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ON SOME PHASES OF A FAILING CIRCULATION.

BY

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Year by year the array of new facts, which modern research is constantly bringing to light, affords us new vantage points from which we are able to look back and see clearly the mistakes that have been made in the past. Owing to this enlarging view the physician finds that from time to time many of his concepts of disease have to be altered and with each alteration his therapeutics become more correct and more successful. Illustrations of this are to be found in every domain of medicine. This morning I desire to call your attention to one in the domain of the pathological physiology of the circulation, viz:—So-called heart failure—a misconception, as Janeway said in a recent address, hallowed by long usage, the alleged cause of more deaths than any other medical term. As popularly employed heart failure is merely a synonym of sudden death. It is a term made use of by many writers and teachers and yet it is a term often at variance with the facts as we now know them, and, therefore, misleading to us as therapeutists.

Sudden death at the height of an attack of lobar pneumonia was the illustration taken by Janeway and it is certainly a pertinent one. Permit me, however, to quote two out of several cases occuring in the wards of the Alexandra Hospital for Infectious Diseases, of which the details have been kindly given to me by Dr. Fyshe, the Medical Superintendent under whose care they were.

Case I.—M. N., female, aged 5 years, admitted on the second day of a well marked case of pharyngeal diphtheria. On admission a slight amount of laryngeal stenosis was present, but not enough to justify operative interference.

*Read before the Maritime Medical Association at their meeting in St. John, N.B., July 18, 1907.

Her highest temperature was 101°F; her pulse varied from 128 to 148; the cardiac dulness was normal. There were no adventitious sounds within heart or lungs. The patient re-acted fairly well to antitoxin, the laryngeal stridor disappearing within twelve hours. On the afternoon of the third day of the disease, however, the patient suddenly collapsed, the pupils became widely dilated, the heart action very rapid, the sounds indistinct. The breathing remained regular until death took place about twenty minutes after the beginning of the collapse. Microscopic examination of the heart showed absolutely nothing pathological in that organ. The liver, the lungs, and the spleen were normal beyond a certain amount of injection.

Case II.—G. W., male aged 19 years, was admitted to the hospital on the third day of a very severe attack of scarlet fever. His temperature while in the hospital varied from 103° to 105° F, his pulse from 140 to a rate uncountable. The notes on admission stated that he was a well nourished muscular adult with anxious facial expression; his pupils were equal and active; his conjunctivae much injected; the lips abnormally pale. A most intense dusky red rash covered the body. The breath sounds throughout both lungs were normal but the respiration was of a sighing character. The pulse was rapid, of small volume and low tension, and very shallow. Cardiac dulness was normal in extent, but the heart sounds were feeble. No adventitious sounds were present; he responded feebly to a normal saline injection and collapsed a few hours afterwards, during the administration of a second. The autopsy findings as to the cause of death were most unsatisfactory. Nothing definite could be found either macroscopically or microscopically leading to a fatal result.

If questioned as to the exact pathology of these suddenly fatal cases, the general answer would be that the heart muscle, as the result of the action of certain toxins, has undergone degenerative or inflammatory changes which so impaired its functional ability that it was unable to meet the demand made upon its ventricles by some slight extra strain. Such an explanation is plausible, but what does the post mortem show? A striking contrast between the empty heart chambers of such a death and the engorged auricles and right ventricle of true heart failure. Microscopically we find only a slight change in the muscle cells; rarely any significant inflammatory lesions. Looking back on the clinical history of such a case we note also the absence of all ordinary signs of a failing heart. No œdema, no venous stasis, no cyanosis, but extreme prostration; a blanched cool skin, and a rapid ineffectual heart beat.

The stage of collapse thus resembles the condition present after a severe hemorrhage, or in surgical shock.

This striking absence of all ordinary signs of heart muscle failure has made investigators question whether, after all, the heart is really involved in the circulatory failure. Romberg and several of his pupils in the Leipsig clinic, notably Pässler have recently investigated this problem very carefully and have published their results in a series of papers. At the outset they studied the mode of death in animals inoculated with one of the following infections; the pneumococcus, the bacillus pyocyaneus and the bacillus diphtheriae, and found that death occurred after rapidly developing symptoms of collapse similar in every way to the so-called heart failure in man.

They then repeated the inoculations in a large number of fresh animals and observed the blood pressure at short intervals. They found that it remained normal during the major part of the illness, only beginning to fall when collapse was impending.

Blood pressure, as we know, is dependant upon four separate factors, which may vary independently of one another:—

1st. The energy of the heart. 2nd. The peripheral resistance. 3rd. The elasticity of the arterial walls. 4th. The volume of the circulating blood.

The last two have little interest for us at the moment. The tone of the vessels regulating to a great extent the peripheral resistance depends upon impulses from the vaso-motor centres. Experiments show conclusively that this vaso-motor tone is an absolute necessity for the maintenance of the circulation, not only in the arteries, but also in the veins. Any grave injury to the vaso-motor center is followed by stagnation of the blood in the veins, and eventual heart failure because no blood is returned to it. To determine what factor was the cause of the great fall in blood pressure, Romberg and Pässler tried the effect of four procedures on their infected animals.

1st. Abdominal massage, which increases the work of the heart by emptying the abdominal veins into the right ventricle.

2nd. Temporary compression of the descending aorta above the diaphragm, calling for a maximum effort of the heart by greatly increasing the peripheral resistance.

3rd. Faradic stimulation of the nasal and anal mucosa producing extreme reflex vaso-constriction, if the medullary vaso-motor centre is intact.

4th. Transitory compression of the trachea producing asphyxia which stimulates strongly both medullary and spinal centers.

They found that with the first signs of impending collapse the heart action became more rapid but the blood pressure began to fall. The chief change noted at this time was a distinct diminution of the reflex sensibility of the vaso-motor center. The heart maintained its full vigor, and, even in some cases, by increased action, counterbalanced the tendency to fall in blood pressure.

When complete collapse set in, blood pressure sank rapidly, an entire absence of any vaso-motor reaction to sensory stimulation and even to the effect of asphyxia was now observed, but abdominal massage or temporary compression of the aorta still caused prompt elevation showing only a slight impairment of the reserve force of the heart. In the pneumococcus and pyocyaneus infection, this impairment was very slight, and could be explained by defective nutrition; but in the case of infection by the bacillus diphtheriae the vigour of the heart beat was distinctly lessened, and on autopsy the heart muscle showed evidence of parenchymatous degeneration. Even this, however, was of minor importance; the real cause of death being, in all cases, complete loss of vaso-motor tone. It seemed to be quite unessential what variety of organism was the infecting agent.

The question still remained as to which part of the vascular system was paralyzed. To determine this, barium chloride, which raises blood pressure by direct action on the peripheral nerves, and the muscles of the small arteries, was injected. Under its action the peripheral vessels responded almost as promptly as in the normal animal, indicating that they were not at fault. Paralysis of the vaso-motor center was evidently the chief cause of the collapse. As a consequence of its paretic condition the vessels lost their tone, the blood accumulated in the large veins of the splanchnic area and was not returned to the heart, and a profound anæmia of the brain, muscles and skin quickly developed leading to rapid death.

In other words the animals, under the influence of the poison on the vaso-motor center, were bled into their own veins and the heart failed for want of blood. That the heart muscle was not at fault was clearly demonstrated in one of their experiments in which, during a severe infection, by the bacillus pyocyaneus an endocarditis involving the aortic valve was developed. In the course of a few days cardiac hypertrophy set in; the animal died later on of vaso-motor paralysis, but the heart was found to have increased about one-third in weight.

Failure of the circulation taking place at the height of the infection, and due to vaso-motor paresis is thus to be sharply differentiated from

failure of the circulation taking place during the later stages of the disease, or actually after the febrile reaction has subsided, and due to a myocarditis. The former is characterized clinically by softness and emptiness of the pulse, and by a rapidly failing blood pressure. The latter is characterized by smallness, irregularity and inequality of the pulse, with indications of some dilation of the chambers of the heart, and developing signs of mitral insufficiency. The former tends to terminate rapidly in death; the latter develops more slowly and eventually all the indications of a failing compensation can be noted. At the autopsy we find no change of any moment in the heart muscle in the cases of vaso-motor paresis, while in the other class there are the signs of an interstitial myocarditis. In the past there has been much confusion of these two conditions and drugs have been unnecessarily and unjustly blamed. For successful treatment it is essential that we recognize what the condition is that we are dealing with before we decide on the therapeutic measures to be employed. In the early stages of vaso-motor collapse pure cardiac stimulants cannot be expected to have much, if any, value. Digitalis is the only exception, and it may do good, not as a cardiac stimulant, but owing to its action on the vaso-motor center and peripheral vessels. Atropine in small doses has also a definite action on the medullary centres and small arteries, and, I think, I have seen some benefit from its administration hypodermically 1-100 gr. twice a day. Strychnine also may be employed, but only for its general effect, and as a stimulant to the respiratory apparatus. Caffein has some value and is superior to camphor. The introduction of normal saline solution is capable of raising blood pressure for a limited period, and it may be associated with small doses of adrenalin. The action of both, however, is fleeting. It seems almost unnecessary to say that nitroglycerine and its allies are distinctly contraindicated. As a mechanical measure raising the foot of the bed twelve or eighteen inches, may be of distinct benefit favouring the return of the blood to the heart.

With our present knowledge undoubtedly one of our most potent vaso-motor stimulants is cold, acting as a peripheral stimulant. It may be employed either in the form of a cold bath or ice-pack or locally. From the experiments of Romberg and Pässler we note that all stimulants have a rapidly lessening value as the stage of collapse approaches. Its action is, therefore, chiefly by way of prevention, and its value is to be reckoned, not by the fall in temperature so eagerly looked for by nurses, and which we know is always followed by more or less reaction, but by a rise in blood pressure, and fall in the frequency of the pulse. Much

of the benefit derived from tubbing in typhoid, is, I think, to be attributed to a stimulating action on the vaso-motor center.

Failure of the circulation in the later stages of the disease with developing signs of cardiac weakness is to be treated in similar lines to that of a degenerative myocarditis.

Closely allied to the collapse met with in acute infectious disease is the condition known as shock in surgery, due to exhaustion of all the important medullary centers, but in which vaso-motor depression plays the chief part. Crile's experiments show that in shock the heart muscle and its nervous mechanism is unimpaired, as is also that of the vaso-muscular system, but the vaso-motor center fails to respond to any stimulus, irritative, electrical, physiological or pharmacological. Vaso-motor stimulants while the center is so exhausted have little action, strong coffee, or a hypodermic of caffein citrate is one of the most effective. Normal saline solution raises, but cannot sustain, blood pressure and found that in a certain number of their patients the blood definite when shock is associated with profuse hemorrhage. Crile recommends the addition of adrenalin to the saline in the strength of 1-50000 to 1-100000 and directs it to be given intravenously, very slowly and continously.

In addition to these measures, absolute rest must be secured. The flow of blood to the heart may be assisted mechanically by raising the foot of the bed and in some cases by gentle massage of limbs and abdomen. The extremities must be kept warm.

There is another form of circulatory failure in which I have recently been much interested; the failing heart of chronic arterial hypertension. During the last few years, much study has been devoted to the condition of increased blood pressure, and perhaps a few remarks on this subject may not be amiss.

When physicians first obtained an instrument to measure blood pressure and found that in a certain number of their patients the blood pressure registered considerably above normal, anxious endeavours were at once made to reduce this excess by the administration of all kinds of vaso-dilators, and again therapeutics were sneered at, because they failed, in these cases, to effect a permanent reduction in the blood pressure. Only slowly did physicians realize the fact that permanent high blood pressure in certain individuals is a necessity of life and is, in a great measure, a compensatory manifestation. A recent writer says, "It is to be regarded as one of the great advances of modern medicine, that we are now able to read between the lines, so to speak, and to obtain

a new and clearer insight into pathological processes." Inflammatory lesions themselves are but the wall the tissues build against foreign invaders. The hypertrophied left ventricle fits itself for the extra burden laid upon it. What rash therapeutist would wish to prevent or hinder such changes? So with increased blood pressure. It certainly is not a condition to be desired; but it is, in many cases, to be regarded as nature's effort to prevent a greater evil; an effort to keep the circulation active through peripheral areas, which through the action of toxic products, metabolic or otherwise, or from a more mechanical interference such as rigid arteries and the like, have their vascular area abnormally limited.

Let me illustrate my meaning. Persistent high arterial pressure is, as we all recognize, one of the cardinal symptoms in chronic Bright's disease. Pässler's researches on this point are noteworthy. By excision of one kidney, and the removal of successive portions of the remaining one he was able to bring about the varying grades of renal insufficiency, uncomplicated by inflammatory or toxic influences. Animals thus operated upon, developed, first, high blood pressure; this was followed by a cardiac hypertrophy and finally by toxæmic symptoms. Very interesting also is the effect that cerebral compression has in raising blood pressure, described in such an interesting way by Leonard Hill.

Sudden cerebral effusion of any nature, owing to the fact that the cranio-vertebral cavity is closed, produces by compression an acute cerebral anæmia which would inevitably lead to loss of function and death if the medulla did not make a counter-balancing effort. Automatically, the vaso-motor center raises blood pressure above the intracranial tension, and blood is kept circulating through the centers. If the compression increases, the vaso-motor center follows with another rise and so on, and thus an effort is made to keep just ahead of the advancing brain pressure. Each rise, however, is not steadily maintained. Over-compensation is succeeded by a fall then a rebound again, giving rise to the Traube-Hering waves in sphygmomanometric readings. Clinically we have all noted a similar rhythmical variation in the action of the respiratory center in the well known Cheyne Stokes breathing, deep breathing coinciding with the period of high blood pressure and established circulation and apnœa with its interruption.

In this effort of the vaso-motor center, blood pressure often reaches a great height; nevertheless, even these high figures may be regarded for the time as salutary. In practice, however, the question arises frequently, Is hypertension in all cases necessary and desirable? May not some part of it represent an over-effort on the part of the organism, a

functional hypertension added to an essential one, or in the case of the cerebral effusion is not this extreme high blood pressure but part of a vicious circle which the physician must certainly attempt to break?

Careful and repeated examinations only will enable us to answer these questions. One point may be borne in mind. Temporary rises occur more readily and more frequently in patients with permanently increased blood pressure, compared with those in whom blood pressure is normal.

The treatment of chronic hypertension must be chiefly dietetic and hygenic. The diet must be made simple and very moderate in amount so as to avoid all excess of food. In this matter Chittenden's experiments on the amount of food necessary to maintain nutrition are extremely valuable. Except in extreme cases moderate exercise is beneficial. When it cannot be taken general massage may take its place with benefit. I have much faith myself in the daily use of a saline laxative especially if combined with the occasional use of a mild mercurial, grey powder or blue pill. A course of one of the nitrites is of distinct value in all exacerbations whenever blood pressure rises above what may be regarded as normal for the special individual. I have not seen any appreciable value from their continuous use in essential hypertension; indeed, such is not to be expected.

I have some faith, however, in the continuous administration of one of the iodides in small doses. I do not think they directly lower blood pressure as some have stated, but they do appear to lessen the viscosity of the blood, a distinct advantage in cases of hypertension.

In all these cases, however, the time comes when the heart begins to weaken, and as a result the blood pressure falls, and gradually all the signs of an uncompensated cardiac lesion develop.

