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# KINGSTON MEDICAL QUARTERLY.

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The KINGSTON MEDICAL QUARTERLY is presented to the Medical profession with the compliments of the editorial staff. Contributions will be gladly received from members of the profession. JOHN HERALD, Editor.

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## · THE KINGSTON GENERAL HOSPITAL.

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ON December the 24th, 1897, the Watkins wing of the Kingston General Hospital was destroyed by fire. As this wing contained the private wards for general medical and surgical cases the loss was a double one. The building was destroyed and would have to be restored. The private wards were for a time lost, and the revenue heretofore obtained from them would not appear in the receipts of the institution. The Board of Governors took prompt action. A meeting was held on Christmas day, and it was unanimously decided that the wing must be restored and improved. It was felt that now was an opportune time to introduce into the Watkins wing modern conveniences and necessities which the old building had not contained. To make the Kingston General Hospital modern and fully equipped in all its departments it was felt that several additions must be made. The Watkins wing must be restored and enlarged, the rooms being decreased in size and increased in number. It was further decided that as it has now been clearly demonstrated that Tuberculosis is an infectious disease provision must be made for the isolation of patients suffering from this insidious and fatal enemy of mankind. It was, therefore, agreed to provide two rooms in the upper story of the main building for such patients—one for males and one for females. One improvement necessitated another. As the new Watkins wing would be a three-storied building, and as the fourth floor of the main building would in future be occupied by patients suffering from tuberculosis, it was

deemed absolutely necessary to include an elevator in the requirements of the new building. Those improvements having been unanimously agreed upon by the Governors as essentials of an up-to-date hospital, it was decided to call upon the public for the funds which would be required to complete the hospital according to these proposals. A subscription list was then opened. The list was headed with a statement of what the subscriptions were required for, viz., the restoration and improvement of the Watkins wing, the fitting up of the wards for tuberculous patients, and the building of an elevator. For these objects subscriptions were asked for and subscribed by friends of the hospital, and these objects the Governors feel they must accomplish so as to keep faith with those who have contributed. The first of these objects—the restoration and improvement of the Watkins wing—is all but accomplished. The other two objects—the fitting up of two wards for tuberculous patients and the erection of an elevator—have yet to be undertaken, but as the Governors are pledged to accomplish these, they will no doubt soon be under way.

As the new Watkins wing will be ready for occupation by the end of the present month we have thought that a short description of it would not be uninteresting to our readers. The building is of stone, and in its general outline and appearance has been made to conform with the main building. The basement contains the following rooms and appliances:—A corridor, nine feet wide, running from east to west, and into which all the other rooms open, except the furnace room; four rooms for servants, a pantry and dumb waiter; a pathological laboratory, which is being fitted up by Dr. W. T. Connell; a strong room for such patients as may require restraint; a bath room and a water closet. The furnace room, which is also in this flat, is completely walled off from the corridor and the other rooms. For sanitary purposes this floor is fitted up with a foul air chamber and shaft and an electric fan. By this means foul air will be carried off from the various floors and a current of fresh air continuously supplied. Connection is made with the Doran building from this floor by means of an underground passage. This will be a great convenience, saving labour and enabling the attendants,

even in the severest of weather, to provide the inmates of the Doran building with warm meals, all the cooking being done in the main building.

The first and second floors of the new wing are alike. A corridor nine feet wide runs from east to west, and on each side are arranged private wards. Each of these rooms is about 12x16 feet, and opens out on to the corridor. At the eastern end of the corridor is a pantry, a nurse's room, a dumb waiter and a medicine cupboard. Each floor is provided with a bath room and water closet.

The third floor, like the first and second, has a nine-foot corridor. On the northern side are three semi-private wards, capable of accommodating four patients each. These wards are intended for those who are not in a position to pay for a private ward and yet do not wish to go into the public wards. Patients occupying these wards may be attended by their own physicians, just as if they were in one of the private wards. On the southern side of the corridor are three rooms for children, two dormitories and a recreation room between—one dormitory for boys and one for girls. There are no means of communication between the dormitories and the recreation room. All open out on to the corridor, and the walls are deadened. The recreation room also opens out on to a balcony, which in winter will be enclosed with glass and in summer will be open. Thus those children who are convalescent will have the advantage of a play-room—fresh air in summer and sunlight in winter. This flat has the same conveniences as the other two—bath room and water closet, pantry and dumb waiter, nurse's room and medicine cupboard.

Communication with the main building is provided for on each flat with a door at the western end of each corridor, which is fireproof. A stairway at the western end of the building provides a ready means of access to each floor, independently of the stairway in the main building. Thus in case of fire the Watkins wing could be completely shut off from the rest of the building, and means of ingress and egress are provided. The whole building is to be finished in natural woods, which will give a bright, fresh appearance, and enable the attendants to keep it always clean and cheery. The building is to be heated by hot water.

The Board of Governors feel that in this building they have a model modern hospital, and we agree with them. When the Governors complete the other improvements which they have decided upon, viz., an elevator and isolated wards for tuberculous patients, they will have a hospital which will meet all the requirements of medicine, surgery, gynecology and sanitation. In order to carry out these laudable and necessary improvements the Governors require more money. They themselves have contributed largely of their own private funds. They ask the public to share in the good work. A hospital such as the Kingston General appeals to all classes. We trust that there will be a generous response. The hospital is not local in its operations. It is open to the sick and the afflicted. Let those who now enjoy the blessings of health assist in the noble work.

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#### ULCER AND CANCER OF STOMACH.

**I**N the last number of the QUARTERLY I drew attention to some of the diagnostic features of chronic gastric affections and recited the histories of a few cases which had come under my care. Since then I have had charge of a case which I deem of special interest, both on account of the complications which existed and of the bearing which it has on the diagnosis between ulcer and cancer of the stomach. Miss S., aged 44, came under my care on March 2nd last, with the usual history of indigestion for several years—pain on eating—vomiting at times of “coffee ground matter”—emaciation, 45 lbs. in six months—constipation. Her appearance was that of a cancerous patient. The age of the patient and the duration of the illness might suggest cancer, ulcer or inflammation. The pain would not throw much light upon the nature of the disease, and the emaciation might be looked for in any of these conditions. The “coffee-ground” vomit was highly suggestive of cancer. Judging from the information obtained from the patient and from her general appearance cancer would in all probability be diagnosed. On physical

examination the stomach was found to be smaller than normal, and thus we would feel inclined to exclude a simple chronic gastritis. On palpation a distinct nodule was felt over the pyloric end of the stomach. This would naturally lead one to think of cancer, though, of course, it might be due to the cicatrization of an old ulcer. Everything so far elicited from the patient and obtained by physical examination pointed to cancer of the stomach. An examination of the stomach contents was made and free hydrochloric acid was found in excess. Now, in cancer of the stomach free hydrochloric acid may be found, but never in excess so far as I am aware—generally it is diminished in amount or absent entirely. In ulcer of the stomach, on the other hand, free hydrochloric is always found in excess. Here, then, was a case whose clinical history pointed to cancer, and that diagnosis was apparently confirmed by physical examination. On the other hand, the examination of the stomach contents as clearly pointed to ulcer, to the exclusion of cancer. A diagnosis of ulcer of the stomach was made, complicated with cancer, of that portion of the pancreas lying behind the pylorus. This diagnosis would account for the stomach not being enlarged; for the chemical nature of the stomach contents; for the nodule clearly demonstrable at the pyloric end of the stomach, and even for the character of the vomit, though we would have expected blood in larger quantities; and for the general emaciation and cachexia.

The patient was at once put upon rectal alimentation and morphia to relieve the pain. Her temperature never went above  $101^{\circ}$ , and for two days before death was sub-normal. She died on March 13th.

Dr. W. T. Connell made a post-mortem examination, which I append:

Miss S. died March 13th. Post-mortem on 14th, eighteen hours after death. Body that of a fairly nourished woman (she had lost, however, 45 lbs. in past six months).

No points of import to external examination.

*Head* not examined. No permission.

*Thorax*.—Slight old adhesions at apex right lung; left lung adherent over entire surface; old pleuritis; lungs themselves present no pathological features.

*Trachea.*—Bronchi and bronchial glands normal. Pericardium *nil*. Heart weighs 8 ounces. Muscle fibre well nourished. Valves and chambers normal.

*Aorta* shows a few patches of fatty atheroma.

