient elementary knowledge of reading, writing, arithmetic, as well as general education, in order to allow the candidate to go up for the examinations. The course generally given covers from twenty to forty lectures with practical demonstrations, and the examinations are of a thoroughly practical character. The system followed in England of separating the inspector of nuisances from food inspectors is not called for on sanitary grounds, but is owing to the fact that these

officials work under different departments and the requirements of each must be satisfied.

REGULATIONS OF HYGIENE BY BOARDS OF REGENTS.

Your committee would strongly urge that the health boards as far as possible insist upon only such men receiving the title of sanitary inspector as have earned it by passing the examination, to give the necessary encouragement to induce people to obtain these qualifications and by classing those who have not taken them as unskilled, making a distinction in their official titles.

Your committee would also suggest that such portions of this report as may be adopted by the Association should be submitted to those interested for their criticism and the effort made to secure their co-operation; and if a distinction is made between the minimum amount to be made compulsory everywhere and the normal standard which might be optional in some cases, we think that the matter can be practically tested without very serious delay.

We have appended to this report a short schedule showing the extent of the requirements in each of the above branches, as well as memoranda received from individual members of the committee.

(Signed) W. T. SEDGWICK, Chairman.
A. C. ABBOTT.
C. O. PROBST.
M. P. RAVENEL.
A. W. SUITER.
SEVERANCE BURRAGE.
WYATT JOHNSTON, Secretary.