A few years ago, under the delusion that the high blood pressure, in these cases, was the important condition I feared to employ digitalis, except in small doses, and only when combined with full doses of the nitrites. I was surprised, however, to find that in many of my cases I had no results until I had gradually raised the amount of my digitalis until I had reached full doses, and the blood pressure had gained its former level. Only then did the œdema disappear and the signs of stasis subside. Many trials have convinced me that such patients demand digitalis in fairly full doses sufficient to maintain blood pressure at its high level lest their capillary circulation fails, and venous stasis develops.

We cannot, however, be forgetful of the fact that all those suffering from permanent high blood pressure are specially liable to sudden and

dangerous complications; anginal seizures, hemorrhages, uræmic convulsions, and the like. In the presence of such acute exacerbations threatening life, nitrites must be employed freely in larger doses and one need not hestitate to increase still further the dosage if the symptoms continue or become aggravated. Venesection in such cases may sometimes prove an effective agent if the nitrites fail us, or the case is very threatening. It seems almost unnecessary to add that at this stage of the disease rest in bed and the simplest diet are imperative.

A CASE OF TYPHOID FEVER. DEATH BEFORE ULCERATION.

BY

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(From the Pathological Laboratory of the Montreal General Hospital.)

Patient V.M., male, aged 25. Occupation fireman. Admitted to the Montreal General Hospital on June 5, 1907, complaining of headache, diarrhœa, weakness, fever and loss of appetite.

History of the present illness:—Onset very indefinite. Says he has felt run down for the past three weeks, during which time he has had an occasional headache, no appetite and felt listless. During the past few days has had diarrhœa which has gradually become worse. On June 3rd was at work all day feeling fairly well. That night, however, he was seized with severe headache and diarrhœa and felt chilly. The next day June 4th, was feeling much worse and remained in bed. A doctor was called in, and, suspecting typhoid fever, had the blood tested for the typhoid reaction, the result being negative. Admitted to the hospital June 5th. Present condition (June 7th): Patient is a well nourished and well developed man of 25 years. Is in a semi-delirious state into which he passed a few hours after admission. He is constantly muttering to himself, reaching out his arms as though trying to grasp some object in front of him, and picking at the bed-clothes. When aroused he will do as he is asked and occasionally will answer questions rationally. A few rose spots, fading on pressure, are present for the first time on the upper part of the abdomen and lower chest. Temperature since admission has ranged from 104 4-5 to 106. Patient does not respond to the baths. Pulse varies from 96 to 112, is of small volume, low tension, regular and dicrotic. Heart sounds are distant and weak; there are no murmurs. Blood count shows 6,200 leucocytes: complains of abdominal pain; abdomen is markedly distended: vomited several times: has diarrhœa and incontinence of fæces: slight epistaxis occurs at inter-

vals: spleen enlarged and easily palpated. At autopsy the following important changes were noted. There are immumerable, small, discrete, hæmorrhagic spots in the subcutaneous tissue over the back, on the anterior surfaces of both thighs, the sides of the abdomen and about the shoulder joints. The mesenteric lymph nodes are greatly enlarged, some being the size of walnuts. They are soft and on section are of a dark brown colour and hæmorrhagic. The retroperitoneal glands are also moderately enlarged. Numerous large and small subepicardial hæmorrhages are seen on the anterior and posterior surfaces of the heart about the auriculo-ventricular groove. Similar hæmorrhagic areas are found beneath the endocardium in the left ventricle. The myocardium in the gross is of good colour and consistence. Sections stained for fat in Sharlach R. show marked fatty degeneration in the form of finely divided particles scattered diffusely throughout the muscle fibres. Spleen:—wt. 565 grams. Greatly enlarged, being five times the weight of a normal organ. Capsule smooth and tense. On section the pulp is swollen and bulged; is of a dark red colour, moderately soft and friable, in places distinctly hæmorrhagic.

Intestines:—The mucosa throughout the jejunum, ileum and colon is swollen and hyperæmic. All the Peyer's patches are greatly swollen and project above the general surface as large flattened plaques, which are soft, intensely reddened and show no ulceration. The solitary follicles both in the small bowel and in the colon, stand out very prominently as small elevated nodules, 2 to 4 mm. in diameter, are soft and of a dark red colour. No ulceration is to be seen anywhere. Kidneys are enlarged and intensely congested.

The other organs of the body show nothing of special note.

BACTERIOLOGY.

A pure culture of a Gram-negative motile non-gas-producing bacillus is obtained from the spleen. The cultural features in every respect correspond to B. typhosus. In semi-solid medium there is a heavy cloud but no gas. Litmus milk is turned a lilac colour, denoting slight acidity which is permanent. Dextrose and mannit-serum-water containing litmus are reddened and coagulated. Lactose and saccharose serum-water are not changed. This organism is agglutinated with the blood of the patient in a dilution of 1-80; also with the blood of a patient in the ward who undoubtedly has typhoid fever. In this latter case the reaction is positive in a dilution of 1-20. Typhoid bacilli from stock culture are agglutinated in the patient's blood in a dilution of 1-80. Para-

typhoid alpha and beta in dilutions of 1-20 are negative. See following tables:—

Semi-solid	Litmus Milk	Dextrose Serum Water	Lactose Serum Water	Saccharose Serum Water	Mannit Serum Water
Clouded	Slight	Acidity plus	No change	No change	Acidity plus
No gas	acidity	Coagulation	24 hours	24 hours	Coagulation
24 hours	Permanent	24 hours			24 hours

The histological lesions in the various organs such as the intestines, spleen and lymph nodes are specific of bacillus typhosus infection. In general, sections of spleen show marked congestion. The Malpighian bodies are everywhere swollen. The most noticeable feature in the sections is the presence of great numbers of large phagocytic cells containing chiefly red blood corpuscles. These cells are found filling up the blood sinuses, and often contain twenty or more erythrocytes. Many of the lining endothelial cells are swollen and show mitotic figures. The lymphoid and plasma cells throughout the pulp are moderately increased.

In sections of Peyer's patches and of the lymph glands, here also the most striking feature is the presence of large numbers of phagocytic cells similar in every respect to those in the spleen.

The sinusoids of the liver contain many of these large phagocytic cells which Mallory claims may come from the spleen through the portal circulation and are arrested in the sinusoids. Places are seen in which the blood channels are occluded by these cells, giving rise to definite areas of focal necrosis.

Here we have a most unusual case of typhoid fever. Clinically it is of interest only on account of the very severe toxæmia from which death resulted during the first week of the disease. However, it is in the pathological aspect of the case that the chief interest lies.

To fully understand a pathological lesion we must follow and study every step of its development, especially the initial changes. Where lesions can be produced experimentally this is a comparatively simple matter; but on account of the difficulty of producing typhoid fever in animals we are dependent on material from post-mortem examinations. As a rule death from typhoid fever does not occur until the third week of the disease or later. In this case death occurred on the seventh day, before ulceration had taken place in the intestines, so that here we are afforded a very exceptional opportunity of studying the earliest histological changes.

These early lesions occur almost exclusively in the lymphoid tissues of the intestines, the lymphatic glands and the spleen. They are characterized by the presence of enormous numbers of large epithelioid cells which arise from the endothelial cells lining the lymph channels. Many of these cells while still a part of the vessel wall show mitotic figures. This great proliferation of the endothelial cells and later their increase in size resulting from their phagocytic function, give rise to the splenic and lymphatic enlargement.

Mallory has pointed out that this proliferation is due to the direct action of a diffusible toxin, and further that a proliferation of the endothelium to such an extent as described above and so well depicted in the spleen and lymph nodes, is characteristic of the poison elaborated by B. typhosus Other bacterial toxins stimulate endothelium to proliferation but in no infection do we meet with any such picture as that in typhoid fever.

In any ordinary case of typhoid fever, that is one in which death occurs during the third week of the disease or later, we find these large phagocytic cells free in the blood channels and lymph spaces in the active stage of proliferation as in this case.

The histogenesis of these large phagocytic cells in typhoid was always a source of controversy, until Mallory after studying such a case as ours, was able to trace the changes in the endothelium from that of simple swelling, on up through karyokinetic division of the still attached endothelium, to its separation and migration from the vessel wall. Similar steps are noted in the histological study of this case and I wish to place it on record as bearing out Mallory's observations.

SERUM SICKNESS AND SERUM DEATH: A COMPILATION OF SOME RECENT LITERATURE.

I.—THE SERUM DISEASE.

BY

CAMPBELL P. HOWARD, B.A., M.D.

The contributions of the Vienna School to the study of "Die Serum-Krankheit" offer most interesting reading to those interested in the practical questions of immunity. Since the introduction of serum-therapy there have been noted by many clinicians a series of phenomena occurring in patients who have received injections of the various bactericidal or antitoxic sera. In 1905 Pirquet and Schick published a monograph (Die serumkrankheit: Leipzig und Wein: Franz Deuticke) in

which they have collected all the clinical and experimental data on the subject.

General Clinical Picture:—Occasionally it is noticed that 8 to 12 days after an injection with varying amounts of serum, there appears at the site of inoculation an exanthem, usually urticarial in character, which rapidly involves the remainder of the body. This persists for but a few hours or at the most for 2 to 3 days. With the appearance of this rash the body temperature is elevated. Local glandular enlargement is common, but general glandular enlargement also occurs. Almost invariably there is œdema of a considerable degree, resembling in its site that of nephritis. In a few cases only there is a slight albuminuria on the third or fourth day after the appearance of the œdema. The general condition of the patient is as a rule little or not affected. In cases of long duration, prostration and emaciation may occur. Convalescence is rapid and occurs on the third or fourth day. A fatal termination is rare, and was never seen by Pirquet and Schick.

The incubation period is a very definite one. While immediately after the injection there is local redness, tenderness and slight swelling as a result of the trauma, these disappear in a few hours. Eight to twelve days later, without the occurrence of any prodromata the local and general reactions occur. The duration of the incubation period depends largely upon the disposition of the injected organism and not upon the nature of the serum. How it can be modified by reinjection will be seen later.

Fever is one of the commonest of the symptoms and is more constant than the exanthem. It is remittent in character with diurnal variations of 1° to 3° centrigrade. It lasts until the end of the clinical phenomena. The duration and intensity of the fever depend partly upon the individual disposition and partly upon the amount of serum injected. It is more apt to be the highest when associated with a morbilliform eruption, moderate with the urticarial and scarlatinal forms, and lowest with the erythematous type. The fever may be entirely absent and does not depend upon the exanthem, for both fever and exanthem are co-ordinate effects of the same cause. A gradual lysis with a progressively lower evening temperature is of valuable prognostic significance.

The Exanthem:—Hartung recognized four varieties: (1) Urticarial (2) scarlatiniform (3) morbilliform (4) polymorphous and exudative. It is impossible, however, to place certain of the exanthems of the serum disease under one of these four divisions. The rash appears first at the site of inoculation and may sometimes form the only cutaneous manifestation. Over the remainder of the body it is mostly symmetrical in

its distribution. The morbilliform and exudative forms occur mostly on the extensor surfaces of the extremities. The urticarial form, which is often very irritable, may be so extensive as to become confluent and form an œdematous infiltration of the skin, most marked at the site of inoculation, but sometimes also involving the face. Very occasionally hæmorrhagic eruptions occur.

Glandular enlargement:—The swelling of the glands at the site of the inoculation is one of the most constant symptoms. The local involvement, which early in the incubation period is slight, suddenly becomes marked and may be accompanied by a general glandular enlargement. There is pain and tenderness in the affected glands. This glandular enlargement occurs at the onset of the disease and subsides before its termination and is, therefore, of considerable prognostic value. Much less common is a moderate enlargement of the spleen which may persist for a couple of days.

Leucopenia:—During the incubation period the leucocyte count gradually increases and suddenly upon the appearance of the symptoms of the disease shews a considerable fall. This period of leucopenia towards the end of the disease is succeeded by a normal count. The leucopenia is due almost entirely to a diminution of the polynuclear cells.

Joint manifestations:—Joint pains, according to Hartung were present in only about 1.9 per cent of cases. The metacarpo-phalangeal, wrist and knee joints are involved in order of frequency. In addition to the pain, there is tenderness and limitaton of movement, but no other objective sign.

Oedema:—This is an almost constant symptom. It is not only visible and palpable but can be estimated by an increase in the body weight. In its situation it resembles the œdema of nephritis. It expresses the duration of the disease, hence its subsidence is also of great prognostic

Albuminuria is found only rarely and that at the height of the disease, two or three days after the appearance of the œdema, that is in the second to third week after the injection of the serum. It never exceeds ½ pro mille. In the urinary sediment one may find a few blood corpuscles and casts. A hæmorrhagic nephritis has once or twice been seen. value.

The mucous membranes of the mouth and throat remain uninvolved in marked contrast to their condition in measles and scarletina. On the other hand a diffuse bronchitis occurs not infrequently and in a few cases a bloody diarrhœa.

General symptoms:—In a mild case the general condition of the patient is practically undisturbed. Restlessness and slight irritability

may be present. Vomiting is an exception. In prolonged cases a marked listlessness, anorexia and in the severest cases a profound prostration may occur.

Differential diagnosis:—The following points will aid in the differentiation of the serum disease from measles. 1. The time of the appearance of the rash (7th to 14th day after the injection). 2. The first appearance at the site of the injection. 3. The local glandular enlargement. 4. The absence of involvement of the mucous membranes. In scarlatina (1) the initial vomiting (2) the angina (3) the high fever and (4) the infection of other children in the ward are all points which will aid us.

Prophylaxis:—Ruffer and Daut have shewn that the serum disease is less frequent the smaller the amount of horse serum used. Hence one should aim at using the most concentrated serum possible. Further, Bujwid has demonstrated that the fresher the horse serum, the more toxic it is: hence he recommends that the serum should be kept a few months before use. Sproncks has also shewn that heating the serum to 59°C lessens its toxicity, and rarely produces an exanthem.

Reinjection:—Pirquet and Schick in 1903 shewed experimentally that the organism retains the ability for a long time upon further inoculation of the pathogenic substance to respond more quickly with the disease phenomena and to complete the process in a shorter time. This is seen clinically in the tuberculin reaction in a tuberculous patient, in revaccination for small-pox and following the administration of various antitoxic and bactericidal sera. Pirquet and Schick pointed out that a certain interval must elapse between the first and second inoculations for this increased susceptibility to occur. If the interval between the two injections be three to six weeks an "immediate reaction" (sofortige Reaktion) occurs i.e. the incubation period is reduced to 24 hours. If, however, the interval between the injections be a long one as three months or more, an *accelerated reaction* (beschleunigte Reaktion) occurs i.e. the incubation period is shortened from 7 to 12 days to 5 or 6 days. Further upon reinjection it has been shewn that the serum disease occurs more frequently, requires a smaller dose of serum for its production and is accompanied by more intense symptoms.

The immediate local reaction:—It has been noted that in the reinoculated the local œdema at the site of inoculation was more marked, and out of all proportion to the amount of serum used, being in this respect unlike the local reaction in previously uninoculated people. The *immediate general reaction* implies the appearance of the fever, rash and other general symptoms within the first 24 hours after the injection.

This occurs not only in re-injected people but also occasionally after primary inoculations. In one case Pirquet and Schick noted a profound and alarming collapse. They believe that lethal cases have occurred only when an intravenous injection—purposely or accidentally—has been carried out. As far as they know all the fatalities have followed primary inoculation.

