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ON THE STAGES AND FORMS OF SYPHILIS WITH MORE ESPECIAL REFERENCE TO THE HEPATIC MANIFESTATIONS OF THE DISEASE.[1]

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It is remarkable how during all these centuries, syphilis has remained sharply distinguished from all other forms of human disease, constituting a class apart. There are many other contagious maladies, not a few chronic infectious disorders, and, now-a-days, we are able to group these together; we can recognise common principles governing their course and effects upon the organism, and can see, or think we see, a common principle underlying the morbid changes in one or other tissue from the onset of the disease to its culmination—a principle identical in the infant and in the aged. So we can with syphilis—only habitually we do not. We treat and regard it as a something distinct.

Take for instance that disease which in its chronicity as in the lesions which characterise it, most closely resembles syphilis—tuberculosis. From the onset to the end, at every stage, from the primary lesion to the most extensive generalisation of the process, we see one morbid change at work, namely, the focal multiplication of the bacilli leading to the development of tubercles. It is true that according to circumstances these tubercles may vary in their characters from a condition in which small cell infiltration is so extensive as closely to approximate to miliary abscess formation, through conditions of so-called epithelioid cell overgrowth to a state in which fibroid connec-

[1] Delivered at the meeting of the Ontario Medical Association at Toronto, June 1st, 1898.

tive tissue development is so excessive as to mask everything else, save, perhaps, necrosis and cascation. But the fact remains that we do not sharply differentiate successive stages of the disease, or consider that the successive stages are characterised by the development of specific manifestations. At the most, in one organ, the lungs, we trace such successive stages of the tubercular process, but we never think of laying down that what is to be made out in the lungs obtains for other organs, and for the body in general. On the contrary, a study of pulmonary phthisis alone has convinced us that the course of tuberculosis varies so greatly according to the interaction of two factors—the condition or reactive power of the tissues, and the virulence of the bacilli—that to attempt to plot out the course of the disease in each case into well defined stages is an impossibility.

With syphilis it is quite another thing. From Ricord onwards a primary, secondary, and tertiary stage have been clearly distinguished, and not only this, but according as to whether the disease is acquired in post-natal life, or has seized upon the individual while in the mother's womb, so do we recognise two different types of the disease.

There is, I take it, no more firmly 'fixed idea' in the whole of medicine than that of the absolute existence of these different stages and forms of syphilis. To-day, I do not want to pose as a revolutionist and an iconoclast, for speaking broadly, and regarding the bulk of the evidence before us, I, like all others, must acknowledge the utility of the divisions. But there is a danger in these fixed ideas, in medicine as in all sublunary affairs, and, to say the least, it is of benefit occasionally to enquire whether what is accepted of all men is so absolutely and entirely fixed and assured as we are accustomed to regard it.

What I am about to say is not novel. The unity of syphilitic lesions has been preached for now more than thirty years, in fact, ever since Wagner pointed out that all such lesions might be referred to the developments of a specific neoplasm. Perhaps Wagner went too far, for there are generalised fibroid conditions, which, as I shall have to point out in connection with the liver, are not directly due to the development of circumscribed neoplasms; but it must be acknowledged that neoplasms or infective granulomata are to be recognised in each stage and form of the disease. Nevertheless, the idea of the sharp demarcation of the different forms and stages of the disease seems to be as firmly planted to-day as it was prior to 1864, and the admirable protest of Nevins Hyde[1] and the writings of others do not seem as yet to have influenced the profession in general.

[1] Morrow's System of Genito-Urinary Diseases, Vol. II., 1893, p. 20, *et seq.*

At the outset, I must point out that it is not even necessary to have any recognisable first stage or cutaneous chancre. We know well, that in every case of infection, the infectious agent must make an entry from without into the tissues, and in a great number of cases we can discover the point or points of entry, and at such point or points we find evidences of primary local infection, whether on the skin or mucous membranes, and this local infection is strictly comparable with the cutaneous syphilitic chancre. But we also come across cases in which there is a complete lack of evidence of such superficial primary infection; we may find, for instance, the cervical or mesenteric lymph glands affected with tuberculosis without a sign of tuberculosis of the pharynx or tonsils or intestinal mucosa, cases which usually, though mistakenly, are spoken of as 'cryptogenetic.' What occurs in other diseases must at times occur in syphilis, and in going over my post-mortem records, in which to each case, I have subjoined a record of the clinical history of the case, I have been struck several times by observing that where well marked tertiary syphilis has been present in the organs, there has not been a sign of old penile or other chancre,[1] and more than once, in following up the clinical history, by finding that while the patient has freely admitted that he has led a loose life and suffered, it may be several times, from gonorrhœa, he has denied ever having suffered from chancre (vide case III). Now presumably an individual who had had a hard sore would not wholly forget the circumstance, nor is it rational to urge that a hospital patient who admits without constraint that he has led a life of excess and suffered from other venereal diseases, would conceal the previous existence of a chancre. Either then the chancre was so small and inconsiderable as to cause no inconvenience, or the virus gained entry into the system without causing any cutaneous disturbance.

In the female this absence of any superficial or recognisable first stage is especially noticeable; time after time the disease only manifests itself in the secondary stage. I would go so far as to say that the 'fixed idea' that there must be a chancre developed at the region of primary infection, has led to a thorough and general misunderstanding as to the nature of congenital syphilis. It is a popular fallacy to regard a considerable number of cases in which the father of

[1] In some at least of these cases without doubt, the same process has happened as occurs occasionally in connection with vaccinal cicatrices, namely, there has been so complete absorption of the cicatrical tissue, that the part becomes in the course of years absolutely normal in appearance. This complete absorption I need scarcely say, is characteristic of primary lesions of mucous surfaces, and is very frequent in the female.

syphilitic offspring is syphilitic, and the mother is apparently free from the disease, as due to the sperm being syphilised, or if this view be carried to its logical conclusion, it is supposed that the spermatozoon bears with it the syphilitic virus, be it bacillus or whatever the nature of the specific microbe, and introduces it into the ovum at the moment of conception, and thus the offspring develops, syphilised from the start, the mother being and remaining absolutely free from taint. This, as I say, is a popular fallacy. But it is incredible that the germ gain entrance into the spermatozoon, for the spermatozoon being nucleus and flagellum, and scarce anything more, has not the means of ingesting foreign bodies, while we have not a shred of evidence that the syphilitic germ is amœboid and capable of making its way into the spermatozoon.

It is likewise outside the limits of credibility that a virulent organism could be within the minute almost yolkless segmenting human ovum, lying latent in one or other of the cells, the products of segmentation. Such passages of pathogenic microbes on to the surface, and possibly into the eggs, may occur in insects, as Pasteur demonstrated, but the insect's egg contains relatively abundant yolk, and segmentation then may be little influenced by the presence of the micro-organism, provided that this be in the yolk. Even then, I doubt whether the embryo could develop properly, and am inclined to consider that a more reasonable explanation of Pasteur's observations upon the silk worm's eggs is, that at a relatively late period of their development those which come to maturity become tainted either from the surface or from other eggs which have been killed by the multiplication of the germs within them.

If the syphilitic virus gained entry into the unsegmented human ovum, its effects would surely be to lead to the destruction of the ovum. Fœtal syphilis must originate at a later date, and although syphilis in the parents may doubtless have its effects upon the ovum and spermatozoa of the same, and lead to constitutional disturbances in the offspring, progressive syphilitic lesions, the true syphilomata, in the fœtus and infant are *not* inherited, but are congenital, that is to say, acquired in utero after conception. Or in other words, inherited and congenital syphilitic lesions are two very different things. Thus to return to the main point, if the mother be without sign of syphilis, and the child be syphilitic, the only satisfactory explanation is, that the syphilitic virus has entered into the maternal organism and tissues, and has failed to induce any characteristic lesion at the point of entry, but has, nevertheless, through the placenta and chorionic villi gained an entrance into the fœtal tissues ; the process arrested

in the mother has been developed in the susceptible tissues of the child, and we have here an interesting example of the variability in the manifestations of the disease dependent upon the reactive powers of the tissues.

Were any further word necessary in support of this contention it would be found in the significant way in which the liver is affected in congenital syphilis. Extensive specific lesions of the liver in the acquired disease are relatively uncommon. They are the most common of all lesions in the congenital affection. As Chiari has pointed out, out of 144 cases of congenital syphilis examined by him, the liver was diseased (and that extensively) in 123 cases or nearly nine-tenths. Were the ovum infected it would be difficult to explain why the liver should thus be especially singled out. When we remember that this organ is the first to receive the blood coming by the umbilical vein, then if the infection originates from the placenta hepatic implication is the natural sequence.

The essential difference between such congenital, or ante-natal, and 'acquired,' or post-natal syphilis is, that in the former the virus passes immediately into the blood and so becomes disseminated through the organism, in the latter, the dissemination is delayed. The second stage of acquired syphilis, is the first stage of the congenital disease.

Again, although as a pathologist not in practice, I have not met with and am little likely to come across the condition, continuing the analogy between tuberculosis and syphilis[1] we must, I hold, admit the inherent probability of Kaposi's statement that it is possible to have a primary cutaneous syphilitic lesion, a true specific indurated chancre, not followed by any secondary effects. And further it is well established that women who have borne syphilised children and have themselves shown not a sign of primary or secondary manifestations may, years after, present unmistakable tertiary lesions.[2]

Up to this point, therefore, it may be laid down :

(1) That from analogy, as from clinical history and absence of any indications of the same, in sundry cases there may be an absence of the primary cutaneous or epithelial manifestations of syphilis.

(2) That individuals may fail to present either primary or secondary symptoms that are recognisable, and yet eventually develope definite tertiary lesions of the disease.

(3) That where the subject is relatively insusceptible, it is possible

[1] Every pathologist knows, many from personal experience, how frequent among those performing autopsies are cases of strictly localised cutaneous tubercles not followed by extension. Such primary cutaneous tuberculosis is characterised by its tendency to remain localised.

[2] Vide Finger, Arch. f. Dermat. u. Syph. 1890, p. 331.

that the disease may be limited to the primary cutaneous manifestation not followed by secondary lesions..

(4) That as with tuberculosis so with syphilis, the congenital form of the disease begins at what may be termed the secondary stage of the acquired disease, *i.e.*, the stage of general dissemination of the virus through the organism.

The Relationship between Secondary and Tertiary Syphilis.

I would now pass on to consider the relationship between the secondary and tertiary stages of syphilis.