*Abdomen* markedly distended and drumlike. On opening the peritoneal cavity there was an escape of large amount of gas, with a peculiar but not foul odor. The intestines were almost empty and their serous coats inflamed. The great omentum was curled up and was bathed in a thin sero-pus, as were also the intestinal coils. The lumbar ileac and pelvic fossae held together about 30 ounces of a sero-purulent fluid, showing occasional flakes of fibrin. On the anterior aspect of the stomach, about an inch to the left of the midline of the body, and about  $2\frac{1}{2}$  inches from the pyloric orifice, was a perforation of the stomach wall. Through this perforation, which was circular and averaged half an inch in diameter, the mucous membrane of the stomach protruded slightly. A culture from the fluid in the peritoneal cavity gave a growth of large numbers of colonies of three forms of Bacteria: (a) Staphylococcus Pyogenes Aureus; (b) a Bacillus—putrefactive in nature; (c) Sarcinae—numerous colonies of a white sarcinae, likewise putrefactive in nature.

*Stomach* was small, contracted and empty, measuring  $1\frac{1}{2}$  inches from above down at ulcer line, and being nowhere larger in vertical diameter. Stomach measured  $6\frac{1}{2}$  inches horizontally in largest axis. The mucous membrane was covered with a dense layer of tenacious mucus—the walls were thick, averaging two-fifths of an inch. The ulcer above mentioned was occluded by the mucus membrane, but when this was withdrawn ulcer gave a markedly punched out appearance on serous and muscular coats, but was slightly larger on mucous surface. No other ulcers or scars of ulcers were found in stomach wall.

At the pyloric end of stomach, involving the orifice and a portion of the commencing duodenum, was a nodule involving more particularly the outer coats and measuring about three-fifths of an inch in thickness, one inch transversely, and  $1\frac{1}{4}$  inches in line of lumen. It was situated in lower and posterior part of the orifice; had not involved the mucous membrane and shaded into the underlying pancreas, to which it was firmly attached. Its margins were ill defined, shading gradually into the stomach and duodenal walls. This nodule was very hard, and on microscopic examination proved to be schirrhous carcinoma, in which there was a marked preponderance of

fibrous stroma (Atrophic Scirrhus.) The pyloric orifice was not constricted.

*Pancreas.*—That portion of pancreas lying below pylorus was adherent to the nodule above described at that orifice, and was very firm. Microscopic sections of this portion showed that there had been no invasion of the pancreatic structure by the cancer growth, but that there was a marked increase in the interlobular connective tissue, and that many of the lobules were the seat of proliferative inflammation, the epithelial elements undergoing degeneration and being replaced by connective tissue elements. Elsewhere the pancreas was normal, except at its tail. Here there was a nodule of growth, averaging  $1\frac{1}{2}$  inches in all its diameters and involving the tail of pancreas and hilus of spleen infiltrating both organs. This growth was very soft, almost diffuent. Microscopically it proved to be soft sphaeroidal carcinoma.

*Spleen*, other than commencing invasion of the nodule above mentioned, presents no pathological features. Cultures taken from splenic pulp remain sterile.

*Intestines.*—*Small intestine* quite empty. Mucous membrane healthy. Serous coats inflamed as before described. Appendix normal. Great bowel contained a few scybalous masses. The rectum and sigmoid flexure contained some milk (fed per rectum), otherwise *nil*.

*Liver*, except left lobe, normal. The left lobe was quite completely transformed (within the liver capsule) into a mass of new growth, with all the gross characters of carcinoma. The central part of the lobe (growth) was soft, liquefied—the seat of fatty degeneration of the growth and haemorrhagic extravasation. Microscopically the growth was a typical sphaeroidal carcinoma. Here and there through the growth there was marked proliferation and increase in the bile ducts.

*Kidneys.*—Right  $4\frac{1}{2}$  ounces; left 5 ounces. The cortex was slightly pale (fatty); otherwise normal. Cultures were sterile.

*Bladder* empty. Mucous membrane healthy.

*The gall bladder*, partially filled with bile, was adherent to duodenum. The bile ducts patent.

*The Uterus.*—Virgin. Seven small fibro-myomas, the largest an inch in diameter, were scattered through the uterine walls. Tubes and ovaries *nil*.



*Summary.*—Gastritis. Gastric Ulcer. Perforation and Perforative Peritonitis. Sphaeroidal Carcinoma of Pylorus, the left lobe of Liver, the tail of Pancreas and Hilus of Spleen, the Pylorus being the primary seat. Fibrosis Head of Pancreas. Old Pleuritis. Small Fibro-myomas of Uterus.

*Death.*—Perforative Peritonitis.

From the above report it will be seen that my diagnosis was partly right and partly wrong. There was ulceration of the stomach, the perforation of which set up a peritonitis, which was the immediate cause of death. There was not cancer of the pancreas, but a fibroid threatening gave rise to the nodule, which was felt over the region of the pylorus. There was cancer of the stomach at the pyloric end, but that growth did not involve the mucous membrane of the stomach. This to my mind explains why the secretion of free hydrochloric acid was not diminished as it usually is in cancerous growths of the stomach.

I have related the history of this case and given the post-mortem examination in detail, because I believe it may be as instructive to others as it was to myself. The clinical history and the physical examination pointed to cancer. The examination of the stomach contents as clearly indicated ulcer. The post-mortem examination revealed both and accounted for the apparent discrepancy in the stomach contents with that which we expect to find in cancer of the stomach.

JOHN HERALD.

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#### PERSONAL.

WE wish to congratulate Dr. W. J. Gibson, of Belleville, on his election to the Presidency of the Ontario Medical Association. This is an honor of which any practitioner may well feel proud. While we most heartily and sincerely congratulate the Doctor, we cannot refrain from saying that the Association is also to be congratulated on having Dr. Gibson as its President. Dr. Gibson has had a most successful career. He received the degree of B.A. from Queen's University in 1874, and in 1882 he received from the same institution his M.A. In 1881 he received

his M.D. also from Queen's. He at once entered upon the practice of the profession he had chosen and at which he has made a most decided success. Dr. Gibson for years has enjoyed one of the best practices in Belleville, and his reputation is not confined within the city limits. His opinion and advice is often sought, not only by patients, but by fellow-practitioners in the surrounding towns and villages. Amid the arduous duties of an extensive practice Dr. Gibson has also found time to keep himself posted in all that is new in modern medicine and surgery. He has been a contributor to our medical publications, and on several occasions has contributed valuable papers at the meetings of our medical associations. At all times he has maintained his interest in his Alma Mater. For years he has been elected by the graduates as a member of the University Council. For the past five years he has been a member of the committee which recommends appointments to the Medical Faculty of Queen's. Again we congratulate the Doctor, and again we say the Ontario Medical Association has in Dr. Gibson a worthy representative of the profession as its president.

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#### THE KINGSTON MEDICAL AND SURGICAL SOCIETY.

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A MEETING of this Society was held in the General Hospital on Monday, June 13th. The President, Dr. Oliver, occupied the chair. After routine business Dr. C. K. Clarke exhibited a specimen of aneurysm of the aorta (thoracic portion) from a patient who died from collapse symptoms within twelve hours after the onset. As this case is described fully by Dr. Webster, we refer our readers to his article in the present number of the QUARTERLY.

Dr. Third then read a paper upon ether as a general anæsthetic. He compared it with chloroform in its mode of action and as to the dangers of its administration. He agreed with the now generally accepted opinion, that ether is the safer anæsthetic of the two. The most interesting part of the paper was the method of administration and the means to be employed in cases where dangerous symptoms manifested themselves.

Dr. J. C. Connell next read a very practical paper on Local Anæsthetics. Among others he mentioned ice and salt, alcohol and cold, carbolic acid, chloral hydrate, ether, ethyl chloride and cocaine. With regard to cocaine he advocated the use of weak solutions—2 per ct. for the eye and 4 per ct. for the throat.

Discussion followed the reading of these papers, in which most of the members present took part.

The annual meeting was then held, and the following officers elected for the ensuing year :—

*President*—Dr. Herald.

*Vice-President*—Dr. Forster.

*Secretary-Treasurer*—Dr. W. T. Connell.

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## BOVINE TUBERCULOSIS—ITS IMPORTANCE IN THE CAUSATION OF TUBERCULOSIS IN MAN.

**T**UBERCULOSIS in animals, and more particularly in cattle, is, without doubt, a widespread disease, but, fortunately, it is not yet so common or widespread in Canada as it is in the older inhabited countries, such as England, Denmark and Sweden. Tuberculosis in animals is due to the same causal agent as is the disease in man, viz., the Tubercle Bacillus. Further, the conditions originating susceptibility or predisposition to the disease attacking cattle are in the main those that are active in inducing susceptibility in man. The disease is transmissible, not only from animals to man, but from man to animals, so that we find that while the milk and meat from infected animals may excite intestinal tuberculosis in susceptible persons, we also find that consumptives can infect their cattle by expectorating on their feed or by cattle inhaling their dried sputum as "dust." Tuberculosis in cattle is as a rule a very chronic disease, lasting months and years; only rarely do we note cases of acute type lasting weeks. Like tuberculosis in man, we may find any part attacked—lungs, intestines, bones or joints, lymph glands or meninges, but the lesions are naturally confined in the main just as they

are in man, to seats of entry, viz., the respiratory and digestive tracts, and the tissues or viscera most intimately connected with them by lymph or blood current. In animals we find the main lesions then in the lungs and in the intestines and their peritoneal covering and lymph glands. In animals, when the infection is by the digestive tract, the liver is almost always attacked. Now in man this viscus not infrequently escapes, even when we find marked intestinal lesions, but in animals it seems peculiarly liable to infection, as is evidenced by its early involvement in experimental tuberculosis of guinea pigs and the constancy with which this organ is involved when we have lesions in any part of the intestinal tract or peritoneum in cattle. Rarely, too, will we find the lungs of cattle markedly involved before we have tubercles appearing in the liver. Now, infection of the liver must mean in almost all cases a blood infection, *i.e.*, the circulation of the tubercle bacillus in the blood (generally, not necessarily portal blood alone).