In the *accelerated reaction* the incubation period is 5 to 7 days and differs from that of the immediate in being unaccompanied by the local œdema. Fever, exanthemata, joint pains, and œdema occur after the onset of the disease. As to whether the "accelerated reactionability" will endure the entire life of the patient is as yet unknown: the longest interval on record is 7½ years. The occurrence of an "immediate" or an "accelerated reaction" can be considered to be diagnostic of a previous injection with a specific serum.

Theories of the serum diseases—Hamburger and Moro believed that the phenomena were due to the formation of "Precipitins," which by interference with the circulation mechanically caused the exanthem. It was afterwards shewn that in the living blood no precipitin was produced. Pirquet and Schick also demonstrated that the precepitin appears much later than the phenomena of the disease and may be entirely absent.

Pirquet and Schick believe that the phenomena of the disease are produced by the meeting of "antigens" and "antibodies." The antibodies are not identical with the precipitins. There is then a chemical interaction between the antigens of the horse serum and the antibodies of the vital reaction. Bail in 1905 called attention to the rapidity with which tuberculous guineapigs die when reinfected with B. tuberculosis, or when healthy animals are injected with bacilli plus the body fluids from a tuberculous animal. According to Bail this phenomenon of "rapid death" depends upon the presence of "aggressins," which are the products of the bacteria against the bactericidal substances produced by the infected organism: these aggressins paralyze the leucocytes, and upon a second inoculation are able to act with a more complete virulence.

Wolff assigned the phenomena to the presence of an "endotoxin;" a substance contained in the body of the bacterium which on the destruction of the bacteria through the bacteriolytic serum, is liberated and has an injurious action on the organism. This endotoxin is more quickly set free upon second dosage. In other words Wolff like Bail believes serum disease to be a *bactericidal* immunity in contradistinction to the *antitoxin* immunity of Pirquet and Schick.

Behring and Kretz assign to *amboceptor* production the principle role. Upon first injection there are developed specific side chains: if re-injection

occurs during the production of the latter, there occur many more side chains to the toxicable substance: these new side chains are greedier than those already destroyed and aid the toxic influence as "intermediate bodies." Quite recently Gay Southard (Jour. Med. Research, vol. 16:) have published their views on serum anaphylaxis i.e. increased susceptibility of the serum of the guinea pig. They believe that this hypersusceptibility to intoxication by horse serum in animals previously inoculated with serum "is due to the non-neutralization and non-elimination by the animal body of a factor in the serum for which they suggest the name "anaphylactin." The intoxication caused by the second injection depends upon factors of the serum other than anaphylactin. These factors correspond to constituents of the serum eliminable by the animal body. The reaction of intoxication would seem to be a cellular one, "dependent upon a heightened power of assimilation on the part of the cells which have been subject to the anaphylactic substance over a definite period of incubation."

Before closing one must state that the comparatively rare occurrence of the severer manifestations of the disease in primary inoculations and still more in reinoculations, does not mitigate against the usefulness of antidiphtheric and other sera.

SUDDEN DEATH FOLLOWING SERUM INOCULATIONS.

BY

OSKAR KLOTZ, M.D.

For the last ten years experimentors on the subject of immunity have devoted most of their time to determining the amount and character of resistance which the animal body may develop against foreign substances. This resisting power has been amply shown to develop against certain toxins of both animal and vegetable origin. Several views have been brought forward in explanation of this process of immunity, but the enunciation of Ehrlich has found most favour and has withstood the most critical tests applied to it.

While serum therapy was being investigated by innumerable experiments, the investigators not infrequently encountered a phenomenon which seemed wholly opposed to the teachings of immunity. It was found in some cases, that the experimental animal, instead of developing an increased resistance to the foreign substance, which, in a single dose, was entirely innocuous, would become highly susceptable when the animal was "sensitized" towards it.

These findings in experimental animals were found to be parallel to those noted in man. Over three hundred years ago, an attempt was made to restore the blood quantity in the human subject by direct transfusion of another animal's blood. Cases of anæmia, poisonings, and severe infections were treated by transfusing the blood of sheep. The practise, however, fell into disuse as the dangers from this method became apparent. The treatment was associated with high fever, embolism and hæmorrhages, with death in a certain number of cases. These symptoms have been stated to be due to the hæmolytic and clotting action of the foreign serum. Excess of fibrin is liberated, which, in conjunction with the debris of the broken down red blood cells leads to a blocking of the capillaries in different organs. In some instances the results were not so severe and an urticarial rash alone gave evidence of the unfavourable reaction of the transfused blood.

An attempt was also made to replace the blood solution by milk, but with no better results. A foreign element was present in the milk which led to results quite similar to the introduction of foreign blood.

With the introduction of antidiphtheritic serum, the process of injecting an alien serum into the human body became more frequent, and this practice has continued ever since, and has extended to a variety of other infections besides diphtheria. It was, however, not very long after the introduction of serum therapy that the ill effects of even small doses of serum were noted. The majority of these effects consisted in urticarial rashes and a temporary nausea. In other instances, the symptoms were more severe, being accompanied by vomiting, faintness and respiratory distress, while not a few developed arthritis later on. In a few cases death has been the result of the serum inoculation. Gottstein in 1896 collected eight cases of death following the injection of serum, four of whom had received the serum, only as a prophylactic measure. Rosenau and Anderson have in 1906 collected nineteen cases in the literature, of this unfortunate accident, and they are personally aware of several more cases which have not been reported. The nature of the death following the administration of serum is hardly to be confounded with that of any other disease, even when diphtheria is present. The symptoms in man may come on within five minutes of the treatment, with collapse, unconsciousness, convulsions and death as the result. Death, in some instances, takes place within fifteen minutes, and the symptoms are alike whether the patient is suffering from diphtheria or not. The following cases illustrate the nature of the disease:—

A characteristic case is reported by Dr. Thun, (Cent. f. Kinderheil, 1898, iii, p. 293). G. I., a child of 3 1-2 years was given Behring's

antitoxin at 12 o'clock noon. There was diphtheria in the family, and the child had complained of slight sore throat in the morning. Child was then put to bed. One hour after the administration of the serum, she became restless, and threw herself about. Her face became pale, and the extremities cold. There was no difficulty of breathing. At six o'clock the doctor was called and found the child dead.

Saward, (B. M. J., 1902, 1, p. 1025.) reports two cases of sudden syncope after the administration of antitoxin. The patients were sisters with clinical symptoms of diphtheria. Both received 1500 units of antitoxin and were removed to the isolation hospital. The one girl was attacked by sudden syncope and died shortly after admission. The sister also became collapsed and recovered only with great difficulty.

In another case, Dogge, (Pediatrics, 1896, 2, p. 12.) gave 10 cc. of antitoxin to a child of 3 years with a very mild attack of diphtheria. He used all precautions of antisepsis. A few minutes after the administration he was hurriedly called by the father, and before he reached the house the child was dead.

Probably the most noted case on record is the unfortunate death of Professor Langerhans's two year old son, who was given 1.2 cc. of serum as a prophylactic measure. For five minutes after the injection, the child showed no symptoms, then the child became restless and cried out in an unusual way. He would often throw himself about, while a fit of coughing would ensue. When this had ceased, the child became limp and pale, and his head sank to one side. At intervals he cried out convulsively. The child had no difficulty in swallowing. A second fit of coughing began about thirty seconds after taking some wine, followed by localized muscular convulsions and frothing at the mouth. The child died in ten minutes. At autopsy no anatomical lesions were found, save some vomitus in respiratory tract.

These untoward effects of the treatment by serum, have led to extensive investigations along these lines. The unanimous opinion of the experimentors and physicians has been that the *benefits of serum therapy far outweigh the danger.* When one remembers the thousands of people receiving serum therapy with beneficial results in their disease, and without evil results from the serum, the ill effects fall very much in the background.

Nevertheless, it becomes the duty of the investigator and the physician to minimize the undesirable qualities of the serum. It has been shown that the antitoxic principle in the serum plays no part in bringing about any of these results. The alien serum alone is responsible for this.

This sensitive condition to serum on the part of the animal body, has been referred to as serum anaphylaxis. Experimentally it has been found that an animal becomes more sensitive to the toxic principle of a serum when it has received a single small sensitizing inoculation twelve days previously. If after the lapse of from twelve to fourteen days after the inoculation of the sensitizing dose of serum the animal be given a second much larger amount of serum, violent symptoms arise and even death may occur. The first or sensitizing dose may be an extremely small one, even as little as 1-100000 of a cc., the second or "acting" dose in guinea-pigs is 5 cc. These inoculations may be made, either subcutaneously or intraperitoneally.

Gay and Southard noted definite lesions to occur in both the fatal and recovery experimental animals. Hæmorrhages of the stomach, cæcum, lungs, spleen, heart and adrenals occurred in order of frequency, and a fatty degeneration of the endothelium of the capillaries was present. They also found that the guinea-pigs which had been sensitized contained a toxic principle in the blood which was capable of sensitizing other guinea-pigs.

Along this line some interesting work has been done by Vaughan and Wheeler. They showed that the proteid molecule of egg white contains a toxic and a non-toxic group. By the injection of the egg-white they were able to sensitize animals for a subsequent dose. The incubation period for this hypersusceptibility was 10 to 12 days. They noted moreover, that the non-toxic group did not produce the sensitiveness to subsequent inoculations.

It is remarkable that herbivorous animals may be sensitized by feeding them with the serum. In this case, larger amounts of the serum must be given than when the serum is inoculated. In man and omnivorous animals a single dose has at times the effect which the two inoculatons have in the guinea-pig and rabbit, and it is further found that the definite incubation period of 12 to 14 days is not constant in the former

Attempts are being made to remove from the serum the toxic principles there present, and some success has been achieved by the removal of the globulins by a precipitation. It is further to be noted that with the present methods of concentration of the serum, smaller amounts of it are necessary to administer the same quantity of the anti-toxic principle than was formerly the case. As the quantity of the "acting" dose plays some part in the character of the symptoms, it is hoped that this method too has decreased to some extent the untoward serum effects.

A full study of the cause of sudden death following the injection of horse serum is given by Rosenau and Anderson in the Hygienic Laboratory Bulletin, No. 29, Washington.

GENERAL PERITONITIS.

BY

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ASSISTED BY

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In choosing the subject of general peritonitis for the paper which our secretary was so kind as to ask me to read before you this winter, I had in view rather the recording of certain beliefs which had gradually come to occupy a more or less fixed place in my mind, and the reporting of results in cases that it has been my lot to treat, than the compilation of statistics or the consideration of the question in its general aspects.

I wish therefore to speak only upon certain sides of the subject: first, the definition of the term; then the bacteriology of the condition and its relation to prognosis; then the treatment, especially the moot points of lavage, and drainage; finally, the results. And I may say that I wish to confine the paper to cases of general peritonitis dependent on appendicitis and typhoid perforation.

Definition.—Peritonitis has been classified from several points of view; thus, according to the character of the exudate, whether dry, or plastic, serous, sero-purulent, or suppurative; again according to the variety of the bacteria supposed to have caused it; and finally, according to the extent of serous surface found to be involved at operation or at post-mortem. In judging the results of operative treatment, the last is the one usually understood; and, indeed, in the present state of our knowledge, it is the only one which we can use for purposes of comparison. Even so, it must be said, comparisons must remain rather unsatisfactory; for we find one man claiming to have cured fifteen out of sixteen cases of general peritonitis, and another thirty-five out of thirty-six, while another confesses that from 60 to 80 per cent. of his cases have died.

It is difficult to understand the reason of so great differences; and I think it is legitimate to enquire, on the one hand, of those whose results are so bad, whether they accept as general peritonitis only such cases as show the presence of pus from liver to pelvis; whether in operating they do anything that in the general opinion is shock-producing, such as evisceration; and whether they take active measures to prevent shock, not to mention many other details of importance. On the other hand, one may enquire of those whose results are so good whether they refuse the chance of operation to moribund patients;

and whether they are quite positive that in all their cases there was pus present in the left side of the abdomen as well as in the right and in the pelvis. Differing conceptions as to definition and a differing selection of cases may make a great difference in mortality figures.

As a matter of fact the tendency has lately become marked, especially among American surgeons, to abandon the term general, and substitute for it two others: spreading or diffuse, and universal, the former meaning an inflammation which is not walled off by adhesions, the second an involvement of the whole cavity from diaphragm to pelvic floor. Now, while all will agree that a universal peritonitis rarely recovers, it is equally true that a comparatively limited peritonitis, say, that in which only the right iliac region and the pelvis are bathed in free pus, ought rarely to die. And yet I strongly suspect that in many of the cases which form the late statistics of Murphy, Blake, Deaver, Ochsner and others, the involvement is no greater than of the right side and pelvis. They are given the name of diffuse or general peritonitis because there is no walling off, and the operative cure goes down as a cure of general peritonitis.

It is therefore of importance to state exactly in the publication of results, what one understands by the term general. At the Royal Victoria Hospital I have learnt, following Dr. Bell, to set down as general peritonitis only such cases as show upon a sufficient exploration the presence of free pus over at least the whole lower half of the abdomen. Anything short of this is not general. The term, it may be admitted, is unsatisfactory, in that it means, strictly speaking, universal. Yet the word has become such a solid citizen of medical terminology that it would be difficult to displace it. And after all, such an objection is somewhat of an academic one, natural enough to the mind of the pure pathologist, but with no strong appeal to the operating surgeon, who, if he can manage to cure a patient in whom he finds pus everywhere below the umbilicus feels justified in saying that he has cured a case of general peritonitis. One condition, however, I think it necessary to set, to wit, that in order to be certain that the peritoneum is so widely involved as this definition implies, two lateral incisions, or one large median incision, are required. When Murphy makes one small incision for the removal of the appendix, avoids handling or even seeing the rest of the intestines, and then sets the case down as one of general peritonitis, we are justified in taking issue with him upon the matter of his definition. It is impossible under such conditions to be quite certain how far the pus extends; the purely clinical diagnosis is, in this matter, too uncertain a one to serve as a sure basis for statistics.

And again, when Blake judges the extent of infection by the amount of pus washed out of a small lateral opening through his irrigating tube, the same objection is surely justified.

Bacteriology.—The bacteriology of peritonitis is a chapter which is still far from being closed. The majority of us have been accustomed to believe that nearly all cases of peritonitis, more especially of appendix-peritonitis, were due to one or both of two micro-organisms, the B. coli, and the streptococcus. We have hardly thought it necessary to remember, if we ever knew, that there were numbers of different strains of the colon bacillus, and greater numbers of different strains of the streptococcus; we have even frequently neglected to add to the latter the qualifying term "pyogenes." We have taken comfort when the pathologist reported to us that the smears from that last case showed B. coli only, for then we said, "He will not die," (although often enough he did die); and we have felt depressed when we were told there were streptococci present; for then we said, "He will die" (although sometimes he did not). Now, I do not desire to contradict flatly this general impression, because sometimes it seems to justify its existence. But, the question is, have we come to any sure ground in our knowledge of the bacteriology of peritonitis?

As a partial answer to this question, certain observations, as I believe, may justifiably be made.

First:—A large part of the published results of the bacteriological examination of peritonitis cases is untrustworthy, because of faulty or insufficient technique. I need not here go into the technical details; and will only say that in most instances, so far as one can gather from the reports, no plating out was done, no examination as to motility; often, indeed, no differentiating into acid and alkali-producers. I know, that in the hurried routine of hospital work,—and I am not here speaking of our own hospitals, although we too in the past have sinned,—many times a report of bacillus coli and of staphylococci or streptococci must have been sent in, based upon nothing more than a smear stained by Gram. A stumpy Gram-negative bacillus was B. coli; a Gram-positive coccus was a staphylococcus unless it showed chains, when it was a streptococcus. Such reports are worse than valueless; they are misleading. We know that the range of the intestinal bacteria is very great. Ford isolated and classified over fifty varieties. In smears obtained from an appendix abscess I used constantly to discover, in so far as morphology and staining reactions carry us, a great many more forms than would grow out on culture media. The question in my mind always was whether some of the forms which refused to grow on aerobic media were nevertheless pathogenic.