Where in any infectious diseases we have widespread eruptions, affecting both skin and mucous membranes, we now feel assured that such eruptions are due either to the irritation set up by the actual presence and growth of the specific germs of that disease in the subcutaneous and submucous layers, or to the irritation produced by the products of these germs growing in other parts of the system. And the more we study infections of which we can isolate the specific microbes (streptococcus and pyococcus infections, typhoid, &c.), the more we find the first of these alternatives in force, and in the case of syphilitic eruptions, the fact that the cutaneous eruptions are infective, affords clear evidence that the specific virus is present in them.

Such generalised infections of the skin and mucus membranes can only be brought about through the agency of the blood stream, or, otherwise, what is termed the secondary stage of post-natal, acquired syphilis, is the stage of general dissemination of the virus through the system by the blood stream, and of the more immediate results of such dissemination. What has been described as the second period of incubation (the interval elapsing between the development of the chancre and the appearance of syphilodermiæ) is the period requisite for the virus to infect and traverse the lymphatic system on its way from the primary lesion into the blood stream, and then to proliferate in the cutaneous and other tissues up to such a point that eventually it produces a reaction.

It is usually held that the syphilitic virus now especially affects the skin and mucosæ, and that the abundant and varied crop of syphilides—of syphilodermiæ—are the peculiar sign of the second stage, gummatous and more fibrous growths being characteristic tertiary developments. Certainly the eruptions are the prominent features of the secondary stage, but it is too much left out of account that in the early stages of generalisation of the disease, the internal organs may be, and perchance often are, affected. And what I wish more especially to bring before you this evening is this lack of sharp definition

between the anatomical changes in early and late generalised syphilis. This lack is well shown by a study of the syphilitic liver; indeed, it is a study of several cases of syphilitic hepatitis which have been revealed in the post-mortem theatre at the Royal Victoria Hospital during the last four years, which has prompted me to select this more general treatment of the stages and forms of syphilis to bring before you this evening.

The reason why tertiary and secondary syphilis are regarded as so widely distinct is not difficult to comprehend. The disease is rarely directly fatal, especially now-a-days, and it is rarely that we obtain an opportunity to study the viscera during the earlier stages. As Jonathan Hutchinson has pertinently remarked: "The visceral pathology of the secondary stage might form a chapter in the history of syphilis which has not yet been written, and for which we possess few data. It is however, I feel sure, a great mistake to state that there are none to be obtained." In the address from which I take these words, an address which opened a celebrated discussion at the Pathological Society in London in 1876, he pointed out that abundant facts are on record to disprove the assertion that large gummata are not to be seen in the secondary stage. He noted that two cases of death from syphilitic disease of the heart which had come under his notice, had both occurred during the secondary stage and presented myocarditis with gummata, and in one of the two there were also distinct gummata in the spleen and in the testes, while he went so far as to state that the best example of gumma in the liver which he had encountered was in an infant.

To bring forward the evidence presented by the liver as to the identity of the anatomical lesions in the two stages, and as to the continuity or unity of the disease, it will be well to discuss the ways in which the liver is affected in syphilis and run over the different forms of specific hepatic lesions.

It is difficult to realise that scarce fifty years have elapsed since it was first clearly established that the liver is affected in any form of syphilis. The chapter in medical history bearing upon the liver in relation to syphilis is of some interest. Hutten and Fallopius and many of the earliest writers upon the morbus gallicus, held that syphilitic ulcers wherever appearing were the result of a corruption of the humours, the origin of which was to be looked for in the liver which had become diseased from the action of a volatile contagion. Others held that this organ was the first to be affected consecutively to disease of the genital organs. This was when every disease was regarded as due to a disturbance of the humours, and the liver being large and

of unknown functions, the severity of the disease almost naturally led to the liver being seized upon as the guilty party in syphilis When autopsies became more frequent the implication of the liver became seriously disputed : Paracelsus denied that it had any rôle in disease, stating that he had frequently found other organs affected, but had rarely met with any disturbance of the liver. Morgagni again in the middle of the eighteenth century, but expressed the views of his contemporaries when he denied any relationship between syphilis and hepatic disorders.

The Liver of Congenital Syphilis.

Only in 1848,[1] or according to Hutinel and Hudelo in 1847, was there published any serious study of the liver in syphilis, and then it was not the characteristic gummatous liver of the acquired disease, but the enlarged liver of the congenital condition to which attention was called. In that year and yet more fully in 1852, Gubler described the liver of congenital syphilis, pointing out its enlargement, its firmness and elasticity. He noted that the changes were often in circumscribed spots only, and here in these very earliest careful studies upon the subject, he pointed out that while the other lesions in the infant were of a secondary nature, the changes in the liver were of tertiary type and allied to the gummatous developments.

Time forbids that I should describe minutely the histological changes occurring in the liver of congenital syphilis. You will find them clearly stated in an article upon diseases of the liver by your distinguished confrère Dr. Graham, in the recently published Loomis-Thompson System of Medicine, an article which is far and away the best treatise on hepatic disorders by a single individual that has appeared in our language for many years. Suffice it to say that the affected portions of the organ present a combination of the development of minute, somewhat ill-defined collections of small round cells, which we know as miliary gummata, together with a wide-spread development of fibrous tissue, not only along the portal sheaths, but also spreading between the groups of liver cells which present more or less atrophy, in short, a condition of pericellular fibrosis. This fibrosis is in itself what we are accustomed to regard as a peculiarity of tertiary syphilitic manifestations. Yet here it occurs within a very few months of the primary lesion, when cutaneous eruptions and other secondary symptoms are abundant. And not only this, but at times we have appearances more closely resembling the gummata of the acquired disease.

[1] Gubler, Gaz. des Hop. Jan. 1848, and Gaz. Med. de Paris, 1852, p. 262.

What is the meaning of this general interstitial fibrosis, or more correctly, what is the series of changes which leads to its formation? It is difficult to state with precision. At times it appears to be wholly in excess of any development of the above mentioned miliary gummata. Indeed, it looks as though it had not been preceded by any characteristic syphilomatous lesion, and the peculiar manner in which the connective tissue development extends between the rows of the liver cells, and becomes pericellular, and *pari passu* the liver cells show evidences of atrophy, would seem to indicate that here we are dealing with, not so much the results of the productive granulomatous inflammation, as with a process of tissue disturbance set about by the diffusion throughout the system of the toxic substance generated by the virus. These toxines lead to the atrophy of the liver cells with synchronous development of connective tissue; in short, the appearances are largely, but by no means entirely, those of a replacement fibrosis.

A somewhat similar condition is occasionally to be met with in the kidney and that in the earlier stages of the disease. The only further point I need impress upon you here is that this generalised fibroid change may be developed in the earliest stage of the generalised disease, and by no means necessarily indicates a tertiary condition.

In some rare cases this extensive fibroid condition appears to be present with very little evidence of syphilomatous or granulomatous change in the organ. Marchand[1] has recently described and collected together about half a dozen examples of this condition. Curiously enough, this form of cirrhotic liver with atrophy in most of the cases has occurred in one of a pair of twins and that one still-born. It is also associated with evidences of profound hepatic disturbance in the shape of icterus. Marchand's cases are not wholly satisfactory so far as regards the history of syphilis in the parents, but, as he states, it is difficult to explain this remarkable condition of atrophy of the organ with extreme fibrosis, save on the supposition that the cases were syphilitic.

Coming now to the presence of gummata, Gubler noted in his earliest communications that scattered through the cirrhotic areas in the infantile liver were numerous fine paler flecks, which he likened to grains of semolina, and Virchow, studying these, spoke of them as miliary gummata. More and more evidence has accumulated as to the relationship between these minute, ill-defined tubercles or collections of small round cells, and the caseous gummata seen in the acquired disease. The relationship is identical with that between

[1] Ctbl. f. Allgem. Path., Vol. VII., 1896, p. 273.

miliary tubercles in the lung, and encapsuled tubercular caseous nodules in the same. In the great majority of cases, the liver of congenital syphilis presents the admixture of a diffuse pericellular cirrhosis and scattered miliary gummata. Sometimes the whole of the organ is affected, at other times the process is observed only in parts, either at the edges or in a portion of one lobe. In this latter case, one has circumscribed yellow masses sharply defined from the, in general, congested, but otherwise unaffected hepatic tissue. Sydney Coupland,[1] indeed, goes so far as to regard these circumscribed masses as enormous gummata. It is, however, open to doubt whether these masses strictly conform to our idea of gummata, although I must confess that it is difficult to define with precision what we include under this term. For myself I am inclined to regard them as more nearly resembling the large nodular syphilomata occasionally to be met with in the adult liver, where they may be so well defined as time and again to lead to the erroneous diagnosis of non-infective neoplasms.

There are frequent cases on record in which true gummata have been recognised in the liver within a few weeks after birth. Several French cases will be found quoted by Hutinel and Hudelo[2] in 1890, while Cohn in 1896 quotes several German authorities.[3] In English literature, I have found cases described by Canton,[4] (in an infant of two weeks, with numerous small gummata), Barlow[5] and Hutchinson (loc. cit.)

In Barlow's case the child showed no syphilitic symptoms until it was seven weeks old; it died five weeks later, and upon the upper surface of the liver were several depressed areas, varying in size from that of a pea to that of an almond, one having a slight tail-like prolongation; there were a few also upon the under surface. As he points out, from these depressions it is clear that the gummata were, as he terms it, *receding*, although it would seem clear that they presented no central caseous change.

From the above description it would seem that in the liver of the new born infant, presenting externally evidence of what is known as as the secondary stage of the disease, there may be several varieties of syphilitic manifestations :

1. Well defined gummata.
2. Admixture of miliary gummata, with generalised fibroid change

[1] Trans. Path. Soc. London, Vol. XXVII., 1876, p. 303.
[2] Arch. de Med. Experimt, 1890, Vol. II.; p. 509.
[3] Virchow's Arch., Vol. 146, 1896, p. 468.
[4] Trans. Path. Soc., Vol. XIII,, 1862, p. 113.
[5] Trans. Path. Soc., Vol. XVII., 1876. p. 292.

not affecting the whole organ but forming relatively large circumscribed areas.

3. Admixture of miliary gummata and generalised fibrosis affecting the whole organ, which is in consequence enlarged.

4. Generalised "atrophic" cirrhosis without much evidence of gummata, but associated with icterus, œdema, etc., the organ being granular and definitely contracted.

In other words all the changes seen in congenital syphilis are those which ordinarily are considered to characterise the tertiary rather than the secondary stage of the disease.

