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Books and Pamphlets.

THE ILLUSTRATED SCIENTIFIC NEWS. Published by Munn & Co., New York.

This is an admirable little monthly, devoted to science, handsomely illustrated. It contains valuable and useful instruction for all classes, and is published at the low price of $1.50 per annum.

THE BACTERIA, by Dr. Antoine Magnin, Translated by George M. Sternberg, M.D., Surgeon U. S. Army. Boston: Little, Brown & Co., 1880. 8vo. pp. 227. With plates. Price $2.50.

This admirable little work will be of great interest to scientific men, professional and otherwise, especially in view of the investigations by Pasteur, Tyndall and others. The text has been materially elucidated by the introduction of several drawings and some beautiful photo-micrographs. The work treats of I. their history, morphology, organization and classification, and II. their physiology, development in general, and development in different media. We strongly recommend the work to any of our readers who may desire to learn just what is known about bacteria.

DIAGNOSIS AND TREATMENT OF EAR DISEASE.—By Albert H. Buck, M.D., Aural Surgeon to the New York Eye and Ear Infirmary, etc., pp. 411. Wm. Wood & Co., Publishers, 27 Great Jones St. New York. 1880.

This is a condensed and practical work on the treatment of diseases of a part of the body very often overlooked or unheeded by the general practitioner. The author has given his experience in private and hospital practice of a large number of cases, which can not fail to be of great value to general practitioners. In the first chapters is given a brief sketch of the physiology of the organs of hearing. This is followed by a description of the instruments necessary to examine the ear, and how to use them, and the remainder of the volume is devoted to the pathological conditions of the ear and their appropriate remedies.

A TEXT-BOOK OF HUMAN PHYSIOLOGY. By Austin Flint, Jr., M.D., Prof. of Physiology and Physiological Anatomy in the Bellevue Hospital Medical College, etc., etc. Third edition; revised and cortected. New York: D. Appleton & Co., 1881. Toronto: Willing and Williamson: Price $6.00.

This work is already well and favorably known to the medical profession, and the author has spared no pains in the revision of the present edition, which is fully in accord with the existing state of physiological knowledge. The only objection which can be urged against the work is that it is rather large and unwieldly for general use as a student's text-book, but as a work of reference it is highly to be commended. The author we observe adopts the views, in the present edition, advanced by Bowman, and lately confirmed by Heidenhaim and others with regard to the functions of the Malpighian bodies of the kidneys. He also takes occasion to air his views on the production of animal heat in the body, by the union of oxygen and hydrogen in the formation of water. The latest views on fecundation and the development of the ovum are fully and clearly stated. The work is illustrated with upwards of three hundred wood-cuts, and several lithographic plates.

THE PRINCIPLES AND PRACTICE OF SURGERY. Being a Treatise on Surgical Diseases and Injuries. By D. Hayes Agnew, M.D., LL.D., Professor of Surgery in University of Pennsylvania. Profusely illustrated. Vol. II. 8vo. Pp. 1,066. Philadelphia: J. B. Lippincott & Co. Toronto: Hart & Rawlinson. Price $7.50.

The above excellent work on surgery will be published in three volumes, and when completed will be second to none in the English language. The author is a man of large experience, both in hospital and private practice, and he is giving to the profession and to the world the full benefit of his talents. The work is one which the profession in the United States has reason to be proud of, as it reflects the highest credit on American surgery. We will not, we cannot, attempt anything like a review of it; suffice it to say that the author has embodied in this great work, besides the principles and pathology of the subject, a thorough description of all the instruments, appliances and manipulations belonging to surgical treatment.

Births, Marriages and Deaths.

At Napanee, on the 17th ult., Dr. W. T. Stuart, Professor of Practical Chemistry in Trinity Medical College, Toronto, to Margaret B., daughter of Rev. H. Gibson, of Lachine.

On the 8th ult., Dr. J. A. Gregory, of Fredericton, N. B., suddenly.

On the 14th ult., Dr. George Burnham, of Peterboro', in the 67th year of his age.

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Original Communications.

RENAL CIRRHOSIS—WITH SPECIAL REFERENCE TO ITS LATENCY AND TO SUDDEN, FATAL MANIFESTATIONS OCCURRING IN ITS COURSE.