If, however, we leave aside for the moment the question of pathogenicity and consider only that of the mere occurrence of bacteria in peritonitis we may say briefly that the few reliable observers agree that the bacillus coli is by far the most frequent organism to be recovered from the peritoneal exudate; that the staphylococcus albus comes perhaps next in frequency, less often the aureus; that mixtures of the bacillus coli and staphylococci are also very frequent; and that pure streptococci are quite rare. There is a general agreement concerning the effect of a pure streptococcus infection. It is extremely apt to be fatal. This remark does not apply to cases in which the streptococcus is found associated with the others. There is considerable disagreement, however, concerning the rôle of B. coli. Those who have done really good work upon the subject may be counted upon the fingers of one hand. Tavel and Lanz in 1893, and again in 1904; Veillon and Zuber in 1898; Hewetson, Harvey Cushing, E. M. Corner, and finally Dudgeon and Sargeant in 1905. The first named considered the bacillus coli as of no greater pathogenic importance than other intestinal bacteria. On the other hand, Laruelle and Malvoz considered it really the only pathogenic agent in perforative peritonitis. Cushing stated his belief that the bacillus coli, being more in evidence, was frequently held responsible for peritoneal infection due to a more virulent but culturally less vigorous organism; in other words, that the frequency of the bacillus coli in cultures was due to its crowding out the streptococcus which is more likely to be the essential cause of the peritonitis. This opinion is the most popular one at the present time. Veillon and Zuber believed that these aerobic bacteria had comparatively little to do with any serious case of perforative appendicitis, the real cause of which resided in the anærobes of the colon. Hewetson failed to find anærobic bacteria. Corner supported Cushing's view. Finally, Dudgeon and Sargeant, whose work seems to be by all odds the most extensive and careful yet published, declare their belief that the bacillus coli is responsible for the majority of the deaths, and that the streptococcus and the bacillus pyocyaneus, while rather more fatal than the bacillus coli, are comparatively rare. They also allotted a considerable share of importance to the staphylococcus albus, which they found very frequently in combination with the bacillus coli. They believed it to be the first to pass out through the intestinal wall, and they attributed to it a certain protective action against the subsequent inroads of the bacillus coli.

It is thus seen that there exists very great disagreement among competent observers as to the essential pathogenic organisms concerned and as to the pathogenicity of each.

Some years ago I had the opportunity of examining a considerable number of cases of local and general peritonitis bacteriologically, and I endeavoured to do so with more than ordinary care. As to the actual occurrence of bacteria in the pus my results corresponded approximately with those of other observers, save that I was able to find as a rule by plating direct from the pus a larger number than others had found of bacterial species in any given case. I found as many as five different varieties in one case, and frequently three. I need not repeat, in this place, the bacteriological details, but would only say that many of them were less well known varieties, described in Ford's monograph upon intestinal bacteria. There were to be found both alkali and acid-producers. Inasmuch as many of these possess pathogenic properties it seemed to me then, and it still seems to me, that it is practically impossible to say that one or the other of these bacteria is pathogenic and the others not. Nobody dreams of asserting that some less well known organism of the Petruschky, or the Eisenberg group is the cause of peritonitis following appendicitis; still I have found such not infrequently in association with the bacillus coli and others, and strictly speaking, who can say that they were not, if not in whole, at least in part, the cause of the peritonitis in question? Among five organisms isolated which of them is the cause of the disease? Another consideration which adds difficulty to the subject is this, that if there are different strains of bacillus coli, of the staphylococcus, and of the streptococcus varying from each other not only in cultural characteristics but in their pathogenic effect, we must be unaware of the pathogenic power of any particular organism that we may happen to isolate. Suppose we endeavour to ascertain its pathogenicity by animal experimentation, we are met with a further difficulty in the fact that an organism which is pathogenic for one animal may not be so for another or for man, while the contrary is also true. Further, we know also that the virulence of any one strain, say of bacillus coli, varies under varying conditions. A bacillus which is nearly harmless in the normal intestine may become, under conditions of intestinal stasis, or symbiosis, extremely virulent. I do not wish to exaggerate the difficulties, but I maintain that we are far from being in a position to speak with accuracy upon this aspect of the subject. As for me, I am agnostic. I do not know. When we come to consider all these difficulties it becomes comprehensible that the prognosis based upon the bacteria found in the exudate must rest upon very insecure grounds.

One further remark as to this habit of establishing the prognosis from the bacteriology,—the prognosis depends enormously upon the stage of the disease, the resistance of the individual and the character of the

operation. For instance, Dudgeon and Sargeant are strongly impressed with the great pathogenic power of bacillus coli, but when we look into their results we find that their cases of general peritonitis showed a mortality of 70 to 80 per cent., probably because the majority of them were treated by large incisions with evisceration and extensive lavage. Many of them we find died of shock, probably on account of the evisceration, and the authors acknowledge that such heroic measures counted to a considerable extent for the high mortality. The death, which they ascribed to the presence of the bacillus coli was more likely due to evisceration, and if it had been operated upon by, let us say, Murphy, would probably have got better.

Thus we may conclude that a prognosis based upon the nature of the bacteria present must be dubious because unjustified by accurate knowledge. As a matter of fact, clinical considerations are far more reliable, and, consciously or unconsciously, however much belief we may place in the report of the pathologist, we judge the outlook for our case upon his clinical condition. On the other hand, the extent of the involvement of the peritoneum, while unsatisfactory upon the whole, offers much more reliable ground upon which to base a prognosis. In an analysis of seventy-three cases of appendicitis which I made in the year 1899, I found a mortality of the unperforated cases to be nil, that of the localized abscesses also nil, that of general peritonitis as defined above, 90 per cent. That year showed an excessively high mortality, but the yearly average since then has varied between 40 and 70 per cent. In other words, when the lower half at least of the peritoneal cavity is bathed in free pus the outlook becomes very dubious. Where the pus is localized, even though the abscess be very large, the outlook is enormously improved.

I would suggest, therefore, that for a basis of comparison the extent of involvement of the peritoneal cavity is for the time being the most reliable. No doubt we have need of a very large series of statistics which will take into account the various clinical conditions of cases, such as the lapse of time from perforation to operation, the age of the patient and various other things, before we can advance materially in the refinements of prognosis in this condition.

Treatment.—The treatment of general peritonitis should be based upon an accurate knowledge of the mode of action of the attacking organism and the mode of defence of the organism attacked. In these respects we have made considerable advances in recent years.

As to the means of offence, we now know that the ordinary bacteria to which we ascribe pathogenic action in peritonitis do harm mainly

by their endotoxins; they do not liberate toxin until they die; to kill them and leave them in the peritoneal cavity is in a sense dangerous. But, in fact, the bacillus coli and allied organisms kill, probably by a bacteræmia, an excessive invasion of the blood current, either rapid or gradual, with liberation of an overdose of endotoxin in the blood as a result of the bactericidal action of the latter. Lately, Peiser of Breslau, and Buxton of New York, have demonstrated that the greater part of bacterial absorption, when the peritoneum is suddenly flooded experimentally by a mass of bacteria, occurs within the first hour or two; that its course is through the diaphragm, the anterior mediastinal glands, and so into the blood current. Following this Peiser showed that there was a period of greatly-slowed bacterial absorption. This corresponds with the period of reaction on the part of the peritoneum, the exudation of plasma and deposition of fibrin, with outpouring of leucocytes. Buxton regards the early deaths after infection with bacillus coli or streptococcus as being probably due to the rapid early invasion of bacteria, with their subsequent proliferation in the blood and organs. No doubt the organisms are still slowly absorbed from the peritoneum following the early rapid rush into the circulation; but they are absorbed in reasonable doses. In this connection an experiment of Peiser's was very remarkable. He found that if he disturbed the process of slowed absorption by injecting salt solution in small amounts into the peritoneal cavity of his animals, this disturbance was sufficient to cause death; in some way it evidently promoted absorption of bacteria. The controls lived. The exact mode of disturbance was unknown; but probably it interfered with the endothelial and phagocytic defence of the peritoneum.

The means of defence have been indicated partly. There can be no doubt but that the main part of the battle takes place in the peritoneal cavity. First, the peritoneal exudate, which is the response to infection, has been proved to be bactericidal to a certain degree. I believe that, in addition, the exudate of leucocytes and fibrin is mechanically a hindrance to the absorption of bacteria. But more important than this is the phagocytic power of the endothelium and the exuded leucocytes. We are too apt to forget Lennander's dictum that "it is the infection that kills; the peritonitis is what saves." The peritoneal reaction therefore, is what we must be thankful for. The most fatal cases are those in which this reaction is practically absent.

It has often been a matter of surprise to me to find a very widespread distribution of the infection, evidenced by sero-pus, in what was evidently a very short time after perforation. The explanation of this

may be found probably in an experiment of Peiser's; injecting a small amount of a bacillus coli culture into the peritoneal cavity, he found it distributed to the remotest recesses of the abdomen within five minutes. This by the way. When therefore we come to consider the question of operation in general peritonitis, we have, I think, to keep certain principles in mind. In a general way we must first remove the cause; next, avoid helping the bacteria present to a quicker absorption; and third, we must not hinder the organism in its struggle against bacteria; on the contrary, we must help it if we can. I do not wish in this place to discuss the various operative procedures of well-known surgeons, but will set forth briefly the principles of operation which appeal to myself.

1. The removal of the cause must be carried out in the shortest possible time, because prolonged ether and much handling lessen the patient's resisting power. This is self-evident.

2. Irrigation of the cavity is certainly to be done when there are gross intestinal contents free in the cavity. The sero-pus, which is the expression of a saving peritonitis, as well as of a bacterial infection, and which consists only of exuded blood-plasma turbid with numerous phagocytic cells, and with bacteria, many of which are already killed, may or may not be washed or mopped away. I do not think that in the present state of our knowledge we are able to determine the question of irrigation definitely. Being left, the endotoxins liberated by the destroyed bacteria may be of danger to the organism, or the bacteria still unkilled may overcome the phagocytes and go on to produce bacteræmia or local abscesses. That irrigation will remove a large number of these bacteria and at the same time the bactericidal and phagocytic fluid in which they are held, is, I think, self-evident. That it may also wash off the microscopical layer of fibrin and the degenerated endothelium of the bowel serosa, and in this way open paths for very rapid absorption of the bacteria which remain is also possible; although, to my mind, not at all so certain as some maintain. Lavage respects neither friend nor foe. I believe that it does harm at times and good at times. Buxton suggests that rapid death after irrigation as we see it occasionally, is due to very rapid absorption of bacteria, and Peiser has brought experimental evidence which points more or less to the same conclusion. The clinical experience of modern surgeons, who irrigate without evisceration or much handling, does not, I believe, confirm this belief, rather the contrary. I am speaking now of those cases in which the intestines are bathed in a thin sero-pus which is quite without localizing tendency. Murphy and Blake are perhaps the two who claim the great-

est percentage of recoveries. One irrigates, the other does not. It really does not seem to make so much difference as experimental work would indicate. The charge that irrigation spreads infection is to my mind unfounded. At least, it need not occur. For my part I believe from experience that if one makes moderately free incisions, and holds them well open, and if one in addition elevates the head of the table, the upper half of the abdomen will never be reached by the fluid. In a large number of cases, combining my own and others, I do not think I have seen a spread of the infection in this way. On the contrary, when patients die, after irrigation, we nearly always find it is not from a spread of the peritonitis, but from the infection already absorbed.

Let us suppose then that we have removed the cause, and that we have either washed or not washed, mopped dry or left the sero-pus in as a supposed element of protection, what are we to do about drainage?

It is in regard to this point that I have come to hold more or less radical opinions. In the last four or five cases, I may say at once, I have mopped the cavity dry, with or without lavage, without caring much whether I got it quite dry or not; and then closed without drainage. These cases have done uniformly well. The reasons for this procedure I may set forth in a series of propositions; and, to provoke discussion I will make these absolute, realizing, however, that they must suffer exceptions.

1. Having removed the cause and, if you like, the serous fluid holding probably dead bacteria in suspension, what need is there for drainage? It must be understood that only such cases are meant as show no material amount of lymphous exudate in patches, nor any foci of necrotic material. The live bacteria may go on, it might be urged, to cause infection and abscess. This is occasionally the case, although experience proves that the peritoneum in nearly all cases is fully able to dispose of what is left. But, in any case, what do we see clinically, when a drain is put in? The drain yields after the first few hours, being exhausted, a drachm or so of clear serum, which comes from the immediate neighbourhood, from the canal formed by adherent bowel. What becomes of the general infection in the rest of the abdomen? It is overcome without a sign. If the case dies, what do we find at post mortem? The peritonitis cured, and the cause of death septicæmia; or else, if the patient live for a number of days after operation, localized abscesses here and there through the abdomen, with no tendency to break into the drainage tract. One exception must be made. Where there is any

area of necrotic tissue left in the cavity, a drain must be placed to that point, because it will certainly go on to frank suppuration. It must be made extra peritoneal. When the appendix is removed, and the stump turned in, and there is no infectively infiltrated tissue in cœcum or omentum left, or in the pelvis, then a drain is not required. When there has been local abscess of some days standing, it must of course be drained; or where the general peritonitis is of some days standing and has resulted in pocketing of pus, these pockets must be drained. But with free pus and no adhesions, drainage is unnecessary.

2. I will go further, and say that drainage of the general peritoneal cavity is impossible. Drains do not drain. To this conviction I have come both from clinical and experimental work. Often and often has it been my duty to exhaust a tube drain in the pelvis. In my experience it was impossible, after the first three or four hours, during which one got two or three drachms of turbid serum, the remains of the irrigation saline, to exhaust more than a drachm of fluid; and this was clear serum at first, evidently the reactionary fluid from the surrounding bowel and omentum which, plastering itself against the drain, walled it off in a very short time. If gauze were used as a drain the walling off was all the quicker and greater.

In a long series of experiments upon the production and prevention of adhesions, I have found that there is no surer agency to provoke walling off than gauze. Yates of Chicago a year or so ago proved this fact experimentally to a superfluity. The relative encapsulation of the drain, he says, is immediate; the absolute encapsulation occurs in less than six hours. In one case of mine done two years ago in which I had inserted a pelvic drain, there occurred a further peritonitis with free fluid. This fluid had entirely failed to find its way into the pelvis and be drained. Drainage was an entire failure. Therefore drainage of the general peritonitis cavity is impossible, at any rate after the first few hours.

Suppose, however, that infection should persist and abscesses be found, will a drain then serve its purpose? I believe not. If localized abscesses develop, in the majority of cases they are not situated where the drain is, and do not evacuate themselves into the drain.

3. I will go further and say that drainage in these cases may do harm. They excite the adhesion of bowel to bowel or omentum; they actively provoke adhesions.