There is yet another form of congenital syphilis which has to be noted; the form termed "Syphilis hereditaria tardiva" or more correctly, delayed congenital syphilis. Several examples of this delayed syphilis are on record; it is to be made out from them that frequently the cutaneous changes may not show themselves for years after birth. Taking up more especially the English literature upon the subject, Henry Morris [1] has described a case of a girl aged 20 with marked syphilitic family history and evidences of interstitial keratitis, who had suffered from sore throat at 12, enlargement of the liver at 18, ascites at 19, and at the autopsy, the liver was found much puckered and deeply fissured as well as altered in shape, while there were several large gummatous nodules imbedded in different parts of the organ. Wills[2] records a case that seems somewhat more definite, in which a male, aged 22, presented a small dense liver with thickened capsule, which was constricted into lobules by numerous thickened bands of connective tissue, a condition which he could only explain as being due to congenital syphilis. Bristowe describes a condition of gummata in the liver of a boy of 15 which gave way to treatment with potassium iodide, a condition which he could only regard as an excellent example of the results of delayed congenital syphilis; and Osler, in his well known Lectures on the Diagnosis of Abdominal Tumours, gives two cases which are similar, one of his own, the other which had been recorded by Dr. A. C. Wood, in the University Medical Magazine, Vol. II. Both of these cases were in boys of 13, presenting clear evidence of congenital syphilis; in both there was the enlarged irregular liver which diminished under the action of potassium iodide. A further case in a male, aged 22, has just been published by Post and Councilman.[3]

It is clear then that congenital syphilis, like the acquired disease, can manifest a tertiary stage long years after infancy, and that the

[1] Trans. Path. Soc. Vol. 21, 1870, p. 214.

[2] Liverpool Medico-Chirurgical Journal, July, 1892.

[3] Boston City Rep., 1898, p. 233.

tertiary symptoms when thus delayed are liable to be of the gummatous type with cicatricial contraction. In other words, congenital syphilis may in every respect follow a course identical with that seen in the acquired.

The Liver of Acquired Syphilis.

Passing on now to consider the syphilitic manifestations in the disease of post-natal acquirement, it is very interesting to notice that whether we are dealing with cases in which death has occurred within the first year after the disease has been communicated to the individual, or whether we obtain the liver long after the acquirement of the disease, the morbid changes are of the same order, the only recognisable difference being that the longer the time that has elapsed after infection, the greater is the tendency to the development of cicatricial changes with contraction and deformity of the organ.

Taking up the hepatic disorders in series, perhaps the earliest disturbance that has been noticed with common frequency is the development of icterus in the early secondary stage. The association has been noticed by several writers. Hilton Fagge, writing in 1867, was able to quote Portal, Ricord, Gubler and Lancereaux upon this point, and noted that Lancereaux alone had collected 21 other descriptions of the connection, and, intermittently, observers have since described the association which is now generally recognised. The last writers upon the subject are Neumann, Joseph, and Uhlmann.

Inasmuch as patients have very rarely died in this stage, it is impossible to state with precision what is the condition, but by analogy with what occurs in the infantile liver, it may be suggested that there is here a generalized toxic disturbance of the organ, with catarrhal hepatitis, which may or may not lead on to the generalised fibroid state which has been described in connection with the infantile liver. Indeed, Hilton Fagge[1] has recorded a very interesting case of what he terms yellow atrophy of the liver consecutive to a diffuse change in the organ due to acquired syphilis. The case is that of a female of 23, in which there was a history of syphilitic rash with falling off of hair, and macular syphilides. Jaundice appeared to be of the obstructive nature. The patient became drowsy, then unconscious and comatose. At the autopsy, the liver weighed 46 oz., and was of an opaque bright yellow colour, and of dense consistence. The surface was mottled, the left lobe resembled very closely that of the infantile syphilitic liver; it was pale and semi-pellucid, and the parenchyma was replaced by connective tissue; there was no amyloid reaction.

[1] Trans. Path. Soc., Vol. XVIII., 1867.

Here then we have the jaundice, associated with the generalised atrophic condition of the organ, which in the absence of full microscopical description, may be taken to be either closely allied to the atrophic form already mentioned in the infant, or to the general pericellular fibrosis with miliary gummata seen in same. Without deciding positively to which form this case belongs, I would point out that generalised change in the organ is more common than I think is generally recognized.

The gummatous syphilitic liver is so characteristic and so well-known a form of hepatic disease, that now-a-days, it is rarely described, save from previous authorities, and thus in general, once gummata are recognized in the liver not much further interest is taken in the case; thus the old classic descriptions and ideas are perpetuated.

It is generally laid down that in tertiary syphilis affecting the liver, gummata, whether well marked and caseous, or the cicatrised remains of such with well-formed stellate surrounding of fibrous bands are the characteristic changes in the organ, while a condition of generalised and pericellular cirrhosis is wanting. It must, however, be remembered that even years after the primary infection such cirrhotic change may be recognisable, and not a few cases are on record of such a condition.

In three out of eight cases of tertiary syphilis affecting the liver which have come to the post-mortem room at the Royal Victoria Hospital during the past four years, there was clear and fairly extensive pericellular fibrosis along with gummatous change. The fibrosis, it is true, was not generalised over the whole liver, the condition more nearly resembled a condition of circumscribed fibroid change seen in the infantile liver.

These three cases are of some interest as throwing light upon the hepatic changes. In one case there was no clear history given of the date of infection.

CASE I.—Male, æt. 35. The patient became anæmic and emaciated in April, 1895, œdema and ascites supervening, and death occurred early in July. There was albuminuria and the œdema of the legs had been complicated with an erysipelatous condition which seems to have been directly caused by the hepatic disturbance, for the heart was fairly normal, and the kidneys although large and white, showed scarce any sign of interstitial change. The liver showed numerous large deep stellate cicatrices with some diaphragmatic adhesions. The organ in general was soft with advanced fatty nutmeg condition. Sections through the cicatrices showed a characteristic gummatous appearance with large bands of fibrous tissue running deeply. Microscopically, there was a considerable amount of pericellular cirrhosis in the neighbourhood of the gummata with much small-celled infiltration. The spleen was large, firm and congested. The stomach presented diffuse submucous hæmorrhages.

I shall return later to consider this condition of small celled infiltration. Case V. to be referred to later showed a similar condition.

In another patient of Dr. Stewart the condition was more widespread.

CASE II.—The patient aged about thirty-seven, was infected 14 years before his death, and, although himself a medical man, had never undergone proper antisyphilitic treatment, on account of the intense gastric disturbance induced by mercury and potassium iodide. Four years after infection he suffered from gastritis and diarrhœa alternating with constipation and a condition of gastric disturbance and general malaise which continued at intervals for the rest of his life. It is to be noticed that there was a frequent development of a febrile temperature. Ascites and jaundice supervening in the middle of October he was tapped; the fluid collected again, and he died in the beginning of November.

At the autopsy, there was no sign of old ulceration or chancre; there was well marked jaundice; the heart was healthy in appearance, though there was moderate atheroma of the aorta. There were dense bands of adhesions between the diaphragm and the abdominal wall, and the liver was profoundly contracted and small, scarcely passing beyond the middle line. It had a puckered and coarsely nodular surface. The right lobe was especially contracted presenting frequent and well-marked gummata with surrounding cicatrices so that the surface was broken up into numerous small nodules of the large hobnailed type. On section there were numerous white gummata from two to four mm., in diameter scattered through the liver substance, and in addition there were relatively large areas of fibrosis here and there throughout the organ. The spleen was large, turgid and smooth, weighing 570 grammes.

Upon microscopical examination the organ showed well-formed caseous gummata with a zone of surrounding congestion together with a very general advanced interstitial fibrosis, somewhat irregularly distributed, in addition to the ordinary fibroid bands of tertiary syphilis. The bile capillaries were richly injected with inspissated bile, and the kidney showed a condition of parenchymatous nephritis.

The interest in this is, that here we have presented to us a progressive syphilitic disturbance not arrested to any extent by specific treatment. The amount of change in the liver was extreme, and as above mentioned, consisted in the development of numerous gummata, pericellular fibrosis and catarrhal hepatitis, with jaundice. Indeed, we have here an example of syphilitic infection of the liver in an active stage fourteen years after primary infection. The appearance of the liver in this case appears to throw light upon what is the true nature of so-called tertiary syphilis. This is not by any means a receding process. While the tendency of the disease is, as it were, to burn itself out, and while in the majority of cases, if properly treated, the virus is completely destroyed in the secondary stage, nevertheless there may be persistence of the virus, and under favourable conditions the disease may light up again. Here, in this case, the patient enjoyed very fair health from 1888 to 1893. Gastric and intestinal functions were well performed; he increased in weight, and led an active life. During this time the process was certainly arrested, then the malaise and indigestion returned, and the ascites and jaundice which supervened can only be ascribed to the progressive development of syphilitic disturbance in the liver.

The analogy between syphilis and tuberculosis in this respect is perfect. In about 30 per cent of our autopsies we come upon evidences of old tubercular cicatrices in the lungs, and in the majority of these cases the process is undoubtedly wholly arrested; thus we are dealing with healed tuberculosis. In some however, we see that the virus is still present in the encapsuled caseous masses, for we can in a certain number of cases cause the disease in guinea pigs by inoculating into them the caseous contents of the old tubercles, and again in some cases we can recognise that these tubercles have been obsolescent and not obsolete, for around them we can make out progressive tuberculosis evidently originating from them. Speaking of these cases in the language which we employ to syphilis, we might describe them as being examples of tertiary tuberculosis, or to put it otherwise, we include under the term tertiary syphilis, two conditions:

1. The cicatrices and fibroid changes which are indications of a previous syphilis now healed and obsolete.

2. The lighting up again of an obsolescent syphilis from old foci in which the virus has remained latent.

The two cases just recorded are examples of the latter condition, while the case which follows is one of several examples of the former:

CASE III.—Male, æt. 49, who had led a wandering life in the South American States, Pacific Islands, and over the world since 25. Had suffered from all the diseases of childhood, gonorrhœa at 18, stricture five years later, yellow fever while serving in an American Army, small-pox and malaria when he was 19, had chronic dysentery about six years ago, was a heavy drinker of spirits, and had been a soldier, a sailor, and of late a backwoodsman; he denied having had a chancre. The cause of death was acute lobar pneumonia of the upper lobe of the right lung with purulent peritonitis.

The liver was deformed, weighed 1960 grammes, and showed four large areas of puckering on the upper aspect of the right lobe. A rounded mass 3 cm., broad and 2.5 cm. long projected from the lower end of the right lobe, the truncated end of which was characteristically puckered; the appearance of the organ was typically syphilitic. The intestines showed no signs of previous dysentery.