A cow may show no external appearances of tuberculosis, and yet present when killed easily appreciable lesions. This fact was very well shown in the cattle which were slaughtered (after the application of the tuberculin test) at Rockwood asylum in May of this year. Here some of the choicest animals in full milk showed numerous lesions in the intestines, mesentery, liver and in some instances in the lungs. Speaking to a butcher, an observant man, who was present at this slaughtering, he stated that one-fifth at least of the large number of fatted cattle that had passed through his hands showed the lesions of tubercle. We see from this the chronicity of the process and the slight effect the disease during its early stages seems to have upon the general health of the animal. Yet we have here in many cases the circulation of tubercle bacilli (few in number) in the blood, and we can readily see that in a milch cow some of these bacilli may be thrown out in the secretion of milk and tubercle bacilli be found in the milk of cows, even though they be not suffering from any local tubercular disease of the udder. This fact is now accepted by all those who have studied the question carefully, and is recognized by the health authorities of nearly all the larger American cities and upon the continent. Of course when there

is local disease in the udder there is much greater danger of the presence of tubercle bacilli in the milk. The British Royal Commission on Tuberculosis, which reported in 1895, insisted upon the infectivity of milk from tubercular cows, whether presenting local disease of the udder or not. This report has been further confirmed by the recently issued report of the second Royal Commission. This report deals more particularly, however, with the means to be advised to check the spread and limit the disease. The meat of tubercular animals may also be infective, particularly when the disease is generalized; but as it is but rarely such meat would be exposed for sale, there would be but little danger from this source. When the disease is not generalized the risk is minimized, and is reduced to zero if the meat be well cooked, as it usually will be, before eating. We see, then, that the main danger in tubercular cattle lies in the milk. Consequently, the seat of attack in man from the use of such will be the digestive tract.

Now tuberculosis of the intestinal tract (including the mesenteric glands and peritoneum) in man, we find in persons over the age of puberty, and even over ten years, to be commonly secondary to tuberculosis of the air passages. Under this age we find, however, a large number of cases of primary tuberculosis of the tract, more particularly during the first few years of life. We class these cases of primary abdominal tuberculosis under the generic term "Tabes Mesenterica." In these cases of primary abdominal tuberculosis the causal agent must make its entry (with few exceptions) *via* the digestive tract in the food. Now, while it is true that the tubercle bacillus can and does exist in the dried state outside the body for long periods (three to twelve months) and might accidentally contaminate food falling upon it as "dust," yet this means of infection can hardly account for the large numbers of cases which we more particularly see in the cities of "Tabes Mesenteria." I think, then, that we must come to the conclusion that milk from tuberculous cattle is at fault. Children and invalids are those who consume most milk in the raw state, and they are the ones, too, most susceptible to infection. Not only do we find, however, in children these lesions of "Tabes Mesenteria," but there can be no doubt at all

that in many cases where we have joint, bone or meningeal lesions, that in such cases the primary seat of entry has been through the intestinal canal; the local lesions have been quite insignificant, more usually an involvement of one or more mesenteric glands, and from these general blood infection has occurred, either via lymph or direct involvement of veins. As children get older, infection via the respiratory tract and bronchial glands becomes more and more a factor till in adults involvement of bones, meninges, or more particularly, genito-urinary apparatus, is nearly always an infection from such primary source.

To Bovine Tuberculosis, then, is not only to be ascribed nearly all the cases of "Tabes Mesenterica," but most of the tubercular lesions of bones, joints and meninges of young children.

It becomes, then, a matter of some moment to effectually deal with this source of infection. Our Registrar General's reports show for 1897 an increase in the number of deaths from tuberculosis, and this is emphasized by our Provincial Board of Health report for the same year. While the greater increase has been in "consumption," and while I recognize that contact with consumptives and aerial infection are the chief factors in contributing to this increase, yet we must also ascribe some share to animal infection, and must open our eyes to the fact that so long as the disease is allowed to ravage unchecked among cattle then so long will that source of infection be open to attack our infants and children.

The Dominion Government under the "Animal Contagious Diseases Act" has made Bovine Tuberculosis a notifiable disease under penalty, and has appointed district veterinary inspectors to investigate cases when called upon to do so, but there is no efficient machinery for the actual enforcement of the act, and it is practically a dead letter law.

Our Provincial Government, too, in 1896 passed an act providing for the inspection of meat and milk supplies to cities and towns, but its operation was at once negated by an Order-in-Council suspending the operation of the act, and it is not yet in force. Owing to our strictly party government the measure is likely, too, to lie in oblivion for some time to come.

These, then, are the actions which our governments have taken, but it is time now for the medical men to attempt to educate the people and make the enforcement of the acts necessary. There can be no doubt at all that it is in the interests of the public health that there should be a careful inspection, more particularly of the milk supplies of our cities and town. By the application of the tuberculin test in the hands of any careful veterinarian tuberculosis can be readily detected in a herd and proper measures be taken to check further spread of the infection. Given freedom from the tubercle bacillus in our milk supplies, the young of our population will escape one of the dangers to which they are exposed. By this means we shut off a minor yet important factor in our case and mortality rate from tuberculosis, and it is high time some measures were taken to this end.

W. T. CONNELL.

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## SOME LABORATORY NOTES.

### WIDAL'S REACTION IN TYPHOID FEVER.

**I**N those cases of typhoid fever which are atypical, especially in the early stages, we have a comparatively simple yet reliable diagnosis test in Widal's reaction. The following is a brief outline of the procedure :

With a sterile needle, or skin lancet, a drop of blood is taken from the tip of the patient's finger, rendered sterile by washing with one to twenty carbolic solution, followed by sterile water. The drop of blood is preserved between two sterile watch glasses, with their concave surfaces opposed and held together by two small straps of adhesive.

Various dilutions of this drop of serum in the watch glass are made by the addition of loopfuls of sterile water.

A drop of a twenty-four to forty-eight hour broth culture *Bacillus Typhosus* (previously examined to ascertain motility of

organisms) is placed on a sterile cover film by means of a sterile loop platinum needle.

From the diluted serum a small loopful is added to the drop of broth culture on the cover film.

The cover film is inverted over a hollow glass slide, and examined under an oil immersion lens.

The reaction produced in a true case of typhoid fever is, firstly, a loss of motility of the Typhoid Bacilli, and secondly, their aggregation into small clumps containing on the average between ten and twenty bacilli.

Cases tested in the medical wards of the Kingston General Hospital, September, 1897.

Case I.—R. B., æt. 18. First week after onset—24 hour broth culture *Bacillus Typhosus* used; serum dilution 1 to 9; gave loss of motility and positive clumping of typhoid organisms after five minutes. Subsequent dilutions of 1 to 18 and 1 to 36 gave reaction in seven and ten minutes respectively.

Case II.—B. J., æt 31. Second week after onset—28 hour broth culture used; serum dilution 1 to 9; clumping and loss of motility after four minutes; serum dilutions 1 to 18 and 1 to 36; gave reaction in seven and nine minutes respectively.

Case III.—Mrs. V., æt 49. Third week after onset—Serum dilutions 1 to 9, 1 to 18, and 1 to 36. Reaction after ten, eighteen and thirty minutes respectively.

The above cases ran the typical course of typhoid fever, with the exception of the last, which was complicated with pneumonia.

Control tests were usually made with my own blood, and in no experiment was there the slightest reaction.

We have now seen that in cases I and II, when the fever was ascending towards the acme, the characteristic reaction appeared four to five minutes, and in case III, when fever was defervescing, fully ten minutes elapsed before appearance of reaction.

These are typical of many other tests which have led up to the conclusions:

1. That the occurrence of Widal's reaction is generally pathognomonic of typhoid fever.

2. That the TIME of the appearance of the reaction, taking



into account the foregoing cases at early and advanced stages, and also the different grades of serum dilution, is a fair index of

- (a) the virulence,
- (b) the stage of the attack.