Being a Clinical Lecture delivered May 28th, 1881, in the Summer Session Course, by WILLIAM OSLER, M.D., M.R.C.P. Lond., Professor of the Institutes of Medicine, McGill University; Physician and Pathologist to the Gen. Hospital, Montreal.

Stenographical Report by S. A. Abbott, Esq., of the "Hansard" Staff.

GENTLEMEN,—I speak to you to-day upon renal cirrhosis, or chronic interstitial nephritis.

The various modes of onset of disease constitute an exceedingly important and interesting subject of study. You know that one of the very first questions we ask a patient is, how did the disease begin? The answers got to this question are very varied. One patient will say, it began suddenly; I was feeling quite well; it came on with a headache; I got feverish; I had a pain in my back; I was taken with vomiting; and various other answers, all of you have, no doubt, received in ascertaining the clinical history of cases. In another set of answers the patient will tell you that he cannot fix definitely the commencement of the disease; that he has not been feeling very well, but cannot state the precise time at which the failing health began.

Now I wish to call your attention in this particular affection to its remarkably stealthy method of onset. There is no disease with which we are acquainted which comes on so insidiously and so stealthily. Indeed, its victim may know nothing whatever of the existence of any grave disease until he is prostrated by one of its severe accidents to which I shall shortly refer. It is this insidious course which makes it at once an exceedingly formidable affection and one worthy of your closest attention.

The patient before you offers a very good example of the disease in question, and has many of its most characteristic symptoms. I will read to you a concise clinical history of his case as obtained by Mr. R. J. B. Howard:—

E. L., æt. 31, sailor, large, strongly-built man, admitted May 18th, with headache, vomiting, and partial blindness. Has been a healthy man; a beer drinker and has occasionally gone on "sprees." Has had bubo; no evidence of secondary syphilis. Two years ago lost his nose from frost-bite.

When coming across on his last voyage, about 12 days ago, had a slight pulmonary disorder; the doctor called it inflammation. A week ago he had swelling and inflammation at inner canthus of right eye from lachrymal abscess. During these attacks he had headache, and latterly the feet have been swollen. On the 17th, the headache became much worse and partial blindness came on. Vomiting had been present for several days.

Condition on examination was as follows:—Well nourished man, good complexion, complains of headache and blindness, cannot see fingers six inches in front of the eye. Has perception of light. Pupils of medium size, respond to light, but there is a peculiar dull look about the eyes. Dr. Buller reports, "optic discs somewhat hyperæmic and indistinct at margins, nothing abnormal, retina present. Headache is general. Vomited last night and this morning. Bowels are freely opened. Tongue a little furred. Temperature normal. Chest well formed; apex beat half an inch outside the nipple line; impulse slow, heaving and forcible. Pulsations 60 per minute. Heart's dulness slightly increased. On auscultation, no murmur; sounds loud and distinct. There was nothing of special note in lungs. Examination of abdominal organs negative. Urine clear, light colored, sp grav. 1009, acid, contains a moderate amount of albumen and numerous pale casts. Radial artery feels firm, pulse hard and strong, tension greatly increased."

The patient improved very rapidly. On the 20th he could count fingers, but could not see to read. The amount of urine passed has been estimated, and found to be about 100 ozs. daily. Urea diminished, only 299 grains for the 24 hours. The headache has gradually disappeared and the

vomiting is now checked. The feet are not swollen. The state of the urine remains unchanged. The circulatory symptoms persist; the high degree of arterial tension which exists is well shown by this sphygmographic tracing which I hand round.

Summing up the chief symptoms which this man had, they were: headache, vomiting, and disturbance of vision. These were the symptoms he complained of; but the symptoms which we discovered, and of which he had no knowledge, were—that he was passing nearly double the normal quantity of urine, that it was albuminous and contained hyaline and finely granular casts; that his heart was hypertrophied; that he had increased arterial tension, and that there was slight dropsy of the feet.

This latter group of symptoms which I have mentioned, excluding altogether those he complained of when he came in, is alone sufficient to enable you to frame your diagnosis of the disease, particularly if they occur in connection with slight degrees of dropsy. There may be exceptions, but in the great majority of cases they will be sufficient for your purpose. The affection which is indicated by them is one of the forms of chronic Bright's disease. The three varieties of this disease, characterized according to the special morbid condition of the kidneys, are: first, that associated with the large white kidney; second, the form associated with the waxy kidney; and third, the form associated with the contracted kidney. It is the latter which this man suffers from.