In this case, the section through the puckerings showed singularly little fibrous tissue despite the extreme contraction and deformity of the region where they were present. There were no proper gummatous areas to be made out, and the only satisfactory explanation is that in this liver which presented so obviously the appearance of tertiary syphilis there had been complete or almost complete absorption of the syphilitic deposits.

It may be suggested that the resemblance between tuberculosis and syphilis which I have thus emphasised is imperfect, in that when tuberculosis once affects the organ, the virus always remains latent in that organ, and there is not the complete absorption which we must acknowledge takes place in the majority of cases in syphilis. For

it is admitted that at the most (and that in untreated cases) only 30 per cent. of those infected show tertiary symptoms. Among those treated the percentage is only about 10 per cent. But this is another modern popular fallacy: there may be complete cure of tuberculosis and complete disappearance of the tubercles even when they have become distinctly fibrous. This is proved by the experimental infection of dogs with peritoneal tuberculosis and arrest of the process by repeated laparotomy. I need scarce remind you that it also has been recognised in some cases that there may be arrest of tubercular peritonitis in man by similar means. Definite cases are on record in which there has been a complete disappearance of well marked fibroid tubercles from the serous coats of the intestines. The analogy, therefore, between tuberculosis and syphilis must be regarded as complete in this respect.

Rarely we come across a syphilitic liver showing very clear evidence of the progressive development of the hepatic condition.

Case IV. Such a case have I met with in a male of 28, who entered the Royal Victoria Hospital under Dr. Stewart with a rupture of one or more branches of the middle cerebral artery, and who had two years before been treated by Dr. James Bell for syphilis. Whether the syphilis then was primary, secondary, or of later manifestations I cannot ascertain, for the patient died before his history could be elicited. In this case there was syphilitic inflammation of the ventricles of the brain, and early atheroma of smaller arteries. The liver showed three or four puckered scars, and microscopically, fairly frequent gummata, with giant-cells and small localised infiltrations of leucocytes. There were no signs of tuberculosis anywhere, and the sections of the liver stained with carbol-fuchsin did not show any tubercle bacilli. In this case, the puckered scars indicated gummata which had undergone cicatrisation and fairly complete absorption. The small infiltration of leucocytes can only be regarded as miliary gummata resembling in every respect those seen in the infantile liver of congenital syphilis. The case must be regarded as one in which, as shown in the ventricle of the brain and in the liver, the active syphilitic process had been rekindled or had progressed with rapidly fatal results.

Case I. was probably an example of the same condition.

The only manifestation of syphilis in connection with the liver which is to be found in the acquired and not in the congenital form is the condition of perihepatitis. I have not come across or met with in literature any indications of the development or presence of such a condition in the newly born child; in the adult, more especially at the late stages, it is not very uncommon.

Out of the eight cases, I came upon it in a fairly extensive condition in a female of 62, in which syphilis must have dated back for 20 years, more or less, for she gave a history of having seven children, of which five were miscarriages, and the other two died in infancy. Here the most extensive syphilitic changes were in the neighbourhood of the longitudinal fissure and round the gall-bladder. An interesting point was the fact that a mistaken diagnosis was made of atrophic

cirrhosis with ascites. The capsule of the organ was throughout thickened, the upper surface very smooth, the abnormal lobulation and puckering. Near to the gall-bladder in the right lobe was a caseous nodule, the size of a filbert, showing some calcification on section.

Upon section, the organ showed a thickened fibroid capsule, many small central scars, caseous gummata, and more or less diffuse and apparently recent fibroid change. Here it should be added that during the last five months of her life, she had been repeatedly tapped, and following upon tapping, there was found at the autopsy a condition of sub-acute peritonitis with inflammatory lymph covering the intestines. Thus the perihepatitis might not have been entirely syphilitic indeed I am a little doubtful whether syphilis pure and simple will lead to the condition of generalised perihepatitis.

Case V.—Female, æt. 62, with a history of having had seven children, of which five were miscarriages and the other two died in infancy. The husband said to be phthisical. In 1895, Dr. Roddick removed an epithelial wart, which on examination was found to be non-malignant. Suddenly upon July 31st, 1895, while the patient was feeling in good health she had an attack of hæmatemesis which was repeated next day and again two days later. It was accompanied by melæna and great weakness.

Upon admission to the hospital in August, 1895, under Dr. Stewart, the patient was sallow and there were dilated venules on the face. The liver extended from the fifth rib, two and a half inches downwards; it was not palpable nor could the spleen be felt; there was a low systolic murmur. The urine was high coloured with a trace of albumin. While in hospital epistaxis occurred once or twice. This history together with the progressive loss of flesh, the subicteroid tinge, the abdominal swelling with epigastric pain, the slight œdema of the legs, led to a diagnosis of atropic cirrhosis of the liver; 270 ozs. of fluid were removed from the abdomen, and after removal the fulness in the epigastrium was found to be due to the hard large mass passing down to within 8 cm. of the navel. This mass, hard and rounded, was continuous with the right lobe of the liver. The fluid re-accumulated and the patient underwent numerous tappings. She became progressively weaker, and the hepatic tumour appeared to be undergoing progressive diminution.

After having left the hospital she was re-admitted early in January 1896, dying upon January 24th. The condition was thought to be one of atrophic cirrhosis with enlargement of the left lobe.

Autopsy.—Œdema of the legs; extensive ascites with distended abdomen. Upon opening, the visible intestines were found coated with lymph; the omentum which was fatty and œdematous was adherent to the parietes in several places. The liver presented numerous adhesions, especially to the intestines and surrounding organs, specially in the region of gall-bladder. The right lobe did not reach the costal margin, the left extended just below the xyphoid. The organ weighed 1425 grammes, its greatest breadth was 22 cm., the right lobe especially being contracted (breadth 10 cm.); this lobe was also diminished from above downwards, its length being 15.5 as compared with 19 cm., of the left lobe. The capsule was thickened; the upper surface fairly smooth with, however, linear indentations, but the under surface presented extensive abnormal lobules especially in the region of the gall-bladder and upon the right lobe, so that several small lobules of liver tissue were produced, and near to the gall-bladder a caseous nodule the size of a filbert showed some calcification upon section. The under surface of the left lobe showed numerous cicatrices, some distinctly stellate. The largest was close to the longitudinal fissure and was 3 cm. in diameter.

Upon section the organ showed many small central scars, caseous gummata and *more or less diffuse and apparently recent fibroid change*. There was further a moderate amount of chronic passive congestion and cholelithiasis with obstruction of the cystic duct. The organ gave no amyloid reaction, and the spleen was large, firm and tense, with thickened capsule. In addition there was fat necrosis of the pancreas, brown atrophy of the heart, moderate atheroma of the aorta, slight chronic pachymeningitis, emphysema and bronchitis.

It will be noticed that the most extensive syphilitic change was in the neighbourhood of the longitudinal fissure and round the gall-bladder.

Yet another case of syphilitic liver with great thickening of the capsule showed this same complication of peritonitis. In this case (a patient under Dr. James Bell) there had been recurrent attacks of appendicitis during the last four years, with much chronic fibroid typhlitis, and the patient died of acute peritonitis following operation. Although the liver showed numerous stellate scars with abnormal lobulation, these were on the upper surface and there had been no signs of hepatic disturbance. There were in addition moderate arterio-sclerosis and fibroid syphilis of the left testicle.

CASE VI.—The patient, a male, entered for recurrent appendicitis and died of acute peritonitis following the operation Feb. 27th, 1896. Four years previously had had illness with symptoms of appendicitis. For five or six weeks before admission had dyspepsia, followed by right iliac pain beginning about Feb. 7th, kept at work till the 12th. Operation: Purulent appendicitis with perforation, followed by general peritonitis and death.

Autopsy.—Acute peritonitis with evidence of much chronic fibroid typhlitis. Liver large, surface very irregular, owing to great irregular thickening of capsule. Weight 2000 grams, numerous large superficial stellate scars with abnormal lobulation; greatest thickening about longitudinal ligament in front. On section, fairly firm, a few small internal cicatrices; portal vein and vena cava free; periportal glands free. Liver showed large periportal fibrous glands with thickened arteries and atrophy of some liver cells. Spleen, large 155 grams, 12.5 x 6.5 x 3 firm, soft and rather pale. Pancreas, hæmorrhages and necrosis. Other organs showed evidences of the acute febrile disturbance with moderate arterio-sclerosis and its effects; there was in addition fibroid syphilis of the left testis.

In the case of a male, æt. 75, the organ had the characteristically puckered appearance and showed upon the anterior surface of the right lobe a large convex cartilaginous plate, 9.5 x 7.5 cm., or about 4 x 5 inches, associated with chronic thickening of the capsule, together with numerous small cartilaginous plates elsewhere; the left lobe did not show much thickening of its capsule. The spleen, however, as is the rule in chronic perihepatitis, presented a much thickened capsule. (In this case there was very extensive gummatous hepatitis with practically no clinical evidence of affection of the organ.)

CASE VII.—Of this patient, a male, æt. 76, under Dr. Jas. Bell, with cancer of the tongue and general arterio-sclerosis, there is no history bearing upon the date o infection.

Autopsy.—Emaciation; rheumatoid arthritis of fingers; slight icterus; retracted abdomen, abdominal cavity dry; liver 1335 grams, 21 x 16 x 8 cm, right lobe anteriorly and inferiorly broken up into several small lobules; at hilus five or six small accessory lobules between a bean and a walnut in size. On anterior surface of right lobe,

left superior angle, large convex cartilaginous plate 9½ by 7½ cm. associated with chronic thickening of capsule. Numerous small cartilaginous plates elsewhere on the right lobe, the left also broken up into several lobules. On section, organ fairly friable with here and there small fibrous strands of tissue intersecting collections of lobules. Spleen, capsule very much thickened, weight 240, enlarged 10.5 c 7.5 x 5 cm. On section, soft, with slight fibrosis. Other conditions: Cancer of tongue, arterio-sclerosis, sclerosed heart valves and coronary arteries, hypertrophy of prostate, hydronephrosis, chronic interstitial nephritis, etc. (This case again shows very extensive gummatous hepatitis with singularly little clinical evidence of the liver disease.)

In two other cases, there were localised adhesions between the organ and the diaphragm. These adhesions, however, bore no clear relationship to the areas of cicatrisation in the organ.