Inasmuch as the adhesions which are apt to follow a peritonitis play a large part in its mortality, I would like to make here a short digression, and refer briefly to the pathological histology of peritonitis, insofar

as it concerns the development of adhesions. Briefly then, the stimulus from bacterial infection causes a swelling, and probably also exfoliation, with ultimate degeneration of the endothelial cells. With this there occurs an outpouring of the blood plasma from vessels in the sub-peritoneal alveolar tissue together with an emigration of leucocytes. By a process of clotting the layer of fibrin is soon deposited upon the serosal surface, and, as the result of its sticky qualities, we get shortly an agglutination of neighbouring coils of bowel, a condition which is favoured by the reflex paralysis of peristalsis so frequently found in peritoneal infection. If adhesions are to occur, these fibrinous layers must become organized, and I believe that the degree of organization goes more or less parallel with the destruction of the endothelial layer of cells and the rapidity of its repair. Where the infection is light, the endothelium not much destroyed, and repair rapid, the fibrin is reabsorbed before organization can take place. In such cases the early fibrinous adhesions may disappear completely. On the other hand, if the infection be severe, the endothelium will largely be destroyed, exposing the sub-serous connective tissue; under such conditions organization is more apt to occur strongly with the result of firm adhesions which will never disappear. Sero-serous adhesions are light, as Greig Smith long ago pointed out; sero-fibrous, or fibro-fibrous adhesions are strong.

As a matter of clinical experience peritoneal infection will frequently not destroy endothelium to such an extent as to cause permanent adhesions; witness the cases in which re-laparotomy after general peritonitis shows the non-formation of adhesions such as one would have expected, or the confining of these adhesions to the area drained by tube or gauze, the rest of the cavity being free, although the peritonitis had been general. And again, those cases in which adhesions seen at one operation are found to have disappeared at a subsequent one.

Now, the adhesions caused by a foreign body are apt to be permanent; this I have had occasion to observe in the series of experiments mentioned in which my endeavour was to produce adhesions. In these experiments, when adhesions had been caused by sterile gauze, they were never found to have disappeared or even to have been modified by the lapse even of four or five months, and clinically we see the same thing. Where we have occasion to reopen a patient for an obstruction which has developed as the result of a general peritonitis which had been overcome years previously, we frequently find that the peritoneum is free of adhesions save in the neighbourhood of the old operation wound which had been drained by tube or gauze. I mention this because in my opinion it has a strong bearing upon the advisability of using drains in the peritoneum.

The danger of post-operative obstruction is no slight one. Of forty-seven cases of this condition observed in the Royal Victoria Hospital during the last ten years, more than one-third died.

Therefore, I conclude that the insertion of a drain in cases of general peritonitis, where the cause has been removed and the abdomen cleaned, is first, unnecessary; second, useless; and third, may be harmful.

It remains in a few words to state the results of my own operations for this condition. The principles above enunciated have been applied only in the last four or five cases, all of which have recovered. Twelve cases have been operated upon of which two were typhoid perforations. Both these, by the way, recovered. Of the 12, 3 have died—a recovery percentage of 75, and a mortality of 25 per cent. I am aware that such a small series cannot carry very great weight; but I believe that it indicates at least the direction in which progress is tending. I may add that of the three deaths one occurred after a week; at post mortem the peritoneum was found to have recovered to a very large extent, and this death was properly due to an acute miliary tuberculosis with a double tuberculous pneumonia and acute bronchitis.

I believe very strongly in the extrinsic aids, particularly enemata of saline, every two hours, or intravenous injections. The latter has seemed on more than one occasion to be the essential factor in saving life. I have made somewhat frequent use of eserine, employing the salicylate in doses of 1-60 to 1-40 of a grain; and it has nearly always yielded very definite results in the way of stimulating intestinal movements. While there may be some reason for objecting to early moving of the intestinal coils after operation for the less severe cases of general peritonitis, there can hardly be any doubt as to its necessity in bad instances of intestinal paresis, where advancing distension and persistent vomiting after the second day threaten a fatal issue. Such cases may sometimes be saved by an enterostomy; but also, I believe, by bold doses of eserine without the enterostomy.

The Fowler position I have used in a certain proportion of these cases, with one or two tubes to the pelvis. In these I could not see that more fluid was drained than when the patients were recumbent; in neither case did the drains drain. Nevertheless, I have continued to elevate considerably the head of the bed, because of the greater evident comfort to the patient; the diaphragm is relieved of pressure from distended bowel, and the heart and lungs are given freer play. Experiment has shown moreover, that the normally rapid flow of the peritoneal effusion to the diaphragm is retarded somewhat by the sitting posture, though by no means prevented, and the Fowler position is therefore an added aid to the prevention of toxic absorption.

A CASE OF MIDDLE MENINGEAL HÆMORRHAGE, WITH OPERATION AND RECOVERY.

BY

J. GUY W. JOHNSON, M.A., M.D.,

Cumpas, Mexico.

The patient was a little Mexican (three-quarter Indian) boy, about 5 or 6 years old. While playing with his younger brother, he tripped and fell, hitting the left side of his head. He got up, thinking nothing of it, and went on playing. About ten minutes later he came to his mother and complained of slight headache and not feeling well. The mother states that he became drowsy and vomited once, shortly after which he began to have convulsions, regaining consciousness between the attacks. She did not notice whether the convulsions were limited to one side or not. I saw him about an hour after the accident.

When first seen, the child was drowsy and did not answer when spoken to. On examination I could find no sign of a bruise anywhere. He moved both legs and arms equally. Babinski's sign was present on the right side, but not on the left. The right knee-jerk was exaggerated. Temperature 98.4°, pulse 100, regular, of good volume and tension. As the boy was having severe convulsions, which were entirely limited to the right side, beginning around the mouth and passing to the arm and then to the leg, I chloroformed him. The convulsions were of a clonic character. As soon as I would stop the chloroform he would go into another convulsion. Whilst under the influence of the anæsthetic I washed out his stomach and rectum. After about an hour and a half of intermittent chloroforming, the convulsions did not reappear; but the boy did not regain consciousness. On examining him, I was surprised to find that the right side of the body was flaccid and paralyzed, although he moved both his left arm and leg freely, when they were pricked with a pin. There was now complete absence of all reflexes on the right side, those on the left side seemed to be normal.

I made a diagnosis of rupture of the middle meningeal artery, and advised immediate operation. After considerable delay the parents consented. I commenced the operation about six hours after the accident; the patient was now comatose and required very little anæsthetic. His pulse was 105. I made a horseshoe-shaped incision, with its base downwards, on the left side of the head. Beginning the incision one-half an inch behind the outer angle of the eye, extending it up to the level of the parietal eminence and ending about one-half an inch

behind the ear, the flap thus having a base of one and one-half inches and a greatest diameter about two and one-half inches. I then raised the flap from the bone, having considerable difficulty with the temporal muscle. I then covered the flap with hot cloths, and trephined the skull, the center of the trephine being midway between the outer angle of the eye and the ear, and about one-half an inch above the line joining these two points. On removing the button of bone a good deal of blood rushed out. The dura mater had been separated from the skull for some distance all around the opening. There was very little attempt at clotting of the blood. On washing out the wound with 1 in 12000 bichloride, blood could be seen to come down from above in small gushes, showing that the bleeding was still going on. I could see no sign of fracture, although I probed around in search of one. The middle meningeal artery could be seen lying in the bottom of the trephine opening. With a needle I placed a catgut ligature around the artery, and on tieing it the blood immediately stopped flowing into the wound. After again washing out the wound I closed it with interrupted silk-worm sutures, leaving a small gauze drain in the lower anterior part of the wound.

The boy came out of the influence of the chloroform very slowly, but without vomiting. He moved his right leg very slightly. Next day when I went to see him I found him moving round in bed, using both arms and legs freely. The reflexes were equal on both sides. His speech was clear. The temperature was 99°, the pulse 90.

I removed the gauze drain and tightened the stitches that I had left in place. There was never any rise in temperature or pulse, and the boy made an uninterrupted recovery, the stitches being removed on the tenth day. I saw the boy about a month later and he was playing as if nothing had ever happened to him. The opening in the bone seems to be growing smaller. The points that I should like to draw attention to are the following:—

(1) The very slight injury, not even causing a bruise.
(2) The rapid onset of symptoms, especially the paralysis.
(3 The rapid recovery from the paralysis after operation.

W. H. Park experimented with typhoid bacilli in ice, using 21 strains. Not one bacillus was living after twenty-two weeks freezing, and less than 1 per cent. of the bacilli after five weeks, six of the cultures having been destroyed entirely. At the end of four weeks storage the ice is as free from typhoid bacilli as if the water had been subjected to sand filtration." (*Jour. A. M. A.*, August 31).

THE

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THE NEW MEDICAL COLLEGE AT McGILL.

The board of assessors have finally declared in favour of the plans submitted by Messrs. Brown and Vallance, from among eight competing firms: the plans have lately been on view at the Redpath Library, and show a wide variation of design. The accepted plans are not in any sense final, but they represent a satisfactory grasp of the general idea of the Governors and the Faculty, both with regard to the building in itself, and its adaptation to the site chosen on Pine avenue, where the land has a slope in two directions, and presents somewhat of the same problem as was offered by the site on which now stands the Royal Victoria Hospital. The general estimate of cost is in the neighbourhood of half a million dollars. The central block will face the campus, and parallel wings will extend towards Pine avenue, one of these being on University street and

the other on or close to Carlton Road. There are many details of distribution of space yet to be decided, but presumably the plans indicate what will be the final idea as to external structure.

ARE SERUMS DANGEROUS?

In the columns of this number will be found some interesting material relative to the occurrence of death following the use of protective serum derived from equine sources; we know quite well the risk that there is, lest some zealous layman seize upon this as campaign-material against the use of antitoxic serums, for there are always protesting people who think that to be in the minority and to shriek are two arguments in favour of the faith they uphold. Disregarding this danger, we publish the dismal side of the story, in the interests of knowledge, and we beg to point out the relative importance or lack of importance of these lethal cases. Stated as a percentage they are so small as to be no argument against the use of serum: to argue against the use of serum on the grounds of this danger would be much less reasonable that it would be to discard anæsthesia because of its dangers. In one, as in the other, the choice of the lesser of two evils must win. This refers to its use as a curative agent; but in its prophylactic usage, the case will bear further argument. In the first place, it may be said that the prophylactic effect of diphtheria serum, for example, is not absolutely proven; in the second place, if the "sensitizing dose" be a correct idea, the use of antitoxin as a prophylactic measure, in a small dose, is merely sensitizing the patient against a future dose, if the disease be contracted, in return for the benefit of a probably less severe attack. Even here, however, the case against serum is not clear, because many hundreds of thousands of immunizing doses have been given and many hundreds of these patients have afterwards been given serum, without untoward results. To sum up, it may be dangerous to give an immunizing dose, but there is already vast practical experience that it is not.

As knowledge of the subject advanced, it became evident that the dangerous element was not the antitoxic principle, but the alien serum, and, as is pointed out in the papers here referred to, the reduction of bulk of serum thus reduces the danger, and in this regard, the most concentrated serums are the best because they provide a maximum of antitoxic principle with a minimum of alien serum; and further chemical studies have been made and are yet being made with a view to neutralizing the deleterious effects of the alien serum.

The physician, more than most others, has to pick his way among dangers, running near to the lesser ones that he may avoid the greater;

and because perfect remedies are not easy to find, and discoveries are slow to make, and time is required for the development of knowledge, we, of the present day, have often to grope in the twilight of insufficient knowledge as had our forefathers: and to sit down and do nothing, as our enemies the anti-vaccinationists and other shriekers would have us do, is to arrive at no end. We must, therefore, choose carefully and conscientiously what we judge to be the least dangerous course, and pursue it, knowing that he who will not walk on the sidewalk because he may be struck by a falling signboard, will probably be run over by a dray or killed by a street-car, if he take to the roadway.

THE GLORIFICATION OF THE UNESSENTIAL.

From the daily papers one may extract a few gleams of medical sunshine at times, and some day there will arise a collector of the comical in the shape of "germs of thoughtlessness" like this latest upon the eating of roller-process flour as a cause of appendicitis. Not long ago our editorial pen was stirred to activity by the exploitation of a certain intestinal worm as the cause of appendicitis, and we are loathe to launch out fully upon this subject again, but the idea of the rollers shedding their metal almost tempts us—and the time cannot be far off when we shall eat roller flour no longer as a food, but as a tonic; steel and quinine! the forests of Brazil and the wheat-lands of our north-west hand-in-hand to produce medicine, and flour-mill stocks dropping with panicky speed. When may we expect to see advertisements setting forth, "Our flour contains the highest percentage of iron per ton of any on the market," or, "Try our Steel-Flakes"?

This kind of etiology for diseases has not been sufficiently worked out, and is gone at in a method far too hap-hazard by the majority of those who employ it. The real method is this—take a dictionary, for example, the Century. Pages one and two are taken up with "a" in all its variations, and are obviously useless, but aardvark comes next, which suggests "gastritis caused by ingestion of aardvark meat," followed by "aba," which at once suggests "Aba as a protective for the skin of nephritics": not to leave this interesting vowel we see chances for the following: "Ecchymoses as a result of ablewhackets," "The abomasum and its pyloric relations," "Absinthe as an etiological factor in mumps," or, should the writer prefer "Absinthe as a cure of whooping cough" (it is one of the few remedies we have not seen suggested), to say nothing of "Abstergents, their use and abuse." Given a fair chance, much can be derived from a dictionary, and if all the nouns

be selected, a fair assemblage of articles can be found which might be eaten or drunk: why not each one of them as a cause of appendicitis? or any other disease?

Truly it seems, sometimes, as if the only claim that certain theories have upon attention, is that they have not been made before. In a few million years, all the permutations and convictions will have been exhausted, and the theorizer of that day will weep for new worlds to invade, if not to conquer; but one's patience even now is almost exhausted, and long before the supply is at an end, there will be a tax on the giving out of useless and unbased theories, and that will probably put a stop to it. The tendency to use the wrong end of the telescope on disease and its problems is widespread in the laboratories, too; there is far too much even in Germany of the "Estimation of the glycogen-content of the orbicularis muscle"—kind-of-arbeit. It may be said that all knowledge is of some use, but some particles of knowledge are so minute that they are not worth the time and trouble of any labouring man, who can use a spade, and sees mountains of ignorance on every side at which he can at least do his share of digging.

Therefore, we are bold enough to say that the view that appendicitis is caused by flour and the particles of metal therein is a foolish overlooking of many pertinent facts, and pouncing upon one small, unessential, even doubtful one.

The causation of appendicitis, at least, now rests upon a firm basis; it is explained in a reasonable way to the satisfaction of most of us, and even if the explanation be not absolutely true, it is sufficiently in keeping with the knowledge we now possess of disease in general that it may well be allowed to rest there, so long as our entire way of thinking upon these problems does not change. We seem to know, in a modest way, even now, a few general laws of disease, and we prefer to consider the etiology of appendicitis in the light of these general laws, and the man who attempts to bring in a striking exception to these laws may be ahead of his age, like the giants of science, but the chances are heavily against him. We shudder to think of what may be the next theory on appendicitis to be dealt with in these columns.

EXAMINATIONS FOR INSURANCE.

After the investigation by authority of the Legislature into the methods of Life Insurance Companies doing business in New York certain restrictions were made upon the expense which might be incurred in procuring new business. To comply with the law every item bearing

upon the first year's cost was scrutinized. Amongst them was the fee paid to medical examiners, which had been five dollars. The companies were obliged under the law to reduce the fee to three dollars for all examinations for policies of less than three thousand dollars.