Now in this disease the condition of the kidney is shown in the description of these organs from the girl who died in the hospital ten days ago, and the post mortem on whom most of you saw. Firstly, the kidneys are reduced in size. Secondly, on stripping off the capsule, you find it is thickened and opaque. Thirdly, the surface of the organ, instead of being smooth, presents a number of irregular nodular projections, or granules, large and small,—hence the term granular kidney. In stripping off the capsule, portions of the kidney substance adhere to it. Fourthly, on section, the organ cuts with great resistance, and it feels tough and hard. Fifthly, on examining the organ, you find that the cortical substance is greatly reduced, forming a very narrow zone above the pyramids. In some places the pyramids approach to within a line or a line and a half of the surface. Sixthly, the arteries are noticed to be unusually distinct, particularly those at the bases of the pyramids, and they often project above the level of the substance. Small cysts are also common, but they are not seen in this specimen. The color of the organ, in this special instance, was pale and not reddish. The pyramids were reddish, but the general color of the organ was pale grey. These are the coarse features of the kidney in this form of Bright's disease.

Microscopically, as you will see in a section taken from this organ, the chief characteristic is an enormous increase in the fibroid elements of the organ. In a healthy kidney there is only a very small amount of fibrous tissue between the tubules, around the Malpighian tufts, and about the arteries of the organ. The amount is so small that Dr. Beale, one of the leading histologists in England, denies the presence of a special fibroid framework of the kidney. But in this affection you will see that between the tubules, there is a large amount of a new growth of fibrous tissue. The tubuli uriniferi, instead of being in close apposition, are separated from each other by distinct zones of fibrous tissue, and the Malpighian bodies are also surrounded with the new growth. The arteries are much thickened, both in the adventitia and in the muscularis. The condition of the renal epithelium in the tubes varies a good deal. In some tubules you will find it healthy looking, in others it is degenerated, granular and fatty; so that in reality the essence of the process is, just as in the case of the fibroid lung of which I spoke to you the other day, and as in the case of the fibroid liver, an overgrowth of the connective tissue of the organ. This produces atrophy of the secreting structure, and impairment of the function of the gland.

Associated with the small, contracted kidneys you have a remarkable condition of the circulatory system. The arteries of the body are thicker and firmer than is natural, particularly the smaller ones. There is usually atheroma in the larger vessels. With reference to the special change which goes on in the smaller vessels, there is still a great deal of dispute. Drs. Gull and Sutton believe that the change is chiefly in the outer coat. They call this degeneration arterio-capillary fibrosis, a fibroid change in the small arteries and capillaries. Dr. Johnson believes that the change is chiefly in the middle coat, resulting in hypertrophy of the muscular elements. Drs. Gull and Sutton hold that

the changes in the arteries and the changes in the kidneys go on simultaneously, and are both the expression of a common cause; whereas other writers think that the changes in the arteries are secondary to the changes in the kidney. In addition to these muscular changes, the heart is found hypertrophied, more particularly the left ventricle. It is increased in thickness and the muscular walls are hypertrophied. Thus, cirrhosis of the kidney, arterial degeneration, and hypertrophy of the heart, are the three main pathological features of this form of Bright's disease which you meet with in a post mortem.

The hypertrophy of the heart, which is a very constant symptom, is supposed by Traube to be due to the increased difficulty with which the blood circulates through the kidney, owing to the destruction of a large number of Malpighian tufts. It is, according to this view, a compensating hypertrophy, that is to say, hypertrophy makes up for the destruction of a considerable vascular area in the kidneys. Others think that the hypertrophy is the result of chronic changes in the arteries, in which the arteries of the kidney participate. Bright's view with reference to the hypertrophy of the heart was, that the blood in kidney disease not being so pure as in health, did not circulate through the capillaries of the body with the same facility; hence the need of the heart to increase its force of contraction in order to propel the blood.