On the whole therefore I am inclined to regard diffuse chronic perihepatitis as more a complication than a direct syphilitic manifestation.

There is one further condition of syphilitic disturbance of the liver in the adult to which I have only referred in passing, and that is the development of large tumour-like outgrowths of the organ which are so sharply defined that clinically the erroneous diagnosis may easily be made of malignant neoplasm. These growths are distinct from the bosses produced by the cicatrices of old gummata and subsequent deformity of the organ, and under the action of potassium iodide, they appear to undergo fairly rapid absoption. Osler in the work already cited has described very clearly one of these cases. So far as I know, in the coarse lesion produced, there is nothing quite similar to this seen in congenital syphilis. Microscopically however, the mass shows a condition not unlike that seen in the circumscribed areas of fibrosis in the infantile liver. There may or may not be cascous or gummatous change in the centre. There is however, much central fibroid change with complete destruction of hepatic parenchyma, and towards the periphery there is a more vascular connective tissue area in which strands of liver cells are to be made out. The nature of the growth and the presence of these liver cells would appear to indicate that what we have to deal with in such cases is a focal syphilomatous change in which, as the process extends from the centre, there is, at the same time, a constant proliferation of the liver tissue, and thus gradually, there is the development of a localised mass of new tissue, in the main fibroid, but at the periphery, where the change is not so extreme, there is growing liver tissue.

With this I have, I believe, mentioned the main manifestations of syphilis as they occur in the infant and in the adult, and it will be seen:

In the first place that the lesions occurring in the congenital and

the acquired disease, are identical, and brought about by the same process or processes.

That whether we have to deal with the disease in the secondary or in the tertiary stage, the same processes are at work. That, if we except those cases as truly tertiary in which we have to deal merely with the fibroid remains of obsolete gummata, and again those cases in which there is perihepatitis (which perihepatitis appears to be a complication rather than the genuine and direct result of syphilis), then we are bound to admit that the study of the liver alone would indicate that no sharp boundary line can be made out between secondary and tertiary syphilis. No more can we make out such a boundary between secondary and tertiary tuberculosis.

While I and all others must admit the utility of recognising these two stages, from an anatomical and histological standpoint one is forced to acknowledge that progressive syphilis is characterised by the same succession of phenomena whether it be studied but a few months or long years after the primary infection. Anatomically and histologically there is no valid distinction to be drawn between secondary and tertiary syphilis.

It may be asked whether such a conclusion is not wholly at variance with clinical opinion and experience? Upon the face of it, it is—but if the subject be looked into carefully, I think that such a view will reconcile not a few of the divergencies existing among syphilologists. We have those (and they are the majority) who state that tertiary syphilis is non-infectious, and those who bring forward clear examples of the production of infection five or ten years after primary inoculation of the disease. This difference can be reconciled if we agree upon the following points:

1. That now-a-days, under proper treatment, syphilis, if not a self-limiting disease, is at least a disease which can be healed, so that many of the lesions recognised as being tertiary syphilis, are truly the indications of the old healed syphilis, and not signs of progressive or latent disease.

2, If the disease has not completely died out and remains latent, the resistance of the tissues of the organism is such that in the majority of cases if it does not tend to light up again; there is so considerable a local reaction, that the infection and consequently the spread of the process tend to remain strictly localised, and the germs (which are probably of bacillary nature) do not become disseminated through the blood. Thus neither the blood nor the secretions contain the virus.

3. In a very small number of cases, the reaction on the part of the

tissues may be so lessened, and the virus retain or gain so high a virulence that either it causes ulceration, or in other ways becomes disseminated and capable of causing infection even late in the tertiary stage.[1]

[1] After reading this paper, Dr. C. A. Temple of Toronto, brought to my notice an interesting example illustrating this point which he very kindly permits me to note here. The patient, a vigorous blonde, contracted the initial lesion five years before marriage, underwent mercurial treatment for four years, and later was treated by Fournier's intermittent method, generally for from six to eight weeks twice a year. His wife a highly educated and cultured woman never showed any symptom of syphilis. In the fifth year of marriage she became pregnant for the first time, and, ten years after the primary infection of the husband, she aborted at the seventh month, the fœtus showing the typical facial characteristics and a greatly enlarged liver. The husband had always tended to have syphilitic eruptions if he neglected treatment, and when last Dr. Temple saw him, after the abortion, there were scaling circinate lesions on the abdomen and groins and ulcerating patches, some ecthymatous, others healing, on both legs from the knee downwards.

HOSPITAL ABUSE.[1]

BY

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Hospital abuse is a familiar term in British and American cities, and not unheard on the Continent. A different signification, however, attaches to the term now, than did in the early part of this century. Formerly, the term "hospital abuse," "asylum abuse," "prison abuse," intimated that the treatment in the institutions was unkind, inefficient, unscientific. At present its meaning is altogether changed. The term hospital abuse has come to mean, when applied to the public, the seeking and acceptance of medical treatment from a hospital, by persons who are able but unwilling to pay for the same, and when applied to a hospital, the treatment without any or only a nominal charge, of those who are not fit objects of public charity.

Objection is raised by the giving public—the hospital supporter, that monies donated by them are improperly applied, and by the medical profession, that hospitals, supported by the charitable public, by treating free of charge, people who are able to pay, interfere with their earning a livelihood for themselves and their families, and both classes urge that the result is a lessening of self-respect and a lowering of the moral tone of those patients who are well-to-do.

Hospital abuse is a growing evil and has come to be one of the social questions of our day. The welfare of our profession is being seriously affected by the growing popularity of the out-patient departments of our hospitals. Complaint is made by members of our profession in many large English as well as American cities and the responsibility for the evil put upon Hospital Boards, Hospital Medical Staffs and the public. It is difficult, in fact impossible, to make very definite statements as to the proportion of people attending out-door departments, who are able to pay a moderate fee. One English writer puts it as high as 40 per cent. The New York State Board of Charities for the year 1897, reports as follows: "While statistics are often misleading and deficient, there are good grounds for believing that nearly 50 per cent. of the population of New York City obtains practically free medical treatment. Dr. Wiggin makes the following

[1] Read before the Montreal Medico-Chirurgical Society, May 9th, 1898.

statement in an article published in the Philadelphia Medical Journal, April 23, 1898, that, prior to 1870, the number of applicants for medical and surgical relief bore only the ratio of 1·5 per cent. to the total population, as against the present estimate of 50 per cent. and the number of applicants is increasing every year. In Montreal we are suffering from hospital abuse but to a lesser extent probably than in the large centres in England and in the United States. I am not aware of the existence of any statistics that I can place before you but from conversations had with the members of the different hospital staffs and with general practitioners not connected with hospitals, I am convinced that the evil of hospital abuse is with us, and in very considerable proportions. A physician told me a few weeks ago that he had visited the out-patient department of one of our city hospitals, and to use his own words, "fully half of the women present as patients were better dressed than I could afford to dress my own wife, and there were three fine seal skin jackets in the room." The evil is not limited to the out-patient department alone. In the public wards people are seen who are known to be worth from ten to a hundred thousand dollars.

Now let us try and find out if we can some of the conditions of things which have led up to this unhealthy state.

First, I should say is the increased efficiency, comfort, attractiveness, even luxuriousness, of the modern hospital-ward. Hospital architecture is now almost perfection. The public wards of a modern hospital are warm in winter, cool in summer, well lighted by day and night, perfectly ventilated, the beds are about the best, the food abundant and of excellent quality, and the nursing such as only the wealthy can afford in their own homes. What more could any one desire, except the exclusiveness of a private ward. It is not to be wondered at that people flock to these hospitals, particularly when they are invited to come "without money and without price."

Secondly, the rapid increase of hospitals in number and amount of accommodation. The wealthy and liberal have felt that money given to a hospital, almost any hospital, was well bestowed. The hospital when built and equipped must of course be filled, and but little care has been taken to determine the suitability of applicants for public charity. Hospital managers have apparently taken it for granted that the old time honour and independence of character still obtained, and that the willingness on the part of an applicant to accept free hospital and medical charity was sufficient evidence that they were needy and poor.

Thirdly, the increased number of hospitals has given rise, very

properly and naturally, to a laudable rivalry, each one endeavouring to attain the highest possible standard of efficiency and usefulness. But emulation has not stopped here. Hospitals have become anxious to excel also in the amount of work done, and in the number of patients treated, until there has grown up almost as much competition among them as among physicians for patients to treat.

Fourthly, the advances in medicine and surgery during the past twenty years have increased the expense of medical and surgical treatment. I need only to point out the increased expense of nursing. The perfection of nursing now attained has rendered apparent the want of it when it is not to be had. A physician to-day feels that he cannot do justice to a case of typhoid or pneumonia without a trained nurse. Trained nurses cost more than people of only moderate means can afford, if the illness is at all prolonged. Hence another reason for sending to the hospital. In surgery the same holds true to even a greater extent. For a major operation there are needed proper preparation of room, an anæsthetist, one or two assistants and one or two nurses. Thus it is seen that the dividing line between those who can and those who cannot pay is a moveable line and families who could afford satisfactory treatment in their own homes twenty years ago cannot do so on the same income, to-day.

Fifthly, the spirit of the times in which we live. This is essentially a commercial age. The acquirement of wealth, the love of luxury and display, are the gods worshipped by large classes in the community. People will buy pianos, bicycles and good clothes, who leave their doctor's bill unpaid or go to the hospital for free treatment when sick. And they will unblushingly accept medical charity before any other. True they can put up with many of the necessaries of life that are not the most expensive, and equally true that when ill they cannot afford anything but the best medical treatment and the best nursing, but this is not the principle that governs the actions of the class to whom I refer.

Sixthly, the starting of the modern abomination, the private dispensary and hospital, by members of our profession for purely selfish and personal reasons.

Lastly, hospitals dependent upon the public for support, must, to avoid alienating sympathy and subscriptions, sometimes receive into their wards those who are very well able to pay. For instance, a large corporation, a large employer of labour, skilled and unskilled, contributes a handsome annual donation. This corporation naturally expects that their employees when injured or sick shall receive prompt and proper attention. But when one of their foremen, earning per-

haps several thousand dollars a year applies for treatment, the attending staff sometimes feel that the annual donation should not be expected to secure free treatment for one so well able to pay. This foreman very likely is earning more, many times over, than the physician who is expected to minister to his needs. However, if objection is raised, a perfect whirlwind of correspondence is started, to be followed by explanations, and perhaps even apologies. This is not right, and I hold that it is quite within our province to object, and to try to promote views more in harmony with equity and justice to all both physician and patient.