#### AGE ANOMALY IN TWO MALIGNANT TUMORS.

Last April two local surgeons each submitted for pathological examination a tumor of the breast. One was removed from a woman of fifty. The second from a young woman of twenty-five. From clinical observation, and also in accordance with the theory of cell activity and decline, the pathologist would expect in the case of the elder a carcinomatous and in the younger woman a simple or sarcomatous growth. In these cases, however, gross and microscopic examination revealed the very reverse.

Case I, æt. fifty.—Clinically : A round nodule, about three-quarters inch in diameter, was situated in the axillary line low down between the sixth and seventh ribs, some distance from the region of the axillary glands. A second growth was situated in the mammæ—round, flattened quite hard, and about two and one-half inches in diameter—axillary glands not affected.

Macroscopically : The nodule below the axillary region was round, smooth, capsulated, and upon gross section with knife exhibited a white, smooth, glistening, comparatively hard surface devoid of cupping.

Mammary growth : round, much larger, smooth, distinctly encapsuled, and on section resembled precisely the axillary nodule.

Microscopically : Nodule below axilla ; sarcomatous ; chiefly spindle-celled, with a few small round cells intermingled with a large number of white fibrous bands running in various directions and giving the tumor its firm consistence.

Diagnosis—Fibro-sarcoma ; spindle-celled.

Mammary tumor : Showed numbers of gland alveoli (adenomatous) lying in a stroma composed for the greater part of spindle-sarcoma cells and white fibrous bands.

Diagnosis—Sarco-adenoma secondary to nodule below axilla.

Case II.—æet. twenty-five. Clinically: A well-marked induration, about one and one-half inches in extent, underneath nipple. Mammary gland atrophied axillary glands not yet affected.

Macroscopically: Growth not limited by capsule but had diffusely infiltrated the gland tissue. Gross section—a whitish milky surface devoid of cupping and not very firm.

Microscopically: Groups of spheroidal cells arranged in round or oval alveoli partitioned from each other by a very small amount of fibrous tissue.

Diagnosis—Encephaloid carcinoma.

G. W. MYLKS.

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### OEDEMA OF THE EYELIDS.

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THIS symptom which is so frequent and conspicuous, very often causes more alarm, both to the patient and to the less experienced physician, than does the lesion which lies at the bottom of it. It may be of interest, therefore, to the general practitioner to enumerate the conditions which give rise to oedema of the eyelids and to arrange them in such a way as will facilitate accurate diagnosis.

In the first place the oedema may be either (1) *non-inflammatory* (oedema frigidum) or (2) *inflammatory* (oedema calidum.)

Non-inflammatory oedema is the result of a simple venous congestion, and is associated with general oedema, though it may be the first part of the body affected. In the latter case it may suddenly appear and disappear (oedema fugax). It is in *nephritis*, *heart-disease*, and *hydraemia* that this occurs.

Inflammatory oedema is found in connection with every violent inflammation of the lids themselves or of the neighboring structures. It is essential to open the lids sufficiently to inspect the conjunctiva and eyeball, and this must be done notwithstanding the swelling and spasm. Desmarre's lid elevators are useful for this purpose, as they minimize the pain and danger

from forcible separation of the eyelids. When the conjunctiva is found to be of a normal color or but slightly injected, and the eyeball itself is normal and freely movable we have to do with a superficial affection: on the other hand we refer the oedema to a deep affection when the conjunctiva or eyeball is abnormal.

The following are the superficial affections which give rise to oedema.

1. Traumatism—This is ordinarily accompanied by suffusion of the lid, and the discoloration apart from the history will determine the diagnosis. The injury may be from the bite or sting of an insect.

2. Dacryocystitis—In palpating the swollen lid it is found that an indurated and sensitive spot fills the internal angle, and pressure over this usually evacuates pus from the puncta, or epiphora has preceded the attack.

3. Hordeolum or styè—Palpation shows a spot of induration and of special painfulness near the free border of the lid. In a few days at the most there develops a point of yellowish discoloration between the cilia or on the inner surface of the lid.

4. Furuncle and malignant pustule.—This produces a nodule of considerable extent, circumscribed, painful and indurated. Abscesses of the eyelid lies deeper in the tissues.

5. Erysipelas—In this condition the oedema is uniform and extends to the neighboring parts. The skin feels thick and hard.

Oedema of the eyelids is also a symptom of certain deep affections.

1. Conjunctivitis.—In purulent, diphtheritic and lymphatic conjunctivitis the diagnosis is easily made from the appearance of the conjunctiva and the nature of the secretion.

2. Panophthalmitis, severe irido-cyclitis, hypopyon-keratitis and acute glaucoma are the conditions of the eyeball which produce oedema. Chemosis of the conjunctiva is likely to be present at the same time.

3. Orbital cellulitis and tenonitis.—With these there is more or less protrusion and immobility of the eyeball, in addition to the oedema and chemosis.

4. Thrombosis of the cavernous sinus produces oedema of the lids. There is also oedema behind the ear in the mastoid region and serious cerebral symptoms.

5.—Orbital tumor may produce oedema of the lids, with very slight inflammatory reaction.

J. C. CONNELL.

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### NEUROLYTIC HAEMOPTYSIS.

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ON Nov. 21st, 1897, I was called in consultation with a physician in a neighboring town. The case presented the following history throughout as detailed for me by the attending physician.

Miss S., aged 30. Family history negative: previous health good; bowels regular; urine normal; menstruation as always regular.

Patient first consulted me November 4th, 1896, complaining of a dry cough, anorexia, dyspnoea and pain below left clavicle. Examination of chest with negative results. Temperature normal. Pulse 80°. Gave a sedative cough mixture—deod. opium and pot. cit—with no result. Cough and pain worse when I saw patient on 6th. Temp. 98½. Next visited patient on 12th. Cough hard, dry and ringing. Put her on pot. brom. and spts. chlorof. with no better result.

During this time patient was a somnambulist. Was often followed by servant. Patient would put on a cloak only and walk half a block and return—sometimes on very frosty nights or in worst of rain storms. Sometimes only knew she had been out by presence of mud on feet and clothes, or finding her watch out of doors in the morning. Patient endeavoured to prevent herself from night walking by bolting door and barricading it, tying herself in bed, etc. Would find herself still tied up in the morning, but her clothes and hair wet with rain. When doors were locked and keys removed she would try all doors or use a window to get out by, or if baffled would return to her room.

Has gone down and rung the telephone. The ring of the telephone bell, however, would always waken her. Would talk, sing, whistle and pray while asleep, changing rapidly from one to the other. This I often witnessed myself.

On Nov. 20th put patient on codeia with no result. Cough just the same. Patient now began to cough up mouthfulls of blood. Tried to hide the fact. Found it out only through the servants. Hemorrhages always occurred while alone or at night, or if anyone was in the room just as she was supposed to be waking up. Only one or two mouthfulls at a time.

Nov. 21.—Temp.  $98\frac{1}{2}$ . Pulse 82. Put her on bromide again. Spoke of sending patient away. Cough immediately ameliorated, but pain below left clavicle continued and very severe. Examination still negative. Microscopical examination of sputa by Dr. Connell gave negative result.

Nov. 23rd.—Trained nurses engaged. Night walking ceased in a few nights, but attempted at first. Other neurotic symptoms accentuated. Temperature continued normal. Patient on bromide, opium and ergot. Fly blisters applied, with no result to pain. Patient asked to have them applied. By Dec. 9 spitting of blood began to lessen. Would only happen two or three times a day, instead of six or ten times.

Dec. 12.—Patient sitting up. Only one hemorrhage, and that in the morning. Would happen just as nurse re-entered room.

Dec. 18.—Hemorrhage now every second day, but pain behind over scapula on left side, as well as in front, below clavicle. Patient, after being up for a few days, had return of cough, with increase of pain and more hemorrhages. Returned to bed.

Jan. 6th.—Great distress and pain complained of after eating.

Jan. 22nd.—Obscure and darting pains radiating down left arm and hand. Did not follow any particular nerve.

Feb. 8th.—Patient up a little each day. Hemorrhages had gradually decreased till they stopped. Pain still complained of in left chest and arm. Cough slight.

I think there can be no doubt that the hemorrhage was

pulmonary and of neurotic origin. These cases are of quite rare occurrence. The text books, and I have searched most of them, give little information on this subject. In fact, the only reference of any value that I could find appears in the "International Clinics," April, 1897, by Dr. May, of Philadelphia. Several experiments are cited in the article, wherein irritation of the vagi and sympathetic called forth hemorrhage into the lungs and pleura. I quote from the article:—

"Laying aside all other influences there is nothing that is more closely interwoven with the life of the blood vessel than its supplying nerve. Experiments clearly demonstrate that injury to the nervous system brings about *hemorrhage into lungs and stomach*. Nor is clinical evidence wanting to show the intimate relation between the brain and nervous system and hemorrhagic lesions.