Some opposition was made to the reduction, but in the main the examiners accepted the new schedule and continued to do their work faithfully. In some localities there was an organized opposition. Examiners were obliged to refuse the reduced fee and the companies restrained by the law were compelled to abandon those fields.

As the result of a year's trial one company, at least, the Mutual Life of New York, has found that all the economies practised have brought the expenses well within the limit. For next year it finds it possible to make a slightly enlarged expenditure. The medical examiners were the first to receive consideration and the fee has been increased to five dollars. At the time we counselled forbearance, on the ground that the companies were merely carrying out the law, and that the interests of the medical examiners were identical with those of the companies. Now, it is a pleasure to find that this confidence has been justified.

Reviews and Notices of Books.

DISEASES OF THE STOMACH. By DR. I. BOAS, Specialist in Gastro-enteric Disease in Berlin, Germany. The sole authorized English American edition from the latest German edition. By Albert Bernheim, M.D. (Freiburg, Germany), assistant to the late Dr. D. D. Stewart at the Philadelphia Polyclinic Hospital and Post-graduate School, and Instructor in the Department of Diseases of the Stomach and Intestines, etc., etc. Illustrated with five full-page plates and sixty-five engravings in the text. 730 Royal octavo pages. Extra cloth, $5.50 net. Half morocco, $7.00 net, Sold only by subscription. F. A. Davis Company, publishers, 1914-16 Cherry street, Philadelphia, Pa.

Boas' text-book upon diseases of the stomach scarcely needs an introduction to any one specialllly interested in the subject, as it is well known, having run through five editions in the thirteen years that followed its first appearance in 1890. By the present translations it is available to English readers, as it has been previously available to Russian, Italian and Spanish readers by translation to those languages. Even now a short description of the book's contents may be useful. It opens with forty pages upon the anatomy and physiology of the stomach,

including the chemistry of the secretions of digestion. The consideration of diseases of the stomach begins with a very full description of a proper history to be taken in a stomach case, together with methods of examination of the patient. A novel and not very widely known instrument is the algesimeter of the author, which is a kind of piston which transmits pressure to various sized plates, and registers the amount of pressure required to produce pain. The gastroscope meets with praise, but it is allowed that a high degree of skill is necessary for its proper employment. With the Rontgen-ray examination, are several good skiagrams of bismuth-food at varying times after ingestion.

One hundred and twenty-five pages are devoted to chemical examination of the stomach contents and as might be expected, the editorial additions here are not numerous, as the descriptions are most exhaustive. We notice that the author states that he considers the bacillus long ago described by him, generally known as "Oppler-Boas" bacillus, to be the lactic acid bacillus, and to be considered characteristic only when it overwhelms the field. The plates which illustrate particles of gastric mucosa from personal observation are very interesting, but their very perfection suggests that a very wide experience has been necessary for the finding of such definite pictures. The chapter on examination of the urine and its significance in diseases of the stomach, is rather concerned with suggestions for future advances than with detail of actual useful tests. Of blood tests, the same may be said. Then follow seven chapters, totalling a hundred pages or more, upon general therapeutics. In speaking of diet, Professor Boas says that we are, perhaps, too lavishly governed by the calculation of calories, but says, in italics, that "every physician who treats chronic diseases, yes, any physician at all, should in my opinion possess a precise weighing apparatus!" a dictum which will find many of us wanting in the balances. The statement of exact diet is admittedly a very difficult one for the physician, and the author cautions against printed diet slips which entirely minimise the individuality of the physician's orders. "General prohibitions, (e.g. the well-known advice to eat nothing acid, fat or spicy) are worthless." To begin with, the physician must know the exact amount of which the average healthy stomach can dispose: pages 288 to 292 contain some very useful information in general upon diet in various common derangements. The most common and popular artificial foods are classified, and their ingredients stated. When speaking of rectal feeding. Boas says "Rectal feeding in cases of gastric ulcer should be the last refuge, and only if the diagnosis is absolutely sure." The editor adds after the word "refuge" the interpola-

tion "if operation is denied" which seems to us a real Americanism of 1907. Mineral waters, electrical treatment, and gastric lavage are dealt with, and with regard to the latter, a protest is entered against "aimless experimenting" with it. An appendix on surgical treatment is added after acids, alkalines, stomachics and prepared ferments have been discussed. We agree here with some of the editorial remarks very heartily, for stomach surgery has run a little wild in America of late years: the appendix closes with this comment, "the highest task of therapeutics does not consist in inventing new operations, but in making them dispensable." Part II consists of the consideration of special diseases, gastritis, ulcer, motor insufficiency, displacement, cancer, syphilis, tuberculosis and the neuroses. This is almost half the entire volume, and many times occur points of great interest upon which lack of space prevents our dwelling. Syphilis and tuberculosis are interesting chiefly by their rarity. On the whole, the editor is to be congratulated on bringing this book into the hands of many who will find it of great advantage. Dr. Boas speaks with much personal prejudice (to use the word in its good sense) on many questions, and no one will deny his right to do so: the book gains value thereby: many of his most striking dicta are italicised, and at times the text bears the mark of its verity in its German construction; this again, is by no means a disadvantage, for it adds a personal element that is often entirely missing in a translation. The text is clear, the bibliography is generous, especially, of course, in German lines, and is added at the bottom of the page, and an adequate index is provided.

THE PRACTICE OF OBSTETRICS. By American authors. Edited by CHARLES JEWETT, M.D., Professor of Obstetrics in the Long Island College Hospital, Brooklyn, N.Y. In one handsome octavo volume of 786 pages with 445 engravings in black and colours and 36 full-page coloured plates. Cloth, $5.00 net; leather, $6.00 net; half morocco, $6.50 net. Lea Brothers & Co., New York and Philadelphia.

The third edition of the "Practice of Obstetrics," by American authors, published by Lea Brothers & Co., which has just made its appearance, revised and enlarged, is evidence of the popularity of the work. Works of composite authorship rarely appear in successive editions, hence this is a notable exception.

In this third edition several subjects have been wholly re-written, notably those on "The Changes in the Maternal Organs During Pregnancy," "The Duration of Pregnancy," "The Hygiene and Man-

agement of Pregnancies," "The Anomalies and Diseases of the Foetal Appendages," and "The Diseases of Pregnancy," all of which have been contributed by Dr. W. S. Stone. The section on "Symphysiotomy" has been condensed, and the newer operations of "Hebotomy" and of "Vaginal Cæsarean Section" are described.

On the whole the work, already excellent, has been improved, and the third edition will no doubt prove quite as popular as its predecessors. The work is to be recommended as one of the very best of the recent American obstetrical books.

DISEASES OF THE INTESTINES AND PERITONEUM. By PROFESSOR DR. HERMANN NOTHNAGEL, late Professor of Special Pathology and Therapy, University of Vienna. Edited, with additions, by H. D. ROLLESTON, M.A., M.D., F.R.C.P., Physician to St. George's Hospital, and to the Victoria Hospital for Children, London; Sometime Fellow of St. John's College, Cambridge. Second edition, thoroughly revised. Authorized translation from the German under the editorial supervision of ALFRED STENGEL, M.D., Professor of Clinical Medicine in the University of Pennsylvania. Philadelphia and London: W. B. Saunders Company, 1907. Price, cloth, $5.00 net; sheep or half-morocco, $6.50 net.

This is the second edition of this work, the previous edition having appeared three years ago: the changes introduced in the present volume consist chiefly of additions to the editorial comment, for Dr. Rolleston is a very painstaking and conscientious editor of whatever work he undertakes; and the addenda are brought well up to date.

The book covers so wide a field and contains so much aggregated material that we find it difficult to discuss it at length; in any of the several hundred conditions discussed, the reader will find a fair statement of our present knowledge of the disease dealt with. For example, the chapter on appendicitis represents a vast amount of material and research, and it is well handled when one considers the bulk of it; we are glad to see that in the vexed subject of etiology the author, and perhaps even in a greater degree, the editor, indicates the direction that is safest to take—viz.: that the mechanical effects of lack of drainage, assisted by the irritation of concretions are the most potent factors; that cœcal inflammation may well be the original point of departure, and that the various fanciful sources that have been from time to time exploited, are efficient in a very small number of cases. This chapter is but used as a test-case, to indicate the sanity of view that pervades the entire work; and at the same time to indicate that no reasonable

work of importance upon the subject has been overlooked. This volume does not need our approbation, but we are glad to give it; and we beg to point out that no small share of its success is due to good editorial work, for we can assure any reader who does not already know, that Dr. Rolleston has perhaps as wide a knowledge of American medical literature as is possessed by any English physician; and in his hands, deserving work on this side of the world meets its due.

ATLAS AND EPITOME OF DISEASES OF CHILDREN. By DR. R. HECKER, and DR. J. TRUMPP, of Munich. Edited, with additions, by Isaac A. Abt, M.D., Assistant Professor of the Diseases of Children in Rush Medical College. With 48 coloured plates, 147 black and white illustrations, and 453 pages of text. Philadelphia and London: W. B. Saunders Company, 1907. Cloth, $5.00 net.

This is a good book, and its excellence lies in no small degree in well selected illustrations, so that the 453 pages of text does not make so formidable a mass of reading as one might at first expect. The text is pointed, direct, and appears to be perfectly sound in its general treatment of the subject. Practically all the diseases by which children are afflicted, are taken up and the volume is thus a very good practical adjunct to any practicing physician's library.

There are, it is true, a few illustrations that are unnecessary, but this is out of a large number: the only purpose served by that on page 54, "introduction of a thermometer in the rectum of an infant" seems to be to show that the brass-capped end of the thermometer is not the end to be inserted; and on page 56, in the illustration of "inspection of the oral cavity of a small child" there is at least the virtue that the pleased expression on the child's face shows that, in skilful hands, the process may become as indispensable to babies as a certain much advertised laxative. But speaking without levity, the coloured plates of measles and scarlet fever just miss being the exact thing, although it is doubtless extremely difficult to portray these accurately. On the other hand, tracheotomy, melaena, dyspeptic stools, and the histological plates, to select no others, are worthy of the best praise. The writer sees for the first time the word colicystitis to designate a cystitis in which the infection is bacillus coli. This term should be put out of use at once, for it can lead only to confusion with cholecystitis, and it is a very late second in the field. As in this whole series, the histological plates are excellent, and one could wish there were more of them; although we well recognize that the price of the book is due somewhat to the cost of reproduction of the coloured plates, and their increase would but mean a more costly volume.

Treatment is stated in a succinct way that is very pleasing, and withal, complete. The editor's comments are timely, and useful. It is a pleasure to recommend the book.

THE AMERICAN POCKET MEDICAL DICTIONARY. Edited by W. A. NEWMAN DORLAND, M.D., editor "The American Illustrated Medical Dictionary. Fifth Revised Edition. 32mo of 574 pages. Philadelphia and London. W. B. Saunders Company, 1906. Flexible morocco, gold edges, $1.00 net; thumb indexed, $1.25 net.

The fifth edition of Dr. Dorland's Pocket Medical Dictonary is a very comprehensive little volume of small size and much information. It contains definitions, pronounciations, and a mass of most useful material in tables. Of the latter there are sixty: may we suggest that a useful sixty-first would be a table of the antidotes to the common poisons? The volume will be found very useful.

A LABORATORY MANUAL OF INVERTEBATE ZOOLOGY. By GILMAN A. DREW, Ph.D., Professor of Biology at the University of Maine, with the aid of members of the Zoological Staff of Instructors of the Marine Biological Laboratory, Wood's Holl, Mass., Philadelphia and London. W. B. Saunders Company, 1907. Canadian agents, J. A. Carveth & Co., Toronto, Ont. Price $1.25.

This is essentially a working text book for the laboratory, and as such its medical scope is very limited; it gives dissection directions for the study of type specimens of the different genera, and these it gives in a brief, terse way. Most, or all of the commoner parasites are so dealt with, from their zoological side. The author observes in the preface that while this type method of teaching widely prevails, care has to be taken to keep the student from making everthing conform to type: this observation, we think, has a wide application in many other departments. A useful glossary of terms and an index accompany the text.

Medical News.

CANADIAN MEDICAL ASSOCIATION MEETING.

The following is a partial provisional programme of the meetings to be held on September 11th to 14th, in this city.

Presidential address, Dr. A. McPhedran, Toronto; Address in Medicine, Dr. H. D. Rolleston, London, England; Address in Surgery, Dr. Ingersoll Olmsted, Hamilton, Ont.; Address in Pathology, Dr. J.

George Adami, Montreal, Twins, Double Monsters and some other Topics; Discussion in Medicine, Cerebro-spinal Meningitis, introduced by Dr. J. J. Mackenzie, Toronto; Dr. H. A. Lafleur, Montreal; Dr. A. D. Blackader, Montreal; Discussion in Surgery, Hypertrophy of the Prostate; Etiology and Pathology, Dr. G. E. Armstrong, Montreal; Symptomatology and Diagnosis, Dr. F. N. G. Starr, Toronto; Treatment, non-surgical, Mr. J. H. Cameron, Toronto; Treatment, operative, Dr. James Bell, Montreal; Section of Laboratory Workers, Dr. G. W. Ross, Toronto; Dr. Gibson, Kingston; Dr. Rankin, Montreal, have promised papers reporting work on Opsonins; Dr. J. J. Mackenzie, Toronto, Generalized Blastomycosis; Dr. Campbell Howard, Montreal, A Study of the Eosinophile cells of the blood; Dr. McKee, Montreal, On Retinitis Pigmentosa; Dr. E. C. Dickson, Toronto, On the Significance of the Glomerular Changes in the Kidney; Dr. J. McCrae, Montreal and Dr. Klotz, Montreal, The Necroses in the Liver in Eclampsia and other Diseases; Dr. Tooke, Montreal, On Injuries of the Cornea.

OTHER PAPERS.

Listerism, Dr. A. H. Wright, Toronto.

Seven Hundred and Fifty Abdominal Sections and the Lessons they have taught me, Dr. A. Lapthorn Smith, Montreal.

A Case of Primary Bilateral Mastoiditis, Dr. Perry G. Goldsmith, Toronto.

Gonorrhœal Metastatic affections of the optic nerve and retina, Dr. Gordon Byers, Montreal.

Notes on Tubercle Bacilli Isolated from Fatal Cases of Primary Cervical Tubercular Adenitis, Dr. Duval, Montreal.

Bacteræmia. Its Diagnosis and its Diagnostic and Prognostic Value, Dr. Fraser Gurd, Montreal.

Cancer of the Breast, Dr. George E. Armstrong, Montreal.

Modern Methods in Diagnosis of Tuberculosis of the Kidney, Dr. R. P. Campbell, Montreal.

The Clinical Side of Ectopic Pregnancy, Dr. W. W. Chipman, Montreal.

Danger Signals in Anæsthesia, Dr. Samuel Johnston, Toronto.

Psychology of the Sick Room, Dr. John Hunter, Toronto.

Paresis: Certain Features in Regard to the Etiology and Differential Diagnosis, Dr. John G. Fitzgerald, Toronto.

The Normal Temperature, Dr. R. D. Rudolf, Toronto.

The rights of Children, Dr. C. J. C. O. Hastings, Toronto.

The Defensive Action of Products of Metabolism, Dr. Graham Chambers, Toronto.

Treatment of Neurasthenia, Dr. E. C. Burson, Toronto.