Now, I think it will be evident to one and all that the blame for the evil of hospital abuse must be divided. It rests not wholly on the hospitals, nor on the medical staffs of the hospitals, nor on the public. It is an outgrowth of the conditions under which we exist. How can it best be remedied? The medical profession takes great credit to itself for the existence of our beautiful and efficient hospitals, and rightly so. It naturally follows then that we are responsible, chiefly for the evil, concerning which we hear such loud complaints. As physicians, as citizens, we should stand united in trying to arrest this growing evil which is demoralizing in its tendencies and inimical to the welfare of our profession.

It will be quite evident to any one at all familiar with the workings of hospitals, that no one hospital can act successfully alone, in combatting this evil. There must be concerted action. If an applicant refused treatment at one out-patient department can get the same at another out-patient department without any inconvenient questions being asked, no lessening of hospital abuse will be likely to ensue.

The suggestion of the London Charity Organization Society as outlined by Sir William Broadbent, is worthy of consideration. The proposal in a word, is the formation of a Central Hospital Board. The Board to be composed of representatives of the Management and of the Medical Board of each hospital, together with a few general practitioners who are not connected with any hospital. A Board formed on these lines would be thoroughly representative. It would bring together the three great classes concerned, the hospitals, the hospital medical staffs and the general practitioners. It would probably be well to have the representatives of the latter class selected from districts. Each man would naturally be familiar in a general way with the population of his district, their earning power and their ability to get constant employment, etc.

The formation of such a Board would remove any feeling that might otherwise arise. It would do away with any misapprehension

on the part of the public, that the action of the Board was prompted by practitioners or hospital staffs at enmity with hospital work. This is very important. Such a Board could employ an inquiry officer, who could inquire into the circumstances of any cases referred to him, and report to the Central Hospital Board.

I do not think that any one would desire that the first aid to the injured should be interfered with. Our ambulances do a good and highly appreciated work in rescuing accident cases and people prostrated by heat, or suffering from poisoning, and bringing them quickly to the various hospitals where appropriate treatment is immediately available. But after the first aid is rendered those able to pay should be referred to the personal or family physician.

In regard to nursing, which is an expensive luxury—or more truly an expensive necessity in many cases, and one of the factors contributing to the increase of hospital abuse, the establishment of the district nursing by the Victorian Order of Nurses and by others will be most helpful. In this way the aid of the trained nurse can be obtained once a day or oftener, to give a bath, assist at a surgical dressing, or to instruct willing home helpers in the care of the sick.

One other point and I will close. I cannot but feel that hospitals should confine their ministrations to the poor. I believe there is no dispensary that does as much harm as the one that collects 5 cents or 10 cents from each patient. The pauperizing influence of that method in my opinion exceeds by far the out and out free dispensary. In hospitals, the acceptance of 50 cents a day from a patient for what costs over $1.00 makes a patient feel that he is under no obligation to any one, either the hospital or the medical attendant, that he has a right to what he gets because he pays for it, and that he is perfectly at liberty to return on any future occasion for treatment on the same terms.

In my opinion it would be better if the hospital ministered to the needs of those who could not pay anything and to no other. Private enterprise may be trusted to see that those who can pay are not allowed to suffer.

It is the pay patient and the private ward patients that set the fashion, and popularize the hospitals. Once the hospitals are popularized, as soon as it becomes "the correct thing" to go to the hospital when ill, the next step is naturally and easily taken, viz., to get there as cheaply as possible.

I have tried gentlemen, to put this question before you in a true, broad and generous way, and I hope that in any discussion which may follow, general broad principles only will be dealt with. It is a very

complicated question. There are many different interests to be considered, and only by avoiding anything that might give offence to any one of those interests can good be got out of this evening's deliberation. Our society is very representative. It is made up of members of hospital managing committees, hospital medical boards and general practitioners. None of us wish harm to our hospitals, and none of us wish to put any obstacle in the way of their efficient working. Let us strive together to evolve some scheme that will root out the tares without injuring the wheat.

ON MILK DIET IN TYPHOID FEVER.[1]

BY

H. R. MACAULAY, Student in Medicine.

At the outset I wish it to be clearly understood that I am basing my remarks more particularly on the routine practice of our larger hospitals, and so far it will, I think, be granted that the course of treatment pursued at our Montreal hospitals is in line with that employed in the hospitals throughout the medically civilised world. It is at the larger hospitals, the centres of clinical study, that the young medical idea is formed, and therefore any routine treatment there observed is apt to be very generally adopted by the profession at large.

I have thought it wise to confine my ideas to this "routine treatment," as in so doing I will have something tangible on which to base my remarks.

Practically the milk dieting at all these hospitals is the same. The patient is given at least three pints of milk during the twenty-four hours. This quantity of milk is given to the patient in two-ounce doses or more, at periods of two hours apart. If the patient is awake when the clock's hands point to the expiration of two hours he gets his milk. If he is asleep he is allowed to remain so, unless in very exceptional circumstances, as when the attending physician does not think he has taken sufficient nourishment.

Now for some statistics as to the results in cases that were so dieted:

In 1896 the Montreal General Hospital had 93 cases with seven deaths, and of these, *three* were due to perforation.

In 1897 to date, Sept. 1897, the Montreal General Hospital had 54 cases with *four* deaths, and of these, *two* were due to perforation.

During 1896 the Royal Victoria Hospital treated 60 cases. No deaths, but eight relapses.

I was unable to get the percentage of relapses at the Montreal General Hospital.

These results are certainly highly satisfactory, are they not? It is enough to make one wonder whether with such a small percentage of deaths in this disease, the acme of both scientific and dietetic treatment has not been reached, and whether the milk diet as prescribed is not such that it cannot be improved upon.

[1] Being the introduction to a discussion upon "Treatment in Typhoid Fever" at a meeting of the McGill Undergraduates' Medical Society, Dec. 3rd, 1897.

Now, before going further, I would wish it to be fully understood that I am not decrying the use of milk in typhoid altogether. That is not my position; far from it. Without milk we would be deprived of possibly our greatest boon as an article of diet for the sick generally, including those suffering from typhoid; but what I do contend is that our diet in a typhoid case should not be so restricted. That it should not be confined to milk alone, milk every two hours, to-day, to-morrow and the day after; nothing but milk this week, next week and the week following.

Let us consider briefly some points concerning cow's milk as a diet: I will not weary you with details as to its chemical composition; suffice it to say that it contains all of the elements necessary to maintain life—infant life. The infant thrives and grows upon an exclusive milk diet, but what about the adult—the young man and young woman with typhoid? They have advanced beyond the infant stage. When in health, previous to the attack, their food has been varied. Appetite and hunger have gone hand in hand, and what they have been accustomed to eat has been guided greatly by an acquired liking for this or for that form of food. Milk as a diet has been discarded long ago, and by a great many is either not taken at all or only as an occasional glassful at meal time

In such cases are we justified in reducing the bill of fare, and that at once, to an exclusive milk diet? Is milk the food *par excellence* in these cases to the exclusion of all others? To-day it is the generally accepted idea that it is. Such men as Prof. Osler, and others of light and leading in medicine advocate it, and our respect for authority is so great that if our thought has not been specially given to any matter, we naturally accept and follow their advice; for we know that in general we cannot do better.

Milk, the exponents of the milk diet say, is a perfect food, it is a complete food, so easily absorbed, so readily digested, not apt to cause gastro-intestinal disturbances; and is, therefore, the food to give above all others If I could concur with these statements there would be no one more ready to say "amen" to the dieting of all cases of typhoid with milk, and milk only, than I would be myself; but these statements I challenge.

Dr. Wm. Thompson (I refer you to the Text-book of Medical Sciences), in an article on milk, mentions that milk contains all of the elements necessary for maintaining life, but—notice that *but*—that these elements, more particularly the nitrogenous elements, and the carbohydrates, are not in proportions such as are necessary for adult life. Milk is rich in nitrogenous material, and not so rich in carbohydrates.

and, he continues, the system in health requires for daily consumption 18.3 grammes of nitrogenous material, and 3.28 grammes carbohydrates.

To get the sufficient amount of carbohydrates such a quantity of milk has to be taken that the nitrogenous elements are in excess, and Dr. Thompson goes on to say that, because of this inequality, if a healthy person be put on an exclusive milk diet there are apt to develop in a short time a great nausea, a great loathing for the milk, and very often gastro-intestinal disturbances.

Exponents of the present milk dietary may argue that in typhoid fevers there is a greater demand for nitrogenous material, and that, therefore, this inequality in proportion is greatly overcome, and, because of this, milk is not so apt to disagree with the stomach. This argument is good, and I would agree with it to the extent that I believe the fever, by increasing metabolism, has greatly modified the demands of the system, and made the stomach and system generally more tolerant of milk; but, after all, is it not merely a toleration? In the great majority of cases, on inquiry, do we not find the same nausea and the same loathing for milk that we are told a healthy person will develop if put on an exclusive milk diet; is there not a great desire for something else? I have found it so with the majority of those I have questioned.

We are told that more nitrogenous material is necessary in the fevered state. That is granted. But another question is: although needed, can the weak and diseased digestive and absorbent systems attend to the absorption of the same? That I very greatly doubt. Do not forget that from 86 to 88 per cent. of cow's milk consists of water, and in this connection we also know that the fevered system requires this water, and therefore this portion of the milk is apt to be readily absorbed. But what about the nitrogenous material? Is it so readily taken up? Theoretically, we are apt to think it is; practically, the typhoid system shows no affirmative sign. We daily see the patient becoming weaker and more emaciated for the first two or three weeks of the diet. To a certain extent it reminds me of the old adage, "one man may lead a horse to water, but ten men cannot make him drink." Of course, the progressive emaciation is due to the direct action of the typhoid virus; what I fail to see is that it is arrested or lessened by the exclusive milk diet; on the contrary, I am inclined to think that it is accelerated,

Confine the diet of a healthy, strong man to milk entirely, and what will be the inevitable result? Life may be maintained for some time, but with this infants' food the *strength* of the adult is bound to be reduced, and, reasoning from this, I cannot but believe that we, with

our exclusive milk diet to the patient, in reality help to bring about and keep up that typhoid condition of weakness which we see in all our typhoid cases.

Now let us look at another feature in this dieting, and see if we can agree with the milk diet as presented on this point. Among the first symptoms of typhoid fever are nausea, vomiting and loss of appetite. It would seem as if the system, knowing that a rest was needed for the whole of the alimentary tract, actually refused to digest what was already in the stomach, and, in the most emphatic way possible, showed to us that it demanded this rest.