Dr. J. C. Handfield Jones, in treating on the respiratory neurosis in his work on 'Functional Nervous Disturbances,' states that 'if the principles I have laid down be correct, haemoptysis may be included among the neurosis of the respiratory organs. I would not be led to affirm this in an exclusive manner as if there were no other causes of this hemorrhage; the only point I wish to maintain is that in certain cases, and these not very infrequent, haemoptysis may be reasonably regarded as a paralytic neurosis of the vasomotor pulmonary nerves. In a fatal case of tuberculous hæmoptysis I could discover no special source of the profuse gush which destroyed life; it seemed as if the blood had escaped from the vessels everywhere. This is the general experience of others. When we look at a good specimen of infected lung and at a thin slice of pulmonary tissue we cannot but be struck on the one hand with the extreme vascularity of the air cells, and on the other with the absence of support of the capillaries. Trousseau relates the case of a lady who was subject when a child to somnambulism, and since then to the most *bizarre* nerve disorders. When about thirty years old she had such profuse attacks of haemoptysis and so much dyspnoea that phthisis was suspected, though no physical signs could be detected. Till the menopause occurred she often had the most alarming menorrhagia. Andral relates a case of fatal hæmop-

tyses, the lungs of which at the autopsy were found free from tubercle. The girl, aged twenty-one, had suffered from loss of strength and was put out of breath on the least exertion. These are common signs of a neurolytic condition.'"

There is a well marked similarity between the conditions and the cases quoted in this article and the one which came under my own observation. Further on in the article above quoted it states: "In a very interesting article on this subject Dr. L. Fleischman contributes the post-mortem results of two cases of pulmonary and pleural hemorrhages, which co-existed with degeneration of the brain. In an original paper an extensive capillary extravasation of bright red blood corpuscles into the pulmonary tissue of the insane, Dr. Jhen, the author, after referring to Northangel's experiments, which show that pulmonary haemorrhage follow irritation of the brain of rabbits, says that this investigation leads one to anticipate the occurrence of wide-spread infiltration of bright red blood into the alveolar spaces of the insane. As a matter of fact this expectation was verified on the post mortem table."

A further study of this interesting subject would doubtless throw much light on many obscure cases of pulmonary haemoptysis.

E. RYAN.

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### EXCISION OF THE ELBOW JOINT.

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J. H., æt 35, came to me early in May, 1897, seeking relief from an inflamed elbow joint. He carried his left arm flexed at a right angle, and the tissues for some distance above and below the elbow joint were much swollen and of a dusky red color. The patient was suffering considerable pain, and the entire limb was useless except for a limited motion of the fingers. A small sinus existed posteriorly below the joint, exuding a small quantity of watery pus. The patient was an active looking man of medium stature, and by occupation a farmer. He gave a history

of a fall from a waggon about a year before, the main force of the fall being received upon the point of the left elbow. There was considerable soreness at the time, with subsequent sub-acute inflammation of the joint, which was treated at intervals variously by painting with iodine, poultices and blisters, and about two months previously to his arrival in the city an incision had been made posteriorly over the ulna, two inches below the elbow joint, and a small quantity of cheesy pus evacuated.

Careful interrogation of the lungs gave no evidence of any tubercular lesion, and the patient could recall no instance of hereditary taint. On examination of the arm, however, a diagnosis was made of tubercular caries of the bones entering into the formation of the elbow joint, and the patient was informed that the treatment advised was the complete excision of the joint. He demurred from this, and so was persuaded to have a radiograph of the diseased region made. This was done, and the photograph showing very clearly the damaged condition of the bones was sent to the patient and the nature of the operation fully explained to him.

In a few days word was received from him that he was ready to come in to the city at any time and have me "fix his arm."

On May 13th, by means of a single posterior incision, the joint was exposed. The synovial membrane was found in a pulpy condition, one-quarter of an inch in thickness, and the distended sac contained about four ounces of pus.

The overlying tissues were carefully dissected from the bones, special care being taken to guard the ulnar nerve from injury. Erosion of the cartilages covering all three bones was very marked, and the internal condyle of the humerus was extensively destroyed by the carious process. One inch of the humerus was first removed by a fine saw, but this not clearing the diseased tissue fully another half inch was sawn off. The ulna was shortened by  $1\frac{1}{4}$  inches, and three-quarters of an inch of the radius was also removed.

The limb was then placed upon a wire splint flexed at somewhat more than a right angle. A drainage tube was inserted at the most dependent part of the wound. The tube was shortened



from time to time, and passive movement of the elbow, wrist and fingers was begun on the third day. On the tenth day the patient had a chill, followed by a temperature of  $102^{\circ}$  F., and on examining the arm a collection of pus was discovered posteriorly over the lower third of the humerus. This pus was evacuated, and it was found that it had no connection whatever with the site of the operation. The presence of the pus could be accounted for only from the degree of force employed in the digital compression of the arm during the operation—the tissues being weak and flabby from non-use for a considerable period.

No further trouble was experienced; the patient walked about the hospital after the first week, carrying his arm in a sling, and left for home after a fortnight, returning to the city each week for dressing and manipulation of the limb. In six weeks the wound was quite healed, and movements of the arm were entirely free from discomfort and the patient again carried his arm in his coat sleeve, the splint having been removed. Gradually the power of motion was restored to the joint—the patient could feed himself and take off his hat, and in fact made the statement a few months later that his arm was “as good as ever it was,” except that he could not hold it rigid in pitching hay.

It is now more than a year since the operation, and there has been no return of the disease. A very useful joint has been the result, the severed bones being rounded off and lying in close apposition.

W. G. ANGLIN.

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#### ANTITOXIN.

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THE use of antitoxin in the treatment of Diphtheria is now almost universal. It is essential that the article used should be pure and up to the required strength. We notice that Mr. John Mackenzie, Official Bacteriologist of the Ontario Board of Health, has recently made a test of the antitoxin prepared by Parke, Davis & Co. The samples tested were purchased in the open market, and were found to be pure and above the strength stated upon the label—the bottles were marked as containing 1000 units, but in reality contained between 1200 and 1500 units. As a result of this test, and in consequence of personal experience, we believe that the antitoxin prepared by Parke, Davis & Co. is a reliable article.

## SURGICAL NOTES.

### EXCISION OF ELBOW.

THE patient T—, aet 39, had been suffering from what he supposed was rheumatism of the elbow for some months. The condition not improving he consulted Dr. J. A. McBroom, of Gananoque, who diagnosed tuberculosis of the elbow joint, and finding a thorough course of treatment made no impression on the disease, advised operative interference. When seen by me, about the middle of October last, the patient, a well developed man with t: 99½, pulse: 80, stated there had been no severe pain in his arm, but it was entirely useless, for, though fairly free from pain when at rest, any movement caused suffering. The arm presented the fusiform appearance characteristic of a tuberculous elbow, and, an examination by means of the X Rays showed that the bones forming the joint were diseased. Iodoformized glycerine was injected into both joint and bones a number of times, but the fluoroscope showed no improvement, and on November 10th I excised the joint. The incision was the straight posterior of Park four inches long—two above and the same below the line of articulation immediately down to the bones and through the tendon of triceps. As much as possible of the periosteum with the insertion of the triceps was peeled off with an elevator from the olecranon and by the same means the internal condyle was cleared, care being taken to avoid the ulnar nerve. The posterior interosseous was similarly dealt with in denuding the external condyle and head of radius. The forearm was strongly flexed and placed palm downwards on the table on the same side of patient's head and was firmly held there by an assistant. Steadying the end of the humeros with lion-jawed forceps the diseased portion of that bone was removed and then the ends of radius and ulna. The disease was so extensive in the radius that it had to be divided close down to the insertion of the biceps. A large cavity containing the characteristic cheesy material and extending in front of the radius and below the middle of that bone was cleaned

out and with forceps and scissors the wound itself was freed from tubercular material. The incision was then sutured and a lateral Mason splint applied. *Result* : The arm has entirely regained its normal motion, and in spite of the unusual amount of bone removed the result is all that could be desired—the patient being again able to use it freely.

#### TREPHINING FOR EPILEPSY.

The patient, Geo. H—, was admitted May 1897 to the Hospital suffering from epilepsy. He gave a history of having been thrown out of a rig about a year before and alighting on his head. The epileptic seizures while in the Hospital were severe and frequent. The only evidence of any injury to head was a slight scar on right side of scalp and about half an inch from the sagittal suture near the lambda. The patient was willing to undergo an operation as his life was becoming a burden to him, and as there was nothing in the nature of the epileptic seizure to localize any injury, it was deemed advisable to trephine over the scar above referred to. On May 15th, under chloform narcosis, I made the usual horse-shoe shaped incision so as to expose the above point. On reflecting the flap back there was no fissure or other injury to the bone visible. The trephine was applied and a button removed and this also was apparently normal. The dura was incised and neither it nor the cerebral cortex showed anything abnormal. The wound was sutured and united in the usual time. The results of operative interference for epilepsy have not been brilliant, and so I was pleased to receive a card from the patient last month in which he states that he frequently has mild spells but has had none of the severe attacks since the operation.