Papers are also expected from the following: Dr. Connell, Kingston; Dr. Keenan, Montreal; Professor Harrison, St. Anne de Bellevue; Dr. A. W. Moody, Winnipeg; Dr. W. F. Hamilton, Montreal; Dr. F. G. Finley, Montreal; Dr. C. F. Martin, Montreal; Dr. Colin Russell, Montreal; Dr. Campbell P. Howard, Montreal; Dr. A. G. Nicholls, Montreal; Dr. Ridley Mackenzie, Montreal; Drs. Lyman and D. A. Shirres, Montreal; Dr. B. W. D. Gillies, Vancouver; Dr. A. H. Gordon, Montreal; and from Drs. Lowrey, H. B. Anderson, H. C. Parsons, W. B. Thistle, R. J. Dwyer and A. R. Gordon, Toronto, and Geo. T. McKeough, Chatham, Ont.

When and How to Resume Normal Feeding in Convalescence from Typhoid Fever, Dr. J. T. Fotheringham, Toronto.

Dr. Maud E. Abbott, Montreal, will present an exhibition of Pathological Specimens from the McGill Medical Museum, illustrating the Circulatory System.

Professor J. J. Mackenzie, Toronto, will also exhibit Pathological Specimens.

Dr. Robert Wilson, Montreal, will give an Exhibition of X-ray plates from the different hospitals.

The Occurrence of Congenital Adhesions in the Left Common Iliac Vein, Dr. J. Playfair McMurrich, Toronto.

Sigmoiditis and Diverticulitis of the Rectum, Dr. D. A. L. Graham, Toronto.

Ample accommodation has been provided in the Arts Building, Peter Redpath Museum, the Physics building and the McGill Union. The Lecture halls are provided with lanterns, and it is hoped papers will be illustrated by this means. Interesting clinical material will be shown at the Royal Victoria Hospital, and Montreal General Hospital on the mornings of the meetings at 8.30.

DR. J. L. TODD'S RETURN.

After several years of valuable work in connection with the Liverpool School of Tropical Medicine, Dr. J. L. Todd, of this city, until recently the Director of the Tropical Research Laboratories at Runcorn, has resolved to return to Montreal.

In the current number of *Canada* the description of a farewell dinner given to him at Liverpool, is reported as follows:

The dinner was given by Sir Alfred L. Jones, president and founder of the Liverpool School of Tropical Medicine. The company included

many distinguished men, including Col. Bruce, Sir Rubert Boyce, F.R.S., Professor Ross, and Professor Carter.

In proposing the health of Dr. Todd, the chairman said they bade a sad farewell to him. Dr. Todd, who had been with them four or five years, did not come to make money, because he spent a great deal more than he got, and had done a great deal more in connection with the school than any one else.

Sir Rubert Boyce supported the toast, remarking that Dr. Todd was a splendid example of an Imperial student. Not only had he given of his time and talents, but also his money. Although Dr. Todd was present, he (Sir Rubert) could not refrain from saying that the doctor had given to their Tropical School something like $1,000 a year, and had risked his life in the interests of the school. He had particularly given money to found the research laboratories at Runcorn, where the best scientific work was carried on under and by Dr. Todd, and where the highest scientists in the world desired to study. It was largely due to the splendid work of Dr. Todd that they had discovered a preventive for that dread malady, sleeping sickness. He trusted that Sir Alfred Jones, with his great capacity for getting over all kinds of difficulties, would find a means of bringing Dr. Todd back again to them from Canada. (Loud applause).

Dr. Todd, who was enthusiastically received on rising to acknowledge the compliments paid to him, was educated at McGill University, where he took the degree of B.A., M.D., and C.M. Coming to Liverpool he, in 1902, took part in the expedition to Senegambia to investigate malaria, and subsequently studied sleeping sickness in the Congo in 1903-1904. Amongst the publications of the Liverpool School of Tropical Medicine, which sent out the expeditions, are many interesting reports from Dr. Todd's pen, notably on malaria and sleeping sickness, as well as tick-fever in the eastern part of the Congo Free State.

CANADIAN MEDICAL ASSOCIATION 40TH ANNUAL MEETING.

Montreal Meeting, 1907—September 11th, 12th, 13th and 14th.

N.B.—Purchase Single First-Class ticket to Montreal, and get at the same time, from ticket agent, Standard Convention Certificate. No certificate required from General Secretary.

When it is remembered that over 800 Canadian physicians registered the first day of the British Medical Association meeting in Toronto last

year, surely it is not too much to expect that at least Three Hundred will journey this year to Montreal to the Fortieth Annual Meeting of their own National Medical organization. If three hundred are present with Standard Convention Certificates all will be returned home free. The Standard Certificate plan prevails in every province, no one requiring any certificate from the General Secretary. This means that all delegates, on purchasing single first-class tickets to Montreal, for themselves, their wives and their daughters (no others) should ask for and get, at the same time, a Standard Convention Certificate, from the ticket agent for each. These, when signed by the General Secretary at the meeting will entitle holder thereof to reduced transportation, which in all cases must be arranged for at Montreal. If three hundred are present holding these certificates, all will be returned home free; one-third fare if fifty are present with certificates. The Canadian Pacific Railway, the Grand Trunk Railway, the Intercolonial Railway, all lines in the Eastern Canadian Passenger Association and the Richelieu and Ontario Navigation Company and Canadian Northern Railway, are included in the transportation arrangements. Delegates from points west of Fort William will be permitted to use the Upper Lake Route, Fort William to Owen Sound, or *vice versa*, on extra payment of $4.25 one way, or $8.50 both ways, when travelling on the Standard Certificate Plan. Passengers going by rail, returning Richelieu and Ontario Navigation Company, or *vice versa*, rate to be one and one-half fare. Tickets will also be honoured via R. and O. Nav. Co, on presentation of rail excursion ticket to the ticket agent at Toronto, or to the purser on board steamer and payment of the following arbitraries, viz.. $6.65, Toronto to Montreal; $3.50, Kingston to Montreal. Those desiring to tour should consult with their local railway agents as to tourist tickets.

DATES OF SALE OF TICKETS, TIME LIMITS, ETC.

Tickets will be on sale in the Eastern Canadian Passenger Association territory—Port Arthur to Halifax—three days before the first day, Sunday not counted a day, and final return limit three days after the last day. From British Columbia points tickets will be sold and certificates issued on September 1st and 2nd, and validated certificates honoured for return tickets up to and including October 9th. Tickets good for continuous passage only in each direction. West of Port Arthur and Fort William, the selling dates are September 5th, 6th, 7th and 8th. From stations west of Winnipeg tickets to be good going *via* trains that will connect with those leaving Winnipeg the

before-mentioned dates; certificates to be honoured at Montreal up to and including October 11th.

PLACE OF MEETING IN MONTREAL.

The meeting place will be the McGill University Buildings. The general meetings will be held in Molson Hall, the Medical Section in the lecture-room of the Redpath Museum, and the Surgical and Pathological Sections in the lecture-rooms of the Arts Building.

CERTIFICATE FEE.

The railway officer at Montreal, when exchanging Standard Convention Certificate for return transportation, will collect from each, for viséing the same, a fee of twenty-five cents.

HOTEL ACCOMMODATION.

Delegates desiring to have hotel or lodgings reserved for them should apply to the Local Secretary, Dr. Ridley Mackenzie, 198 Peel Street, Montreal.

MEMBERSHIP.

The fee for membership is $2.00 and may be paid to the Treasurer, Dr. H. Beaumont Small, Ottawa, at time of registering. For the information of those who will apply for membership for the first time, the same transportation rates apply to them as well, and they are requested to ask for *Application for Membership* forms when registering.

THE SOCIAL SIDE AT MONTREAL.

There is to be a garden party at Terrace Bank through the kindness of Dr. and Mrs. Roddick, a smoking concert in the Victoria Armoury, a reception after the President's Address the first evening, in the Students' Union Building, a drive and luncheon at the Hunt Club for the ladies, golf matches, etc.

CANADIAN MEDICAL PROTECTIVE ASSOCIATION.

During the meeting of the Canadian Medical Association, as usual, the annual meeting of the Canadian Medical Protective Association will take place. Dr. R. W. Powell, Ottawa, the President of the C. M. P. A., will deliver the annual address and present the annual report.

MILITARY SURGEONS.

There will also be a meeting of Canadian Military Surgeons, an organization which the Director-General of the Army Medical Service, Lieutenant-Colonel Carleton Jones, M.D., is promoting.

Full discussion will take place on the report of the Special Committee on Re-organization. For this reason alone there should be a large and representative delegation from each province.

ADDITIONAL INFORMATION.

Additional information of a local character may be obtained from the Local Secretary, Dr. Ridley Mackenzie, 192 Peel Street, Montreal; any general information from the General Secretary, Dr. George Elliott, 203 Beverley Street, Toronto.

W. Pepper and V. Nisbet, Philadelphia (*Journal A. M. A.*, August 3), gives the history of a rather puzzling case of hæmorrhagic diseases in which direct arteriovenous transfusion was performed on two separate occasions, of which the second was followed by a fatal result. On the first occasion the blood (wife's) was allowed to transfuse for a little more than an hour and a half, and the patient, who had apparently been rapidly sinking, showed marked improvement. There was a slight suggestion of hemolysis in discoloration and uroblin, in the urine, but this had nearly disappeared when the second arteriovenous anastomosis was made with another person (brother-in-law) two days later. After the second transfusion the condition changed rapidly for the worse, there was marked evidence of hemolysis, bloody urine, jaundice, high temperature, etc., the patient failed rapidly and died within five days. Full details of the blood count are given. The impression is given that possibly the first transfusion may in some manner have caused the formation of hemolytic bodies that, after the second transfusion, found abundance of vulnerable corpuscles, on which to work. The authors do not venture any positive opinion, however, as to the rôle of the second transfusions, but remark that no bad effects were noticed by Crile, in a second transfusion from two brothers in one case, and that laboratory studies undertaken since their experiences have thrown no light on the matter. They recall the fact in the history of the case that an injection of diphtheria antitoxin had been given the patient some days prior to the first transfusion, since the studies of Theobald Smith, Rosenau, and others have indicated possibilities of danger in secondary injections. The conditions in this case, however, were not parallel. While Crile and others have shown the probable safety of transfusion in post-hæmorrhagic conditions, the authors think this experience indicates a danger in certain pathologic conditions and suggests caution until we are in possession of greater knowledge. Nevertheless the possibility of benefit, they say, may justify transfusion in apparently desperate cases.

The August 17 issue of *The Journal A. M. A.* is the annual educational number which gives a comprehensive survey of the field of medical education in the United States, including a brief description of each medical college and elaborate tables giving medical school statistics. During the past year large sums of money have been given to medical education, and many new buildings, laboratories and hospitals have been built. There have been marked advances in medical education. Forty-four medical colleges have adopted resolutions to raise their entrance requirements to include one or more years of work in a college of arts. Twenty-one of these will require two years of university work. Three state examining boards, Minnesota, North Dakota and Connecticut, have secured laws raising the standard of preliminary education respectively to two years, two years and one year of work in a liberal arts college in addition to the usual four-year high school education. For the session of 1906-7 there were 24,276 medical students, a decrease since 1905-6 of 926, or 3.7 per cent. Regular students decreased 3.5 per cent., homœopathic students decreased 4.2 per cent., eclectic students decreased 15.4 per cent., and physio-medical students decreased 11.8 per cent. The total number of graduates in 1907 was 4,980, a decrease since 1906 of 384, or 7.2 per cent. Regular graduates decreased 5.2 per cent., homœopathic graduates decreased 21.3 per cent., eclectic graduates decreased 34.9 per cent., and physio-medical graduates decreased 77.3 per cent. There were 903, or 18 per cent. of the 1907 graduates in medicine who also held baccalaureate degrees. Without including the graduates of 1907, there are now in the United States one physician to every 636 persons. The total number of medical colleges remains 161, the five colleges ceasing to exist being replaced by five new schools. Colleges are gradually lengthening their sessions; now 102 have sessions of over thirty weeks of actual work, where there were only 86 so reported last year. Of the 152 colleges which give the full four-years course in medicine, 97 are located in cities of 100,000 or more population. There are still 9 colleges having 220 graduates this year, which are located in cities of 10,000 and less population. There are seven medical colleges which teach only the first two years of the medical course.

The first meeting of the Council of the newly formed United Services Medical Society was held on May 30th.

It was decided that meetings be held at the Royal Army Medical College at 8.30 p.m. on the second Thursday in each month, commencing

on October 10th, 1907; that the annual subscription be 5s., payable in advance; and that a notification of the formation of the Society, accompanied by an invitation to join, be sent to all medical officers on the active lists and to those of the retired lists whose addresses can be discovered. Should any medical officer on the active or retired list of the Navy, the British and Indian Armies, or the Auxiliary and Colonial Forces not receive an invitation the council hope that, if desirous of joining the society, he will communicate with one of the Honorary Secretaries, Fleet Surgeon W. W. Pryn, R.N., "Tredown," 25 Idmiston Road, West Norwood, S.E.; or, Lieut.-Col. C. H. Melville, R.A.M.C., Royal United Service Institution, Whitehall, S.W.

The medical men of the interior districts of British Columbia have formed an association for mutual protection and co-operation, following out their announced intention of two weeks ago.

Dr. E. C. Arthur, of Nelson, was elected president, and Dr. A. Sutherland, of Revelstoke, secretary of the new association.

The object of the association is: "To further the interest of the medical profession in the interior of British Columbia by meeting for discussion of medical subjects, and to increase the protection to the public." Incidentally, one object is to secure for the profession in the interior representation on the medical council of the province.

At a reorganization meeting of the Cape Breton Medical Society, the following were the officers elected; President, H. E. Kendall; Secretary-Treasurer, Jas. Bruce; Vice-Presidents, M. D. Morrison, Glace Bay; R. C. McLeod, North Sydney; D. K. McIntyre, Sydney. Committees—Surgery: R. A. H. McKeen, J. J. Roy, L. Johnstone; Medicine: W. J. Egan, J. K. McLeod, T. Smith; Obstetrics: E. O. McDonald, J. W. McLean, F. O'Neil; Public Health: Wm. McKay, E. J. Johnstone, M. T. McLean; Special: Wm. McK. McLeod, J. A. McLellan, S. J. McLennan.

Side by side with the announcement in Hamilton of efforts to form an anti-vaccination league comes the statement from Dr. C. A. Hodgetts, secretary of the Provincial Board of Health, that 95 per cent. of cases of smallpox reported to the board were in unvaccinated cases; of 25 cases in hospital in Toronto, only two were vaccinated persons, one of whom, a physician, had not been revaccinated since early years

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At the eighth annual convention of the British Columbia Medical Association at Victoria, the following officers were elected for the

ensuing year: President, Dr. J. M. Pearson, of Vancouver; Vice-President, Dr. Corsan, of Fernie; Secretary, Dr. R. Elden Walker, of New Westminster; Treasurer, Dr. J. D. Helmcken, of Victoria.

It has been decided that the Dominion Government, through its Port physicians, will assume control of such lepers as are found on the west coast and transport them to Darcy Island, whence foreign cases will be deported. Such cases were formerly under the charge of municipalities.

Dr. R. A. Reeve, of Toronto, has been elected a life vice-president of the British Medical Association in recognition of his services as president.

The Alberta Government is creating a Provincial Board of Health, and has decided to copy the constitution and methods of the Ontario Board.

Dr. Wm. Workman, Assistant Medical Superintendent of the Hospital for the Insane at New Westminster has reisgned.

Dr. Fitzgerald Sutherland of Norwich, Ont., died on the 26th July, in his 76th year. He was born in Scotland, graduated at Toronto, and practiced in Kincardine and Norwich, being thirty-three years in active work at the latter.