A knowledge of the condition of the heart and arteries isa key to explain many of the symptoms of this form of kidney disease. Thus, one of the remarkable features of this disease, remarkable in contrast to the other varieties of Bright's disease, is the large amount of urine secreted. This man has been secreting double the normal amount of urine. This would appear to be due to the hypertrophy of the left ventricle, and to the increased blood pressure within the arteries. You know how much the watery part of the urine depends upon vascular pressure. As a rule, the greater the blood pressure within the renal vessels, the greater the amount of water which is filtered through the Malpighian tufts. Though there is a great destruction of these tufts in renal cirrhosis, still the compensating hypertrophy of the heart is not only sufficient to counterbalance their loss, but even so to increase the pressure in the remaining tufts that a larger amount of urine is filtered off. That this is the case is shown by several circumstanees. In the first place, if you keep a patient with this form of kidney disease absolutely at rest the amount of urine diminishes. This fact hasbeen established by Bartels after several very careful observations. At rest the blood pressure is not so great as when the patient is moving about, as the pulsations of the heart are not so forcible. Then, so soon as hypertrophy of the left ventricle begins to fail, when degeneration comes on, the amount of urine diminishes while its specific gravity increases.

Among the most remarkable symptoms of chronic Bright's disease, are those which come under the heading of *uræmia*. This term was first used when the symptoms grouped under it were all believed to be due to the poisoning of the blood with urea. That view has now been considerably modified, but the old term which embraces these symptoms is still retained. I shall not speak fully with reference to the supposed causes of uræmia further than to mention that some still suppose it to be caused by the retention of urea; others, that it is due to the presence of carbonate of ammonia in the blood. A third view is that it is neither of these substances, but those bodies which we call the antecedents of urea, creatinin, tyrosin, &c., the various nitrogenous excreta, or the products of the waste of the tissues. A fourth view is that these symptoms of uræmia are due to œdema of the brain.

Now, among these manifestations of uræmia some are trifling and others are exceedingly grave. Among the minor manifestations may be mentioned those which this patient has suffered from—headache, vomiting and impairment of vision. The more severe symptoms are convulsions, delirium, coma, sudden œdema of the lungs or of the glottis, inflammation of a serous membrane, pleurisy, pericarditis and meningitis. This patient before you has only suffered from the minor manifestations of uremia, but I would like you all to have this case fully impressed upon your minds, particularly with reference to what I am going to tell you later as to the insidious nature of this disease. You remember that when we first saw this man we did not think of any kidney trouble, but from his symptoms and appearance that he most probably had some cerebral disease. When I first saw him on the day of his admission my first thought was that he had probably cerebral syphilis, mistaking the ragged condition of his nose for an effect of

lues. He had the vomiting, the headache, and the disturbance of vision, three important symptoms of intra-cranial mischief. I would direct your attention specially to the disturbance of vision inasmuch as it is an important symptom, and you will probably not see this form of visual disturbance for some time again. It is what is known as *uræmic amaurosis*. I mention it because I wish you to distinguish it carefully from another form of impaired vision common in chronic Bright's disease, viz., *retinitis albuminurica*. In uræmic amaurosis the cause of the impairment of vision is cerebral. The examnation of the retina is negative. Its clinical features may be briefly summed up in the rapidity of its onset, the shortness of its duration, and the quickness of its departure. It rarely lasts any length of time—in this man only three days—whereas in the retinitis albuminurica, the impairment of vision comes on slowly, the cause is peripheral, and there is a definite lesion in the retina, chiefly seen about the macula, in the form of small hemorrhages, and with these there is usually some swelling of the disc. In this form the impairment of vision comes on slowly and is rarely so severe as in the uræmic amaurosis.

But that to which I wish specially to call your attention to-day—and I am sorry to have had to take up so much time in clearing the ground—is the fact that *these severe symptoms of renal cirrhosis may break out in all their violence in an individual who may consider himself in perfect health, and who may be so considered by his friends, and even by his medical adviser, if the latter has not carefully examined into his case.* The case of the patient who was admitted under my care on the 7th of May, and who died after a residence of two days in the hospital, has directed my attention to certain points in connection with the insidious course of cirrhosis of the kidney.

The first manifestation of the disease may be the onset of severe cerebral symptoms, convulsions, delirium or coma.