#### DEFORMITY FROM A BURN.

The patient, M. T—, aet 11, was admitted under my care May 1897. Three years previously she had been severely burned, her clothes catching fire from a stove and the whole anterior aspect of left arm was burnt. Sloughing occurred and the resulting contraction flexed the forearm to such an extent that the hand lay across the chest and pointed to the opposite shoulder. Operation June 1897 the skin, on flexor aspect of forearm was dissected up, starting from about two inches above wrist and end-

ing at level of elbow joint. The biceps tendon, bicipital fascia and brachialis anticus were carefully divided and the arm straightened. The straightening, however, was limited, as no doubt the olecranon fossa was filled up since the arm had been flexed for three years and that too at an age when growth was so prominent a factor, hence I had made a posterior angular splint with a long screw joining two small uprights, one on each blade of the splint, and worked so that at each dressing of the arm a few additional turns of the screw kept an increasing strain directed towards extending the arm. For the large denuded surface left on the forearm, skin grafting by Thiersch's method was perfectly successful, and when she left the Hospital the arm was almost as straight as the other.

D. E. MUNDELL.

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#### SUMMER DIARRHŒA OF INFANTS.

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THE heated term is upon us and soon will be initiated "the slaughter of the innocents." In New York City during this present month of July more than 1,000 infants will die of Summer Diarrhoea; in many large American and European cities the mortality is even greater. Is it not strange, in view of all that has been done along the line of preventive medicine, this (preventable) disease should continue to number its victims by the million?

In the discussion of this subject there has always been confusion from the fact that the term Summer Diarrhoea has been used to cover every condition from a mere temporary derangement of the bowels to that most fatal of all infantile diseases—Cholera Infantum. It is our purpose to limit the use of the term to that form of diarrhoea which prevails as an epidemic, beginning in June, when the temperature reaches 60° F., increasing in virulence throughout July, then gradually declining till it disappears in September, when the temperature falls below 60° F.

Regarding the etiology of the so-called Summer Diarrhoea many and diverse theories have been advanced. Age, constitution, food, methods of feeding, teething, even thermic atmospheric and telluric phenomena have in their turn been brought forth to explain it. It is now conceded by the best authorities that while the above conditions may precede and even predispose to Summer Diarrhoea they cannot produce it. The recent labors of Baginsky in Germany, Booker and Siebert in America have demonstrated beyond doubt that this disease is of infectious origin; that it is due to a poison produced by certain forms of toxicogenic bacteria of the *Proteus* type; that milk is the natural culture medium of these bacteria and that milk is the vehicle by which these germs or their poisonous products are carried into the intestines of the child.

Clinically we have two forms of infectious or Summer Diarrhoea,

(1) Subacute Infectious Diarrhoea.

(2) Acute Infectious Diarrhoea, or True Cholera Infantum.

The Subacute form is milder in its course and the poison upon which it depends is less intense, but being more wide-spread it is responsible for a greater number of deaths than the Acute form. The Subacute also shows greater variation in its clinical course. We have mild cases in which the onset is gradual, the child is restless, fretful, elevation of temperature slight, may or may not vomit, stools are frequent, thin greenish or brown, in the course of a few days the diarrhoeal discharges sweep the poisons out of the system, the child becomes restful, stools less frequent, appetite and digestion improve and convalescence is established. In the more severe type the onset is sudden, symptoms develop rapidly, temperature  $103^{\circ}$  F. or  $104^{\circ}$  F., skin hot dry, vomiting and purging come on, thirst is insatiable, colicky pain is severe, a large quantity of flatus is expelled when the bowels move. The rapid absorption of the poison, the incessant vomiting and frequent watery discharges from the bowels make a profound impression upon the heart and great nerve centres, and very soon the whole aspect of the child is indicative of serious illness. When the initial shock has passed the symptoms usually abate somewhat and a child with good constitution and favorable surround-

ings may rally, but if the child be young or if the vitality be low, the symptoms persist, the prostration increases, and in a few days death will take place from exhaustion. Many cases of both the mild and severe forms have a tendency to relapse, they respond to treatment and improve for a time, then relapse, then improve again. Such cases may linger on till the cool weather and recover, or develop into chronic entero-colitis or die of some intercurrent disease.

Acute Infectious Diarrhoea or Cholera Infantum is a rare but fatal form of disease: All the symptoms are essentially toxic, the impression made upon the heart and nervous system is as sudden as it is profound. A child apparently well or perhaps suffering from some slight derangement of the bowels is taken with persistent vomiting and purging. The contents of the stomach are first thrown off. Then bile and mucus. The stools, for a time, may contain solid and undigested matter, but later on large, dark, watery motions are expelled with great force. As the disease progresses the stools are more frequent and are composed of almost pure blood serum. Meantime the temperature rises to 104° F. or 105° F., the pulse is weak and irregular, the tongue red and dry, thirst is extreme, the strength declines and the flesh wastes rapidly, and unless there be a reaction within forty-eight hours the condition will be one of extreme depression. In a few of the cases met with a favorable reaction sets in, vomiting ceases, the stools become less frequent, pulse improves, temperature and nervous symptoms subside and the child recovers. In the cases going on to a fatal termination a deathly pallor spreads over the face, the features are pinched, eyes sunken, skin clammy, extremities cold, radial pulse weak or absent, respiration shallow, then stupor comes and death.

Were it possible to secure for infants pure air, proper food, and absolute cleanliness of person and clothing, the treatment of summer diarrhoea would be comparatively easy; but in many cases the best efforts of the physician are thwarted and the success of his treatment limited by circumstances. The injunction of the ancients "obsta principis"—prevent the beginning—is peculiarly applicable. Every case of infectious diarrhoea has its beginning and at the beginning is the time to check it. And

now that a definite knowledge of the Etiology of infectious diarrhœas has brought to us greater possibilities along the line of preventive treatment, our health officers should see to it that every child has fresh pure air to breathe; that milk supplied to our cities is pure and germ free; they should enforce absolute cleanliness in every home and in every street. Mothers should be made aware of the fact that 97 per cent. of the children who die from diarrhœa are bottle-fed and that any mother who refuses to nurse her child (except there be a good reason) assumes a terrible responsibility. Those who have the care of children should know that a child requires *less* food and *more* water in the hot season.

When considering the curative treatment of infectious diarrhoea it must be borne in mind that we are dealing with cases of actual poisoning. The indications will therefore be,

- (1) To evacuate the poison.
- (2) To neutralize the depressing effects of the poison already absorbed.
- (3) To prevent the ingestion of any more poison.

The methods and details of the treatment will vary with (1) the intensity of the poisoning, (2) the stage of the disease when the case is first seen.

In subacute cases, where vomiting has been going on, washing out the stomach may be omitted, but copious draughts of hot water must be given. The bowels must be thoroughly irrigated. A gallon of warm saline solution (an ounce to the gallon) is made to flow slowly into the bowels, the abdomen being at the same time gently kneaded to facilitate the washing out of the poisonous matter. After irrigation a pint of cool water containing thirty grains tannic acid dissolved in two drams whisky should be thrown into the bowel and retained as long as possible. The tannic acid coagulates and prevents the absorption of any proted poisons remaining in the bowel. The whiskey is needed to stimulate the child after irrigation.

To clear the small intestines, which cannot be reached by irrigation, two grains calomel are given—one-tenth grain every hour. When vomiting has ceased a teaspoonful of barley or al-bumen water may be given as often as necessary to allay the

thirst. After the stomach has rested for 24 hours, a little meat broth, beef juice or gruel may be given every two hours. If there be much depression stimulants are to be given by the mouth if retained, if not hypodermically. Milk, being the natural culture medium for these bacteria, and also the vehicle for their dissemination, must be rigidly excluded from the diet. If the evacuation of the poison be thorough, the stomach be allowed sufficient time for rest, the exclusion of milk be absolute, careful feeding with stimulants as required will ensure recovery in the majority of sub-acute cases.

The treatment of acute infectious diarrhoea or true cholera infantum is very unsatisfactory. Most cases go on to a fatal termination uninfluenced by treatment. The only hope of success depends upon the ability of the physician to recognize the condition in the early stage, and to appreciate the necessity for immediate action—delay means death. When seen early the stomach should be washed out with saline solution (a teaspoonful in a pint). The bowels must be irrigated and calomel given in divided doses as in the sub-acute cases.