In all acute inflammations rest to the part diseased is laid down as one of the cardinal requisites to a speedy cure. Notice how this rule is followed out in surgery and in medicine. Let any one of us seriously injure an arm or have an acute inflammation of any sort on the arm. Do we not immediately give that member rest? We do so for two reasons. The first and most imperative reason is because of the pain produced by movement; and another reason, but one not so much thought of at the time, is because we cannot use that arm as formerly. There is a loss of function of the inflamed member. Because of these two reasons we instinctively, of our own accord, give the diseased arm rest, absolute rest. The surgeon when called in, by his bandaging, helps us to keep it at rest, and thus assists and follows out nature's promptings.

One more example: In pleurisy we have another acute inflammation, with intense pain. Here we cannot give absolute rest to the inflamed surface, as we cannot cease to breathe. The interchange of gases carried on in the lungs is imperative to life and cannot be stopped, even for a few minutes, without dire results. But note the condition we find in pleurisy. As far as possible the sufferer keeps the abdominal muscles tense and breathes very superficially with the upper lobe, keeping the lower portion of the lung, where the pleuritic pain is most severe, as quiet as possible. By doing this he gives himself least pain. This is done instinctively, really without thought on our part. The physician recopying nature's plan of treatment assists her by strapping the side, so that expansion of that part cannot occur. It gets the rest needed.

Now, to return to typhoid. The seat of the trouble is in the alimentary tract. This is seriously diseased and has demanded rest—rest to both digestive and absorbent systems. It is no longer able to digest as formerly. It has actually refused to digest what was at the time in the stomach, and enforces this point by taking away the patient's appetite. Surely these indications tell us plainly enough

what course nature wants for the time being to pursue. Would we not be doing wisely in listening to her and helping her, as we do in both injuries, and other acute diseases such as I just referred to?

We must not forget that the patient's system has resources stored away to be used on just such occasions, and when the system tells us that she intends to draw on this reserve, because of the weak, diseased digestive tract, why not let her do so until the stomach rights itself sufficiently to demand food? That to me would seem the rational plan, and the plan in keeping with our other treatments, but for some reason, which the exponents of the milk diet will possibly explain, it is not the course pursued in the treatment of typhoid. In fact the reverse is the course pursued, and the unfortunate stomach is asked to attend to a dose of milk every two hours.

Just at this moment we might find it instructive to look into the condition of the stomach and see how far it is able to assimilate this milk. Dr. Beaumont, who was able to examine daily the stomach of a young man who had a fistulous opening into that organ, resulting from a gunshot wound, found that "in febrile diseases very little or no gastric juice is secreted, so that food taken at that time is not digested." Sir William Roberts, in his work on Digestion and Diet, mentions that "in the seriously sick there is neither pepsin nor acid in the stomach to curdle milk." This is the condition of the stomach into which the milk is put.

Milk in the healthy stomach, with normal secretions, curdles quickly, the casein forming into solid masses, which in turn should be broken up by the gastric juice. Without this gastric juice and acid, how is the milk to be digested? If not digested, of what benefit is it? What about nitrogenous elements which we are told are assimilated?

Or to look at the matter from another point of view. Milk normally in the healthy stomach takes three hours for complete digestion. I confess this was a poser to me when I read it. Milk normally takes three hours for complete digestion, and here to this diseased digestive tract we have it ordered every two hours! Surely we do not believe that the weakened system, and particularly the diseased gastro-intestinal tract, possesses increased digestive properties for the time being? This would be a "reductio ad absurdum."

Then, if we admit that the gastro-intestinal tract is particularly weak, as is really the case, are we wise in ordering milk every two hours? Before the one dose is disposed of the next is on top of it. I leave this thought with you, only wishing to impress on you the point that the stomach has demanded rest, that from practical examinations of a stomach in a febrile state, this rest is shown to be absolutely

necessary, the stomach not giving forth gastric juice and pepsin necessary to digest milk. That instead of this rest, milk is presented, a dose every two hours, while even in a normal condition the stomach requires three hours for its complete digestion. Remember these points and draw your own conclusions regarding the wisdom of this dieting.

To continue regarding the digestion of milk. We find that either in the stomach or duodenum (Sir William Roberts says in the duodenum because of pancreatic juice) it—the milk—is broken up into a whey and a casein, which latter is practically a tough cheese. This, in the weakened condition of the patient, is very difficult of digestion, and very, very little of the proteid matter is changed into peptone. These hard masses of casein mat together and pass down the intestine as one hard mass, constipating the patient, and requiring enemata for their removal in the great majority of cases; this, too, in spite of the lime water and soda which has been given to reduce the size of these masses of casein.

In connection with the amount of hard excrementitious matter given off by the milk diet, I should like to quote Rubner. He found that even in health an exclusive milk diet gave a greater bulk of fæces than an exclusive diet of roast beef or eggs.

Let me emphasize this point. All this casein is formed either in the stomach or in the upper part of the small intestines, so that as a bulky mass it has to pass over the particular site of the lesions. This surely should score a point in favor of other diet. Remember the irritable condition of the ileum, with its inflamed, projecting Peyer's patches, which may in places be ulcerating or sloughing. Remember this condition, and we cannot but admit that everything tending to bulkiness of excreta should be, as far as possible, replaced by a diet giving less bulky, and, therefore, less irritating fæces.

Another feature about milk diet, and one that should give us no uncertain index as to the condition of the stomach, is the condition of the tongue and teeth. Look at the coated tongue. Notice the sordes about the teeth, the very foul breath. Those conditions, bad at any time, are always most pronounced when the febrile state is highest, and are so offensive that the mouth has to be kept washed with an antiseptic wash. These features clearly indicate the stomach's condition. Yet, although we cleanse the outside of the cup, metaphorically, by cleansing the mouth, we take no steps towards remedying the condition of which this is merely a symptom. We merely advise another dose of the cause in two hours.

Occasionally, at the hospitals, there are typhoid patients with

whom milk disagrees at the commencement of their treatment to such an extent that the stomach rejects it. In these cases it is the rule to change the diet for a time at least. Here the physician recognizes the wisdom of attending to the dictates of nature, and some more suitable food is given until milk will remain on the stomach, and then the same milk diet is prescribed to him as to other typhoid patients. Here I think we may raise a question. Are we wise in following the dictates of nature in this particular case? Milk is given, the stomach rejects it, and the diet is promptly changed. I think we will all agree in the wisdom of making the change. But let us carry the course of reasoning a little further. Here are two patients. The stomach of one rejects the milk and his diet is changed. The other one is nauseated, but the milk remains on the stomach; he gets the milk diet. The difference in the nausea between these two cases has been one of degree, yet notice the difference in the latitude allowed to diet. Might we not rightly reason that if it is beneficial in the one case to give what best suited the stomach, that the same rule would be equally beneficial in the other case, where there was a loathing of the milk, although the nausea was not quite strong enough to cause a rejection? Look at this from an unbiased standpoint, and does it not occur to you that the dietary line is pretty tightly drawn in the case of the nauseated patient?

Another side to this question of following nature's lead presents itself, when, during some stage of the disease, the sufferer craves some particular food. I here refer to a real hunger, and not to a morbid, changeable fancy such as might be expressed when the cerebral functions are disturbed.

If we recognize and appreciate the wisdom of nature on the one hand in objecting to a diet that does not agree with the stomach, why should we on the other hand think she has grieviously erred in her desires. It does seem to me that just here a little more latitude might be allowed. It is merely following up the course of reasoning I commenced with when speaking of the stomach demanding rest. The weakened stomach is now able to distinguish between what is acceptable and what is not.

To a great many this may seem rank heresy. Yet, remember in all acute diseases, the nearer we get to nature's methods, and the more we help her in her ways instead of running counter to them, the more nearly are we getting to correct treatment. Let me illustrate my meaning. We do not have to go back so very, very far before coming to the days when physicians, conscientiously believing they were doing the correct thing, would refuse a fevered patient a drink of cold

water. They could have water with the chill off—that insipid, unrefreshing drink that we none of us care for, and for which the fevered system evinced no craving—but no cold water. That was considered dangerous. Now, in happier days, we see the folly of those times, and gladly give what nature advises, allowing the patient as much cold water as he wishes, recognizing its beneficial therapeutic effects. In view of this fact, that the leaders of the profession, not so many years ago, committed a grievious error in ignoring the demands of nature with reference to giving cold water as a drink, is it not possible that the leaders of the profession to-day are making a similar error in ignoring the demands of nature with respect to giving of other foods than milk. Fifty years ago physicians were not infallible. Are we so to-day?

I leave this thought with you, merely quoting a health maxim bearing on this point: "It is a universal rule in health, and with a very few exceptions in disease, that that is best to be eaten which the appetite craves or the taste relishes."

To this my opponents may bring in the objection so much raised that *any change* of diet is liable to cause a relapse, and they will on this point quote many so-called instances. They may tell you that, following some slight change in diet, the temperature has risen, and the patient has once more to undergo another attack of the disease, and all due to some change in diet. If any such statement is made I want it proved.

In these cases I admit there is a coincidence in point of time between the relapse and the change of diet, but that the one has been the result of the other is by no means certain. The real cause of the relapse is not understood. We know from the rise in temperature that the reinfection has occurred, but there our knowledge ends. This being so, are we justified in coming to any such hasty conclusion as that because there is an apparent connection between the time of some change in diet and a rise in temperature, that necessarily the one is the result of the other? How does the change in diet cause a reinfection by the typhoid bacillus? In this connection how is the advocate of the milk diet to account for the relapses occurring where the patients have been fed strictly on milk and have had no change of diet? For such a case there is no haven of refuge. As a general thing we do not hear so much of these cases. They are not heralded abroad with the same vigor as are the unfortunate cases that are said to be due to a change in diet, yet I will shortly quote some figures—the only ones I could find in medical journals for 1897—and these give a much less percentage of relapses following a more liberal

diet than those I have already quoted, where the restricted milk diet was enforced.

Just now, however, while at this point of "change of diet," which our restricted diet friends tell us is the cause of so many relapses, I would like to draw their attention to what might be considered an error in their treatment, if they in reality believe the relapse to be due to a change in diet.