Cases in point are as follows :—A friend of mine, aged 30, a fellow student, and a man whom I had known since 1863, a graduate of McGill College, a strong healthy man, and in active practice, was suddenly seized with convulsions which came on at night with few, if any, premonitions. The day previous to their onset he had done his work as usual and appeared to be, as his wife expressed it, "in radiant health." The examination of his urine by the attending physician showed the presence of albumen and tube casts, and the diagnosis of chronic Bright's disease was made. He become comatose and died in a few days. I saw him a few months before his death and he looked in his usual vigor. He made no complaints of failing health nor were any alterations perceptible on his countenance. Six or eight months before he had had considerable domestic and mental trouble, owing to the sudden death of his father, and he had not been well for several weeks at that time, but apparently had recovered completely. He had no idea whatever that he was in this dangerous condition. It is to be noted that prior to this attack he was a good deal worried and anxious about his children who were ill.

The first manifestation may be delirium passing on to coma. That was seen in the patient named Weir who was admitted on the 7th of May. I will briefly call your attention to the main features of his case.

This patient was a vigorous and healthy man, aged 44, a foreman in G. T. R. employ. Habits temperate for past ten years, previously had been a drinker. Had been in usual health, but had complained of headache, and his wife stated that he had passed water more frequently of late. On May 6th he was admitted with an active delirium which had come on suddenly 36 hours before. Urine found to be albuminous and contained granular casts. The symptoms were regarded as uræmic. He became comatose on the 7th, and he died at 2 a.m. on the 8th, after an illness of a little over three days. A point to be noted in connection with this case was that the patient had had a great deal of mental worry at the time as a strike was going on. The *post mortem* did not reveal extensive renal cirrhosis, as was anticipated, for the kidneys, as you see, are not reduced in size and do not present the external characteristics of interstitial nephritis, but they were firm, and on microscopical examination there is evidence of a chronic nephritis. The arteries are thickened, some of the Malpighian tufts are degenerated, and there is an increase in the fibrous tissue abont the capsules. A fact to be learned from this case is that severe uræmic symptoms may develope at a very early stage in renal cirrhosis, even before the characteristic contraction of the

organ occurs. This is, of course, very uncommon, but that it does take place is evident from this case.

The third case illustrating the suddenness of the onset of cerebral symptoms in this disease was that of the girl who died about ten days ago, and from whom these kidneys were taken She was 26 years of age, and up to the time of her admission to the hospital had not suffered from special symptoms of kidney disease. She came in suffering from headache, vomiting, and hæmorrhage from the nose, uterus and navel. She got dizzy, had convulsions, became comatose and died. The urine was albuminous and contained casts. The condition of the kidneys was as you now see in these specimens. The occurrence of hæmorrhage is worthy of your attention, as it is occasionally seen as one of the severe symptoms in Bright's disease. In the case of this patient it is also worthy of remark that she was friendless and had been ill-treated for years. These three cases will serve very well to illustrate the fact which I wish particularly to impress upon you, namely, that severe uræmic symptoms may be the very first manifestations to the patient, to his friends, or his physician of the existence of kidney disease.

The importance of a knowiedge of these facts is also very evident from a consideration of the medico-legal aspect of such cases. You may be called to attend a man in a profound coma, who has been stricken down suddenly without any premonition, and while attending to his business, and he even may die in three or five hours under circumstances at first suggesting narcotic poisoning.

The first manifestation may be an apoplectic seizure.

In October, 1879, one afternoon as I was going down stairs prior to my lecture at the College, one of the veterinary students, aged about 25, while coming in through the side entrance, was taken with apoplexy before my very eyes. He leaned against the wall and stated that he was powerless in his left side. We helped him into the waiting-room, and from the suddenness of the onset I supposed at once he must have heart disease and apoplexy. On placing my ear on his chest I perceived a pronounced, heaving impulse of the heart but no murmur. There was marked cardiac hypertrophy. By the time we got him to his boarding house the paralysis was complete on the left side; he had lost consciousness and was becoming comatose. He was taken to the hospital and we examined his urine, which was clear, albuminous, and contained numerous casts. The arterial tension was increased. He died in 24 hours. That young man had never suffered from any special symptom pointing to renal disease. He had been attending to his work as usual, though he had never been very strong, and on several occasions I looked at him thinking he might have some constitutional disease. He did not look healthy, but the only things he had complained of, had been occasional headaches and palpitation of the heart, and so far as I remember he had not consulted a doctor.