For the second indication, to neutralize the effect of the poison, nothing has been found to equal morphine and atropine. For a child, a year old, a tablet (morphine  $\frac{1}{4}$  gr., atropine  $\frac{1}{120}$  gr.) may be dissolved in an ounce of water, and a hypodermic of this given every hour till the system is quieted and a favorable reaction produced. To allay irritability and induce sleep 10 drops of Bromidia or 15 drops Tr. Hyoscyamus may be given in a little glycerine and water as often as required. Cerebral symptoms are best controlled by the ice cap, and when there is high temperature and restlessness frequent sponging will be useful. To allay thirst and supply the lack of fluid in the blood, a saline solution (three grains to the ounce) must be injected into the loose cellular tissue of the chest or abdomen—not less than a pint in 24 hours. Stimulants, drinks and nourishment must be given as prescribed for subacute cases. In cases of collapse hot mustard baths, hot bottles around the patient, and friction, etc., for the extremities, will be found beneficial.

The features peculiar to infectious diarrhoea which we wish to emphasize are :



1. It occurs only during the summer months while the temperature is above  $60^{\circ}$  F.

2. It prevails among infants under two years of age.

3. It is rarely, if ever, met with except in bottle-fed infants.

4. It is always due to bacterial poisons, milk being the usual culture medium.

5. These bacteria require a temperature of not less than  $60^{\circ}$  F. before they can grow or multiply.

6. Infectious diarrhoea is not dependent upon previous disease or unfavorable surroundings.

7. It is most prevalent and fatal among infants of low vitality and in filthy locations, but it often attacks vigorous healthy infants in the country and in the homes of the wealthy.

ISAAC WOOD.

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### THE ONTARIO MEDICAL COUNCIL.

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THE following students of Queen's University have passed the examinations of the Ontario Medical Council:—

#### PRIMARY.

C. H. Amys, J. F. Boyle, R. F. Carmichael, E. G. Cooper, W. N. Condell, H. G. Fleming, A. B. Ford, R. C. Hiscock, W. A. Hall, R. Hanley, A. E. Ilett, C. R. Johns, C. A. O. Morrison, J. W. McDermott.

#### INTERMEDIATE.

C. C. Armstrong, J. F. Boyle, W. N. Condell, R. Hanley, A. Ilett, C. A. O. Morrison, J. F. Mather, C. E. O'Connor, R. C. Redmond.

#### FINAL (FOURTH YEAR.)

J. F. Boyle, G. W. Collinson, A. B. Ford, A. E. Ilett, W. Moffatt, R. C. Redmond.

#### FINAL (FIFTH YEAR.)

Honors—G. W. Mylks. Pass—A. W. P. McCarthy.

## PELVIC INFLAMMATION.

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UNTIL recent years the views generally held and taught with reference to pelvic inflammation were exceedingly vague and unsatisfactory, and in many respects erroneous. Clinical observation was so seldom tested on the operating table that erroneous inferences remained, year after year, uncorrected by actual inspection. Almost every attack of pelvic inflammation was believed to be a cellulitis, and if on vaginal examination a hard irregular fixed mass could be felt on one or both sides of the uterus, the diagnosis of cellulitis was held to be established beyond doubt. It was not until the practice of abdominal surgery became extended, and opportunities of comparing the physical signs with the actual conditions present, that the real pathological changes began to be generally recognized and accepted.

Notwithstanding the enormous strides made as the result of clinical and bacteriological research, our most advanced teachers and operators are not yet wholly in unison in regard to the pathology of pelvic inflammation. The intimate relations existing between the Fallopian tubes, ovaries, peritoneum, cellular tissue and lymphatics compels an almost constant intermingling of the lesions of inflammation affecting them, and to such an extent is this true that a very large number of gynæcologists hold that there is no form of pelvic inflammation which did not at first have its origin in tube or ovary, and that every variety of such inflammation is secondary to that.

It seems to be pretty well settled that that form of inflammation long known as pelvic peritonitis, or perimetritis, is undoubtedly of tubal origin, the contents of a diseased and leaking tube having escaped into the pelvic peritoneum and set up septic inflammation there. The results of such inflammation largely depend upon the nature of the septic material which has escaped, that of gonorrhœal origin tending rather to the formation of bands of adhesions, while that containing pus-producing micro-organisms tend to the formation of local abscesses. There is still a form of inflammation existing within the pelvic walls having clinical features peculiar to itself, and which still maintains its own identity among many capable gynæcologists, notwithstanding

the efforts to disenthroned it—I mean pelvic cellulitis, or inflammation of the pelvic connective tissue. Undoubtedly a very large percentage of this form of inflammation is secondary, having its origin in one of the neighboring organs, and reaching the connective tissue by extension from them, but that the primary form does not exist has not yet been proven. If we turn to the distribution of the pelvic connective tissue we find that, except over the fundus uteri, it forms a layer under the entire pelvic peritoneum, parietal and visceral. The so-called “ligaments” of the uterus contain a greater or less quantity of it between the peritoneal folds of which they are composed, and in certain special situations it may be said to be abundant; for example, around the supra-vaginal portion of the cervix uteri, along the base of the broad ligaments, and between the bladder and symphysis pubis. That diffuse cellular inflammation tending to the formation of abscess can exist as a primary condition in other parts of the body, without visible means of infection, and in a part where, perhaps, the cellular tissue is less abundant, is a fact so well recognized as scarcely to be worth mentioning, yet the same form of inflammation, with the same tendency to suppuration, is denied to that connective tissue situated in a region where there is every possibility of infection.

From clinical observation of many cases, and from the subsequent study of them on the operating table, I am convinced that a considerable number of them did not have their origin in a diseased tube or ovary, but were primary in their inception, and that the uterus, tubes and ovaries were perfectly normal, both before and after. Two cases occur to me at the present moment which forcibly go to prove the contention. One, a lady in good circumstances, healthy and the mother of several children, developed, after an abortion, a cellulitis in the left parametrium, and from which subsequently three ounces of pus were evacuated through the vagina. A complete recovery was made in a few weeks. I repeatedly had opportunities of examining her, both before and since, and never had reason to suspect uterine, tubal or ovarian disease. A second, a young girl, almost eighteen years old, unmarried, perfectly healthy in every way, suddenly developed, towards the end of the menstrual period, pelvic

pain, chills and fever. A hardened mass soon formed in the left retro-uterine space, which was quickly changed to the boggy feeling of pus. At the end of the twelfth day several ounces of pus were evacuated through the vagina. In this case cultures proved that the pelvic inflammation and abscess arose from the colon bacillus.

In turning to the etiology of pelvic cellulitis it must be admitted that it is always a result of septic infection in some form, although some observers claim that it may be induced by sudden suppression of the menses. Case number two, referred to above, would rather point to such a cause as being a real one, had not cultures been made which showed its origin to be otherwise. The most common source of infection is the absorption of septic matter through laceration of the cervix uteri, or of the upper part of the vagina, occurring during labor. Other sources of infection are the various surgical manipulations practised on the vagina and cervix. On account of the close proximity of the pelvic organs to the rectum, vermiform appendix and sigmoid flexure, pyogenic bacteria may escape from one to the other and set up a pelvic inflammation.

Septic infection of the pelvic connective tissue may be due to any of the micro-organisms which find their entrance through the vagina into the uterus, and then into the pelvis by way of the tubes; or directly, by way of the lymphatics, through the parametrium, or vaginal vault. The gonococcus almost always travels along the mucous membrane into the tube, where its further extension may be arrested by inflammatory action there, or it may escape from the ostium abdominale and set up a localized peritonitis. The inflammatory process in such infection is almost invariably confined to the pelvic organs and their immediate environment, rarely causing more than a local reaction, and never giving rise to a general infection. Streptococcus infection usually occurs during a badly conducted puerperium, or after an abortion; or it is introduced by dirty instruments, or by applications or vaginal manipulations without proper antiseptic precautions. When the streptococcus gains entrance, it may invade the pelvis by the same route as the gonococcus, or it may penetrate the uterine wall, setting up an endometritis or metritis, and then a parametritis, forming a dense swelling which usually ter-

minates in an indurated plegmon or pelvic abscess ; or it may gain entrance to the pelvic connective tissue directly by means of the lymphatics. The staphylococcus, while comparatively rare, is sometimes obtained from pelvic abscesses, and the colon bacillus is sometimes found.