Dr. James Brien, ex-M.P., died on August 10th, at Essex, Ont. He was a graduate of Queen's University, Kingston, in 1872, and practised at Ridgetown, and afterwards Essex for many years.

Dr. Joseph Leduc died suddenly at Dorion, Que., on August 11th.

Retrospect of Current Literature.

SURGERY.

UNDER THE CHARGE OF GEORGE E. ARMSTRONG.

E. W. Roughton, B.S. London., F.R.C.S. Eng. "Two Cases of Excision of Ruptured Spleen. *Lancet*, June 22, 1907.

Both cases were in female children, the first was run over by a light van, the other fell on her abdomen against a curb-stone. In the former

the symptoms were marked from the first, there being marked collapse, restlessness, a pulse of 120, abdominal wall hard and moving little on respiration. In the latter, it was 12 hours before there was evidence of any serious injury, when the pulse became accelerated and the abdomen was distended and rigid and especially tender to the right of the umbilicus. Laparotomy was performed in both, the one with the acute onset showed laceration and pulping of the spleen, the other two fissures on the diaphragmatic surface, one just below the middle extending completely across the organ and a much smaller one at the upper pole. Excision was performed in both cases with success. The peritoneal cavity was washed out with saline solution, some of which was left in, and intravenous injection was employed in both cases. Blood examinations were made by W. d'Este Emery, extending over a period of three years, during which the patient remained in perfect health and underwent the normal physiological growth of this period. The absence of the spleen did not appear to influence the blood counts from those in an ordinary healthy child about puberty. Those counts immediately following the operation gave the same general results as are found after any operation in which hæmorrhage is a feature. Both cases are now three years after operation, in perfect health; the first has some enlargement of the glands in the axillæ, groin, and both sides of the neck.

CUTHBERT WALLACE, M.B., B.S. Lond., F.R.C.S. Eng. "Diabetic Gangrene." *Practitioner*, July, 1907.

This article is one of twenty dealing with the subject of diabetes in general appearing in the present volume. A very good clinical picture is given of the onset and progress of this special form of gangrene, and the general lines of treatment advocated are those now applied in cases of senile gangrene. These may be palliative or radical. If the dorsum of the part is involved the only treatment is amputation above the knee as recommended by Jonathan Hutchison. So long as the gangrene remains dry, as it does until the more fleshy part of a limb is involved, the condition is not grave, but when it has become moist and septic absorption once begun we are dealing with a very serious condition. In such cases ablation of the entire limb may be called for. The diminution or disappearance of the sugar, after the removal of the limb, and the septic absorption produced by it, is a remarkable feature in a certain number of cases, and suggests that the glycosuria may itself depend upon, or be aggravated by, the septic lesion. Septic conditions in non-diabetics sometimes produce glycosuria, and it has been shown that a patient with active septic absorption cannot dispose of as much glucose at a meal as

a healthy individual, which would seem to support the above conclusion, as well as being an indication to operate rather than the opposite.

W. L. B.

MEDICINE

UNDER THE CHARGE OF F. G. FINLEY, H. A. LAFLEUR AND W. F. HAMILTON.

DIABETES. *The Practitioner* (London).

The July number of *The Practitioner* is termed "Special Diabetes Number," as fully one hundred and sixty pages are devoted to a brief but withal comprehensive discussion of many phases of the important subject of Diabetes. One finds among the names of those who contribute to the discussion, W. D. Halliburton, J. Rose Bradford, I. Walker Hall, Sir Lauder Brunton, Malcolm Morris and R. T. Williamson—household names in medicine. These, with the names of others, speak for the high order of the matter contributed and their papers bring together the results of specialists and thus "reflect the exact state of contemporary knowledge and opinion."

Halliburton reviews the teaching on carbohydrate metabolism and discusses the two chief theories as to the destination of liver glycogen, which may be formed even while the animal is on a purely *pro tem* diet—and which is increased by glycerin and ammonium carbonate. These two theories are: (1) that the glycogen is converted during life by the agency of a ferment into sugar, that this leaves the liver of the hepatic veins and is thus distributed for utilization in the tissues; (2) that glycogen never justifies its name, but is transformed into substances other than sugar. Between these extreme views the opinion prevalent among physiologists is of the nature of a compromise. Halliburton says that the liver is no doubt able to convert part of its glycogen into fat, but most of its glycogen is regarded as leaving the liver as sugar (dextrose). The kind of sugar leading to an increase of hepatic glycogen is *par excellence* dextrose, and those belonging to the monosaccharide family, of which tarvulose is the next in importance to dextrose.

An upset of this glycogenic function is the common cause of diabetes, and consists in either an increased formation of sugar from glycogen or to a diminished formation of glycogen from the sugar of the portal vein. "The organism certainly is unable to burn, that is to utilize sugar."

But diabetes is not a single disease as it can be produced in animals by many and diverse experimental methods. The forms this induced differ from one another in some important points.

In puncture diabetes as well as in alimentary glycosuria there is no fundamental disturbance of the power of the organism to burn sugar. It is a diminution or over-straining of the sugar-holding capacity of certain organs.

While admitting that the profession is still in the dark regarding the exact *modus operandi* or how it is that the pancreas influences the metabolic processes. Halliburton's discussion of diabetes after extirpation of the pancreas shows that (1) it is not the loss of the pancreatic juice in the intestines (Minkowski); (2) nor is the glycolysis accomplished in the blood by pancreatic internal secretion (Lepine) and indicates that most subsequent observers fail to support the view expressed by Otto Cohnheim, (3) that the internal secretion already mentioned stimulates the glycolytic action of the tissue cells. It is suggested that the tissue cells are unable to fully perform their functions and are defective in the direction of oxidative processes. They may not be able to prepare the sugar for oxidation.

In Phloridzin-diabetes there is no increase of sugar in the blood; and after all the carbohydrates in the body has been got rid of the dextrose nitrogen ratio persists, proving that the origin of the sugar is exclusively protein. (Graham Luck).

Body fat may arise from carbohydrate food, and fat may be converted into carbohydrate as seen in vegetable life and also by direct inference this is true in animal life (glycosuria intensified by feeding phloridzin animals on fat). Glycosuria is increased by ammonium carbonate administration (increasing the fat changing function of the liver). To explain these processes chemically showing how the long carbon chains of the fats are linked together from the shorter chains of sugar or how these carbon chains of fats are burnt up is almost a matter of guess work.

A study of acetonuria has helped in solving this problem. Acetonuria is common to a variety of conditions—starvation, febrile conditions, mountain sickness and sometimes in broncho-pneumonia, pernicious vomiting in pregnancy, in recurrent vomiting in children, sometimes as a result of late chloroform vomiting.

In W. Langdon Brown's article on Acetonuria we find the following recapitulation:—"Acetonuria is the result of increased metabolism of fat occurring in any starving tissue, but it is particularly apt to occur if oxidation is deficient, because then the final breaking down of the fat into water and CO_2 does not take place. Deprivation or non-utilization of carbohydrates is specially likely to cause acetonuria, both because the tissues are being starved and because the process of oxidation

is apparently closely connected with the carbohydrates. If the increased metabolism of fat is great enough to cause acid intoxication the condition becomes much more serious. The liver is unable to convert ammonia salts into urea. The diminished alkalinity of the blood renders it incapable of taking up as much CO_2. CO_2 is therefore retained in the tissues and internal asphyxiation results.

A wasting diabetic is being starved as he cannot use his carbohydrates. Fats are broken down and fatty acids result, yielding diacetic-acid and acetone, of which β-oxybutyric acid is the forerunner.

And acidosis may increase the wasting for autolysis proceeds faster in acid than in alkali media. Breaking down of fasting or oxygen-starved tissues produces acids which in their turn produce further breaking down of the tissues.

While the acids themselves are not directly toxic, but the associated diminished alkalinity of the blood may result in an internal asphyxiation of the tissues, expressing itself in vomiting, drowsiness, and coma. The acetonuria in delayed chloroform poisoning finds its explanation in a diseased liver which is unable to deal either with its own fat or with that reaching it from elsewhere.

Dr. J. Rose Bradford's contribution is a brief presentation of the present teaching regarding the pancreas and diabetes mellitus, with illustrative cases briefly sketched. He says diabetes is not an entity but a clinical label attached to a number of different conditions of different morbid anatomy and liable to follow different courses.

The interacinar or intralobar variety of chronic pancreatites is that form with which diabetes is usually associated—diffuse intralobular fibrosis. It is in this form that the islets of Langerhans undergo degenerative changes. From Dr. Bradford's article one must infer that the conclusive proof is not yet adduced showing that diabetes can be attributed to the destruction of these islets, but experimental results certainly associate the glycosuria with the loss of an internal pancreatic secretion.

The paper by J. Walker Hall deals with the Basis of Therapy in this disease. He points out the necessity of arriving at a conclusion about the capacity of the tissue cells in diabetes, and regards undernutrition as the greatest danger of the diabetic subject. Abnormal, intermediate or by-products result in the unusual metabolic processes going on in the disease and these endanger the life of the cell. Already we have seen that these intermediate products express themselves in an excess of ammonia acetone and acetic acid, and quantities of these substances vary directly with the extent of the impairment of the assimilative powers of carbohydrates. Hence the necessity of increasing the activ-

ities of cellular metabolism with regard to carbohydrates and at the same time protecting the cells from the injurious effects of incomplete or abnormal metabolites.

The extent of undernutrition is roughly gauged by the amount of ketonuria—acetone bodies, acetic and oxybutyric acid.

By "tolerance" is meant the utilization power or capacity of the tissue cells of the diabetic for carbohydrates, and thus a key is furnished to the problems of treatment for the individual phase or extent of the condition; a standard test diet is given containing, among nitrogenus elements, say, 100 grammes of bread. If this produces no glycosuria more is given until sugar appears, then it is continued until the sugar is constant and the bread then diminished. Effects of exercise, drugs, etc., on the sugar output and ketonuria may all be observed.

The main object is to raise carbohydrate capacity of the tissues, increasing the utilization of the fed carbohydrates, diminishing the amount of unused sugar and decreasing acetonuria and the acidosis.

Von Noorden and Naunyn's classification of glycosuria in regard to "tolerance" is quoted, and the article closes with special reference to ketonuria.

Alfred R. Parsons' views on the drug treatment of diabetes mellitus are found in the following summary:

1. There is no specific treatment for diabetes.
2. Drugs play at best only a subsidiary part in diabetic therapeutics.
3. Opium is the best anti-glycosuric drug at present available.... most useful in severe cases. Dose 1-5 grs. of the extract.
4. Jambul may be used as an alternative to sodium salicylate or aspirin.
6. A positive ferric chloride reaction indicates the daily administration of sodium bicarbonate in doses of 150 grains and upwards.
7. The intravenous injection of 35 ounces of a 3 to 4 per cent. solution of carbonate of soda affords the best chance of restoring consciousness to diabetic coma.
8. Constipation should be guarded against in all stages.

Dr. R. T. Williamson has studied the action of certain drugs and supplies a few illustrative charts showing the results of the administration of the salicylates and aspirin. He has often found these drugs more satisfactory than opium, especially in the milder forms of the disease.

Dr. Marcel Labbé, of Paris, deals with the subject of Diet and Diabetes. He divides diabetic patients into two main classes:

(1) those without loss of nutrition,

(2) those with loss of nutrition.

In the first class we found the majority of patients, the fat diabetic of older writers,—the arthritic diabetics of the text-books. In them the nitrogen balance is maintained. They possess a *relative tolerance* for carbohydrates, and glycosuria is produced only when the patient exceeds his carbohydrate tolerance; the sugar originates from the carbohydrates of the food.

In the second class the condition is very different. The nitrogen balance is upset, tissues are destroyed. He is unable to tolerate even the smallest amount of carbohydrate, all that he may take is being excreted as sugar. Even when carbohydrates are absolutely excluded from the diet, glycosuria continues, originating from the food and from the tissues—the carbohydrates, albuminuria and fats.

In both classes dieting is the chief means of controlling the disease in the first class of cases, while in the second it is no less important in warding off the three-fold danger of hyperglycæmia, (hyperglycistia)—from loss of nitrogen nutrition and from acidæmia (ketonuria).

Dr. Labbé sees a difficulty in the contradictory nature of the indications, "Hyperglycæmia," hyperglycistia calls for a reduction of carbohydrates, nitrogenous denutrition requires an abundant intake of albuminoid matters; but acidæmia is produced by excessive meat diet and on that account must be fought by vegetarian diet. Of two evils the less must be chosen; the danger of hyperglycistia being less serious than that of acidæmia, excessive use of albumins must be avoided and the patients must not be deprived of carbohydrates; the diet must be plentiful and rich." There are two successive phases in Dr. Labbé's treatment of the first class (diabetes without loss of nutrition) of patients. In the first, the care of hyperglycæmia. The carbohydrates are gradually lessened until no sugar is found in the urine. This may be accomplished in two months or so, as an abrupt reduction is not often satisfactory. The amount of carbohydrates in the diet of tolerance. The amount of carbohydrate is gradually increased until sugar again appears; then again reduced until glycosuria disappears. The actual degree of tolerance lies between the last two amounts tried that which causes and that which does not cause sugar to appear in the urine. On such a diet it is safe to keep such a patient, examining his urine from time to time, while such treatment has a powerful and positively curative effect upon diabetes with loss of nutrition.

Glycosuria and Life Insurance forms the subject of an interesting paper by Bertrand Dawson of London Hospital. "The only safe rules,"

says Dr. Dawson, "with proposers up to 45 or 50 are to treat copper reductions as due to sugar and glycosuria as diabetic unless there is evidence to the contrary. In the absence of such evidence, the case must be regarded as diabetes *in posse* if not *in esse* and the proposal declined, or in special circumstances, accepted for a short term at high rates."

W. F. H.

STEWART AND RITCHIE. *Edinburgh Medical Journal*, May, 1907.

The dose of tuberculin R. found most suitable in the case of adults was 1-500 mgrm., and in children under 12 1-1000 mgrm.

The term "negative phase" is used to indicate the diminution of the tuberculo-opsonic index which follows the inoculation of an infected person with tuberculin. The depth of the negative phase, as a rule, is less where the primary index is low than where it is high. The usual fall in a tuberculous patient was 0.2.

The tuberculous cases which were inoculated number 62, and include all the more common forms of medical and surgical tuberculosis. In 56 of these cases a negative phase was obtained. This represents a proportion of 90.3 per cent. In 13 cases, either normal or suffering from diseases other than tuberculosis, in no instance did the observers obtain a negative phase after inoculation with similar doses.

The following conclusions are drawn:

1. A single estimation of the opsonic index is an unsatisfactory method of diagnosis, as both tuberculous and non-tuberculous cases fall within and without normal limits.

2. If a negative phase appears after inoculation, the presence of tuberculosis may be diagnosticated.

3. The absence of a negative phase indicates the absence of a tuberculous infection.

The method which the authors advocate, as may be seen, compares favourably with all that has been claimed for the reliability of the old tuberculin test. It has, however, the distinctive advantages of being applicable to any and every case, of interfering in no way with the patient's ordinary avocation. The tuberculin employed is a bacillary product, and is free from such toxins as the old tuberculin contained. The dose given is a small one, being within the therapeutic limit. This quantity has been given now in scores of cases and no instance of harmful effects or discomfort attributable to it.—(*Medical Review of Reviews*, Aug., '07.)