Another case in which the first severe symptom of renal cirrhosis was apoplexy occurred under Dr. Ross' care two years ago in 23 Ward. A woman came in with hypertrophy of the heart, high arterial tension, albuminous urine, and casts, finely granular in character. Cirrhosis of the kidney was diagnosed, and she was placed under suitable treatment. Three days after admission to the hospital she died in two hours with an enormous apoplectic effusion into the brain.

The arterial degeneration in this affection renders the vessels fragile, and the powerful contraction of the hypertrophied left ventricle is a source of constant danger. A large proportion of all cases of apoplexy occur in connection with contracted kidneys, owing to the existence of these two factors.

A third way in which this disease may declare itself is by inflammation of some serous membrane, the pericardium, the pleura or the meninges of the brain.

A case which early called my attention to the insidious nature of this disease was the following :—A florid, full-blooded Englishman, an old sailor, aged 63 years, who had usually enjoyed excellent health, though he had occasionally, I believe, suffered twinges of gout, was suddenly seized with symptoms of an acute febrile affection, had high fever and considerable constitutional disturbance. To make a long story short, he died at the end of four days of acute sero-fibrinous pericarditis. He had a large exudation in the pericardium. The only other disease found in his body was fibroid kidneys, perhaps of gouty origin, as gout may be a very important factor in the production of this disease.

The fourth sudden manifestation in this disease to which I will direct your attention is œdema of the glottis, or more frequently of the lungs.

Three years ago an old man was brought from the House of Refuge to the Hospital, suffering from intense dyspnœa. On examination of the lungs hydro-thorax of the left side and œdema of the left lung were diagnosed. He refused all treatment, and died within 36 hours of his admission. The *post-mortem* revealed small contracted kidneys, intense œdema of the left lung and hydrothorax of the opposite side. The effusion and transudation of serum takes place sometimes into the pleural cavity and sometimes into the lungs. In this case there were no adhesions on the left side, while in the other side there were extensive adhesions and the transudation took place into the lungs. There was no œdema of the legs in this instance. The urine was albuminous and there were casts.

An interesting point in connection with the occurrence of this œdematous effusion is the fact that Traube attributed the uræmic symptoms in this disease to the serous transudations, and the post mortem of the man Wier favors this view, as there was considerable œdema of the membranes of the brain and a good deal of moisture throughout the substance.

These are certain of the modes of termination of cirrhosis of the kidney with which you should be acquainted and which it is exceedingly important you should bear in mind.

Now, among other symptoms which I will only mention in connection with this chronic form of Bright's disease, there is the occurrence of a dyspnœa, uræmic asthma, without evidence of œdema of the lungs or chronic bronchitis, dependent upon cerebral causes. It is of rare occurrence, but it is a condition which you should bear in mind. The bronchitis, the vomiting, and diarrhœa are also symptoms to which I will not further refer.

The importance of a knowledge of these symptoms and these sudden manifestations in renal cirrhosis cannot be over-estimated. I have had two life insurance cases referred to me within the past few years, both of which bear directly upon this question. In one the patient had an Accident Insurance Policy. He fell on the ice and was stunned; felt unwell for some days, but did not see a doctor. Three or four months after, I forget the exact time, he was seized with apoplexy. The post mortem revealed contracted kidneys. The question was brought up as to the connection of the accident with the subsequent event. My opinion was asked, as the friends had some idea of contesting the case in the courts, but the existence of renal cirrhosis was to my mind quite sufficient to account for the apoplexy.

In the other, a middle-aged man had insured his life about seven months before his death, which took place quite suddenly. The autopsy disclosed very great atrophy of one kidney and a large red state of the other. No very satisfactory report was obtained of the state of the other organs, and the actual cause of the sudden death remains doubtful. But I have no doubt whatever that it was connected with the condition of renal inadequacy. My opinion was asked as to the possibility or probability of this man not being aware that he was unsound at the time of insuring. After the cases which I have narrated, illustrating the latency of chronic renal disease, you need not ask what my answer was. From the point of view of life insurance, there is no disease about which a company should be more on its guard. Its peculiar insidiousness will have become evident to you by the cases I have cited. The stealthy nature of the disease is increased by the fact, that albumen is not constantly present in the urine. A single examination is not sufficient to enable you to state positively upon its presence or absence, and it is often very slight in amount; and though you may examine for casts, you may go over a dozen sildes before finding one.