Setting aside those forms of pelvic inflammation in which the uterus, tubes or ovaries are first affected, it is my intention to confine my remarks to that form of inflammation which I have endeavoured to show may exist without involvement of these organs, namely, pelvic cellulitis having its origin in infection through those lymphatics which have their starting points in the vaginal fornices and infra-vaginal portion of the cervix. Pelvic cellulitis is often ushered in by a rigor. In puerperal cases the interval between the period of infection and the first manifestation of symptoms seldom exceeds a day or two. The general symptoms are those of a subacute form of septicæmia. There is usually pain in one or other iliac fossa, but it is not well marked, unless the inflammation extends to the neighboring peritoneum. Sometimes the local symptoms are so few and indefinite that the existence of an acute inflammatory process within the pelvis remains for some time unsuspected. In the early days of an attack of acute pelvic cellulitis physical examination gives but little information, but after the lapse of a few days the inflammatory exudation in the tissue of the affected area is at first doughy in consistence, but soon becomes densely hard. The cervix will now be found to have lost its normal mobility, and pressure on the supra-vaginal tissues on the affected side will elicit the presence of most exquisite tenderness, the patient often flinching on mere touching the part. It is seldom that both sides of the pelvis are equally affected, but it is by no means unusual to find the whole supra-vaginal portion of the cervix imbedded in a thick collar of indurated tissue, which more or less completely surrounds it. An area of uniform hardness may sometimes, at this stage, be felt in the abdominal wall beneath the muscles, usually taking the form of a broad band lying along the upper border of the inner portion of Poupart's ligament. Sometimes the exudation spreads upwards and outwards from above Poupart's ligament into the iliac fossa, interfering with the action of the psoas

and iliacus muscles, and leading the patient to keep the thigh flexed. In some instances the inflammation passes backwards, instead of forwards, producing an exudation in the tissue of one or both utero-sacral ligaments, in the tissue surrounding the rectum, and in that beneath the peritoneum lining the posterior pelvic wall. Where there is no suppuration the exudation becomes absorbed, and, in uncomplicated cases, gradually disappears. Unfortunately, in a very large number of cases such a termination is the exception, the earlier symptoms being soon followed by suppuration and the formation of a pelvic abscess.

The situation of the abscess and the position where it may be expected to point depend upon the direction in which the inflammatory exudation has extended. It may point at a site a little above Poupart's ligament, or it may extend downwards and appear to bulge most prominently into the vagina. In some cases, where it extends backwards, the abscess thus formed, owing to its anatomical relations, has no direct access to a free surface; relief therefore is much longer delayed and extensive burrowing may result. Sometimes the pus leaves the pelvis by the sciatic notch and follows the course of the sciatic and gluteal vessels. In other instances it makes its appearance in Scarpa's triangle, while not unusually it makes its exit by way of the rectum. In rare cases the abscess ruptures through the vaulted free surface of the sac, and the pus is poured into the abdominal cavity. Abscesses which open into the vagina may discharge their contents completely, the cavity collapse and heal, and the patient regain perfect health. If the opening is small the discharge will only take place when there is sufficient pressure within to overcome the resistance, and it may continue in this way for months, and even years, each reaccumulation being characterized by a return of pain, fever and distention. When the abscess opens into the rectum, if the opening is direct and large enough, and lies at the bottom of the sac, a rapid and complete recovery may take place. If, on the other hand, the abscess empties into the bowel by a minute orifice, or if the opening is in the upper part of the abscess, so that the pus only discharges when the sac is full, the discharge may go on indefinitely. An abscess opening on the abdominal wall rarely closes, because the opening lies at a higher level than the sac, and pus can therefore only escape as an overflow, or in certain positions of the body.

In the management of pelvic cellulitis it is pretty well acknowledged that when once an attack has been lighted up it is not possible to modify to any extent the course of the disease by any special medication. During the acute stage the patient should be kept absolutely at rest, the bowels freely open, poult-

tices applied to the lower abdomen, mild diaphoretic and febrifuge treatment, and prolonged hot vaginal douches, with, perhaps, some anodyne to allay the pain. When pus has formed, the simplest plan of treatment is the evacuation of the sac, but too early an operation, before the acuter symptoms of pus formation have subsided, is not advisable, particularly if the operation is to be a radical one, on account of the increased danger at such a time.

Vaginal incision and drainage of a pelvic abscess is now considered the best and safest method for relief. The proper point for puncture is posterior to the cervix, slightly to one or the other side of the median line; by the side of the cervix there is danger of wounding the uterine vessels or the ureter. However, the position of the uterine artery can nearly always be determined by palpation at the vaginal vault, and the exact situation of the ureter made out by its characteristic feel. Sharp-pointed scissors, curved on the flat, is an excellent instrument for making the necessary puncture—some prefer a small pair of uterine dilators, sharpened at the points, for the purpose. As soon as the sac is entered the blades are easily separated, being now in a free space, and by withdrawing the scissors, keeping the blades open, the opening is torn wider. With fingers, or with strong dilators, the opening is further enlarged, so that the pus may quickly empty itself and permit of subsequent free drainage. The sac, emptied of its contents, may now be thoroughly irrigated and a loose pack of washed-out iodoform gauze inserted into its cavity, and a few strips into the vagina.

Evacuation through the rectum is only admissible where there is such a marked area of softening that spontaneous rupture is imminent, and then the opening must be made as low down as possible to secure constant drainage. Under no circumstances is it allowable to make an opening high up, above the constriction between the utero-sacral folds. If nature makes an opening in such a position the gases and fecal matter enter the sac and the discharge is kept up for an indefinite period. When the pointing is high up, or when an opening already exists at this point, a wide counter opening should be made through the vaginal vault behind the cervix.

Evacuation of pelvic abscesses by the vagina, aided by the hand introduced through an opening in the abdominal wall, is called for when the abscess is not so clearly defined as to admit of operation by the vagina alone, but such a radical operation is seldom if ever called for when the pus formation exists in the pelvic connective alone and uncomplicated by other serious conditions.

R. W. GARRETT.

## CONTENTS OF VOLUME II.

ABORTION. R. W. GARRETT .....	99
ACCESSORY CAVITIES OF THE NOSE. DISEASES OF THE. J. C. CONNELL .....	25
ANNUAL MEETING OF THE COUNCIL OF THE COLLEGE OF PHYSICIANS AND SURGEONS OF THE PROVINCE OF ONTARIO. THE EDITOR .....	1
ANNUAL REPORT OF THE KINGSTON GENERAL HOSPITAL. JAMES THIRD .....	33
BOOK REVIEWS. W. T. CONNELL .....	37
BRITISH MEDICAL ASSOCIATION. R. W. GARRETT.....	15
BOVINE TUBERCULOSIS, ITS IMPORTANCE IN THE CAUSATION OF TUBERCULOSIS IN MAN. W. T. CONNELL.....	130
CANADIAN MEDICAL ASSOCIATION, PRESIDENT'S ADDRESS. THE EDITOR .....	35
CARCINOMA. W. T. CONNELL .....	9
CHRONIC STOMACH AFFECTIONS. JOHN HERALD .....	90
CONSUMPTION, THE PREVENTION OF. THE EDITOR .....	81
EXCISION OF THE ELBOW JOINT. W. G. ANGLIN .....	142
HYSTERICAL PHENOMENA AS AN EARLY SYMPTOM OF CEREBRO-SPINAL FEVER. G. C. G. WARD .....	69
INFANT FEEDING. J. WOOD.....	109
MEDICAL CURRICULUM. THE EDITOR .....	41
MEDICAL ETHICS, THE CODE OF. J. C. CONNELL .....	5
MICROSCOPY IN DIAGNOSIS. W. T. CONNELL .....	49
NEUROLYTIC HAEMOPTYSIS. E. RYAN .....	139
OBITUARY—DR. D. CUNNINGHAM. A. P. KNIGHT.....	137
OEDEMA OF THE EYELIDS. J. C. CONNELL .....	61
PNEUMONIA, THE TREATMENT OF. E. RYAN .....	54
POST PARTUM HEMORRHAGE. R. H. ABBOTT .....	86
PROGRESSIVE BULBAR PARALYSIS. T. H. FARRELL.....	73
PULMONARY TUBERCULOSIS, THE DIAGNOSIS OF INCIPIENT. JOHN HERALD.	
PELVIC INFLAMMATION. R. W. GARRETT.....	153
PERSONAL. THE EDITOR.....	128
QUEEN'S MEDICAL CONVOCATION. THE EDITOR .....	116
REFLEX ACTIONS. D. E. MUNDELL .....	113
SOME LABORATORY NOTES. G. W. MYLKS .....	134
SECRECY, THE LAW OF. J. C. CONNELL .....	106
SURGICAL NOTES. D. E. MUNDELL .....	145
SUMMER DIARRHOEA OF INFANTS. ISAAC WOOD.....	147
TREPHINING FOR HEADACHE. D. E. MUNDELL .....	46
THE KINGSTON MEDICAL AND SURGICAL SOCIETY .....	129
THE ONTARIO MEDICAL COUNCIL—PASS LIST .....	152
THE KINGSTON GENERAL HOSPITAL. THE EDITOR .....	121
ULCER AND CANCER OF THE STOMACH. JOHN HERALD.....	124
X RAYS. JAMES THIRD .....	64