A patient may come to you who is passing a large quantity of urine, so that he has to get up, perhaps, two or three times in the night (that may be what he comes to complain of); the urine is of low specific gravity and contains albumen—perhaps only in traces. The daily amount of urea is decreased. It deposits, not a thick heavy sediment, but a light cloudy one, which on examination is found to contain hyaline and finely granular casts. There may or may not be œdema of the ankles. If you also find on examination that his heart is hypertrophied, that the arterial tension is increased, you may be tolerably positive with reference to your diagnosis—the man has fibroid degeneration of the kidneys. To be forewarned in such a case is to be forearmed, and a knowledge of what you may expect in these cases will enable you to take measures for the prevention, if possible, of the severe manifestations of which I have spoken. If a patient comes before you with these symptoms, you should see that the amount of his urine is kept up, and on no account allow it to diminish; that his pulse is kept thoroughly well regulated, and that he lives a quiet regular life and does not go to any excess in eating or drinking.

The treatment of the affection is in great measure a treatment of symptoms. Acting with cathartics upon the bowels and keeping the amount of urine up to the standard, are among the most important means to be taken.

NOTE.—June 7th. The patient who was shown to the class on the occasion of the above lecture was recently discharged, feeling as he expressed it quite well. He was still passing about 80 ounces of urine in the day, with albumen and a few casts. He looked well, fit for life insurance, and would pass in many examinations such as I have witnessed. Yet I know of no more likely candidate for sudden death than this same patient, who has the sword of Damocles hanging over his head, ready to fall with fatal effect when the tiny hair which suspends it is suddenly broken by the onset of convulsions, or one of the other accidents to which such patients are liable.

ELEPHANTIASIS.

BY T. T. S. HARRISON, M.D., SELKIRK, ONT.

(Read before the Ontario Medical Association.)

This case which I bring before you with some doubt and hesitation, I have called elephantiasis. It has this characteristic of that disease, that the affected limb is enormously enlarged. It differs, however, from the typical elephantiasis in the absence of the thickened, indurated tuberculated and cracked integument.

Patient, aged 20., Canadian, born of German parents. Parents, and brothers and sisters, healthy; the mother's family consumptive; the maternal grandmother died of cancer.

J. A., at birth was healthy; a very large, fine, child. At the age of two and half his mother noticed that one leg was growing faster than the other. I first saw the boy when about three years of age. I then found the left leg decidedly the longer. The right was normal in contour, while the left was not only longer, but larger and abnormal in shape; the skin hung loosely and it had a soft, doughy feel, was largest at the ankle, and had no bulge or projection at the calf. I gave the opinion that there was arrest of growth in the right leg, but had to say that the left had some peculiar affection of the soft tissues at least. The mother said that other medical men had given the same opinion. The child was merely treated for his general health.

I saw the child occasionally as I attended other members of the family, for several years. The size and length of the limb increased so rapidly, that there was soon no doubt as to the abnormal growth of the tibia and fibula.

Some seven years ago, when about thirteen, I exhibited the boy at the meeting of the County of Haldimand Medical Association. At this time, the disease which at first was confined to the legs, had invaded the thighs; there was enlargement above the knee, and the femur was some ¾ of an inch longer than its fellow. The patella was broader, thinner, and flatter than natural.

Then the entire limb was, I think nearly, or quite five inches longer than the right. The weight of opinion was against surgical interference, though amputation, resection of the bones of the leg, ligation of the femoral artery, division of the nerves, etc., were mentioned. For some years, until he was about eighteen, the deformity increased, but the mother thinks it is now stationary; she, at least, has not to increase the size of his stockings since that period.

You see the state of the limb to-day. The enlargement has extended up the thigh. The femur is nearly two inches longer than its fellow. The circumference above the knee is four inches greater than that of the right, while the circumference at the ankle is 13 inches greater than that of its fellow (the right leg 8½, left, 21½ inches.) This size (at the ankle) would be increased, were he to keep long on his feet, and diminished after his night's rest.

In the cut you will observe the right foot rests on some books. These, though they do not bring it to a level, are 5⅞ (five and seven-eighths inches high.) The femur is bowed, so as to take nearly, or quite an inch off its length. It is increased in size and altered in shape, the spine at the shin entirely absent. The skin is soft, and with the tissues it covers, has a soft, flabby feel. The hairs on the affected parts are very