When the patient primarily came under the physician's observation he had a temperature that was higher than normal, the disease was established, yet what was the first thing advised? A sweeping change, no ordinary change, but a sweeping change in diet; everything he has been accustomed to is denied him, and he is ordered nothing but milk. I can nowhere find any remarks bearing on this point, but if a change in the diet, generally comparatively slight, can cause a relapse, sometimes even when convalescence is established, (in eight days after temperature was normal, in one case at the Royal Victoria,) I do not see how a change, particularly such a radical one, can act so very differently in the earlier or initiatory stage of the disease; only at this stage we would not look for nor speak of a relapse, but we might reasonably expect to find an increase in the severity of the symptoms. Is not that just what we do find?

Another point in regard to relapses is this: The patient has been reduced from a strength-giving diet to an infantile one; surely this weakening diet should make the system more liable to suffer from any change in diet later on.

A third point is that these relapses in hospital practice very frequently follow when the patient has eaten some tit bit that has been smuggled in by his or her friends. They (the friends) out of sympathy have brought something, and the patient has eaten this something; following this the temperature rises, and the cry goes forth that the change in the diet was the cause of it. Admitting for argument that it was the cause, have we not by our own weakening dietetic treatment predisposed to it?

In a case such as this too distinct a line cannot be drawn between a craving and a friend's tit bit. After some weeks on a milk diet one would have to be very careful what change was made, because of the weakened condition due to the typhoid and milk combined.

I have already mentioned that no cause is assigned to those relapses that occur where milk only has been given. The reinfection has occurred, but how? Might we not ask ourselves if the milk itself was not the cause. We know that the milk of the cow is susceptible to any change in the diet of the cow, and that many of these diets

impart to the milk some of their peculiarities. Is it not quite within the range of probability that the effect of some of these peculiarities on the bowels may be deleterious, and that the resisting power of the ileum, already much weakened by the fever and our milk diet, be thereby lessened? To this couple the fact that we have no better culture medium than milk for the development of typhoid germs, and we may get an insight into how a reinfection is made possible, where the unfortunate "change of diet" cry cannot relieve us from further investigation.

Regarding perforations, I will say nothing further than to remark that perforations are now not considered as being due to the change of diet. We are told that a relapse may occur but not a perforation. These perforations are usually found at the bottom of small, deep ulcers, and are believed to be due to ulcerative and necrotic changes that occur gradually.

Dr. Peabody, of New York, writing on diet in typhoid, tells his experience. I quote in full: "We are accustomed to have too great a dread of doing harm at the site of lesion in the ileum in typhoid fever by giving solid food. If I am correct in my opinion as to the inference to be drawn from hunger in a fever patient there is even less likelihood of causing damage to an ulcerated ileum by giving divided egg or beef or chop to such a patient than by giving him milk, and my experience seems to justify the inference." . . . . "It has been my practice for years to allow albuminous foods of these descriptions to such patients, even before the fever leaves him, under these conditions. I have at present under treatment several patients with typhoid fever whose temperatures reach 101°, 102°, 103° F. daily, who are hungry, and who are receiving such solid food once a day. So far as I am aware I do not have a larger percentage of relapses or hemorrhages or other complications or accidents in my practice than I did before I adopted this plan, or than my colleagues do who have not yet adopted it." (*Medical Record*, Nov. 26, '92.) These words are so plain that I refrain from commenting on them.

Dr. Barrs, an advocate of a more generous diet in typhoid, quotes thirty-one consecutive cases that he treated much as does Dr. Peabody Of those thirty-one cases all recovered; two had relapses. Does this not compare well with the statistics of our own hospitals under the milk regime?

Dr. West, in an article in the *Canada Lancet* criticising Dr. Barr's remarks, believes the percentage of relapses is too high, but gives no comparative statistics. I have furnished them. Had Dr. West had them he would have found them a "boomerang." Dr. West believes

strongly in the milk diet. In the commencement of his article he says: "In the febrile stage it is important, first of all, to find out a diet which suits the patient. Milk is the simplest diet, and fortunately it suits most people, but not all. If milk cannot be taken the diet must be modified, and this or that form tried until one is found which agrees with the patient. As soon as the suitable diet is found it should be continued and not changed without good reason."

This is a good common sense argument, and in the main I take it as such. The only objection I offer to it is that he does not in all cases allow the system to dictate, instead of first of all trying milk and then, if that does not agree, casting about until at last he hits on some diet that the system says is suitable, by not rejecting it. I also object to his assumption that milk agrees with all persons except those with whose stomach it disagrees sufficiently to cause rejection.

A little further on in the same paper he practically agrees with this, for he writes: "If I come to a patient and find that patient upon a diet that suits him, although it might be one which I would not myself have originally selected, I should not change it if the patient were doing well."

Considering that Dr. West is a milk feeder, and that his article was in criticism of a physician prescribing a more liberal diet, this last statement is remarkable. I take it that when he wrote that sentiment he had more particular reference to injuring from a change of the diet, as in another paragraph, in writing on the change of diet, he says: "There is no evidence that the food itself produces perforation or any other morbid process except the relapse, and then all the accidents may occur which are met with in the original disease."

One other remark regarding the milk diet in typhoid and I have finished. In prescribing an all-milk diet are we not subjecting the patient to the chance of future disease from certain germs that may be in the cow's milk, notably tuberculosis? In typhoid the great safeguard against the infection of the alimentary tract by tubercle bacilli is removed. The acid secretion of the stomach is either nil or very weak, not sufficiently strong to digest properly, so that we may confidently expect, if there are any tubercle bacilli in the milk, they will pass unharmed into the intestines. But in case the acidity may be too strong we overcome this as far as possible by adding an alkali to the milk. Let the bacilli once get into the intestines, would it not be comparatively easy for them to enter the system through the diseased and weakened mucous membrane, and, having once gained a foothold, may they not, later on in life, be the cause of that dread disease, tuberculosis?

You say this is a remote possibility. I grant it; but, in the following up of my subject, the remote possibility of causing trouble by the milk diet as prescribed nowadays should not be overlooked. And, after all, the possibility may not be so remote as we are apt to imagine.

A closing remark: The articles one reads in medical journals today are nearly all in one strain. It is milk diet, exclusive milk diet, nearly all along the line; yet, with all this writing, we get no comparative statistics.

Dr. West, in closing his article, says: "I do not think this question should be treated as an open one, as if it had not been seriously considered hitherto, for the current opinion is really the outcome of a multitude of observations conducted without bias for a number of years and all the more trustworthy because no attempt is made to prove it by figures." I do not like the sentiment. It is too much like muzzling honest inquiry and research. Remember that as pro-professional men we must, sometimes at least, think for ourselves.

Here I have have given you some results of a more liberal diet in typhoid and some objections to the all-milk diet, and some reasons why a more liberal diet might be adopted. Remember that we are reaching towards the acme of perfection in the treatment of any case when, in obtaining satisfactory results, we have employed methods that have been most acceptable to the patient.

I believe that the time is coming when all typhoid patients will be more generously dieted. Let the day be hastened.

Since writing this article I find that the concensus of opinion of the physicians at both the Montreal General and the Royal Victoria Hospitals is that the change of diet made with care has nothing to do with the relapse. The cause at present is not known, but it is not believed to be due to the change of diet. I trust that before long this view will be generally accepted as correct. That it is not universally so accepted as yet is proved by Dr. West's statement to an interested public that: "There is no evidence that the food itself induces perforation or any other morbid process except the relapse," etc. etc. The idea of the relapse being caused by the change of diet is now considered erroneous. Now if this be so, that the change in diet does not cause the relapse and does not induce perforation nor any other morbid process, what under the sun is it to cause that will be baneful?

REMARKS UPON A NEW DIFFERENTIAL STAIN FOR FAT, SUDAN III.

BY

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During the past few weeks I have been making trial of Sudan III, a new staining agent which has the power of staining fatty tissues without affecting other portions of the preparation.

This stain is an amorphous powder, having the formula $C_{22}H_{16}N_4O$, a disazo-body, of a brick-red colour, which is soluble in alcohol, xylol, ether, and the various aromatic oils, while it is insoluble in water and glycerin. Daddi, of Turin, in 1896, found when he fed animals upon oil impregnated with Sudan III., that after the animals were killed the adipose tissue alone took up the stain and the method of absorption could even be traced. Hermann Rieder in the *Deutsches Archiv f. klinische Medicin*, (Dec.. 1897,) applied the new agent to microscopic sections with success. He was able to determine that the larger fat particles stained a scarlet, while the smaller ones where more of a golden colour, the stain being definitely selective. He states that the method has great value in the determination of fatty infiltration or degeneration in parenchymatous organs, for the examination of milk or colostrum, sputum, fæces, in lipæmia, lipuria, or chyluria, and for fatty casts. Rieder advises that the tissues should be hardened in Müller's fluid and clarified and mounted in glycerin. Absolute alcohol should not be used. After hardening, the tissues are cut upon the freezer and then stained in a solution of Sudan III. made as follows: Ninety-six per cent. alcohol is saturated with the dye and filtered. To this is added two-thirds of its bulk of fifty per cent. alcohol, and the solution again filtered. Sections are left in this for a few minutes, washed in seventy per cent. alcohol, the excess removed with filter paper and are then examined in glycerin. A preliminary stain of hæmatoxylin may also be employed.

From a short experience of this stain I have been able to corroborate most of Rieder's statements, and with a small modification of his method have obtained very good results. The method I employ is to harden small portions of suspected tissue in 10 per cent. formalin. These then can be cut upon the freezer in an hour or two, and very

good thin sections are the result. The sections are placed in hæmatoxylin till stained and then allowed to stand in tap-water. They are then transferred to the solution of Sudan III for three minutes, washed in weak alcohol (60—70 per cent.) and mounted in Farrant's fluid. This produces very clear and permanent preparations. Zenker's fluid or sublimate may also be used to fix the tissues. So far as I have observed, the stain is absolutely selective for fat. Bacteria are not stained by it, but it might be employed to determine fatty particles in their bodies. Myelin is faintly tinged by it, but by no means so markedly as fat.

To stain fatty casts the urine should be centrifugalised, the sediment should then be mixed with 3 to 4 c.cm. of distilled water and an equal bulk of the Sudan III. solution added. This is then diluted with an equal bulk of absolute alcohol and again centrifugalised, and the sediment examined under a low power. The fatty particles are stained and look like red granules on the cast.

The method may also be applied to blood work. It is very important that the coverslip should be absolutely free from grease. The film is fixed for 15 minutes in formalin vapour, and the stain used as before. A subsequent immersion in hæmatoxylin for a few moments is valuable to bring out the nuclei of the leucocytes. The alpha-granulations are not stained, thus affording an additional proof that these bodies are not fat. I have also failed to stain the neutrophile granulations by this method.

Altogether I believe that in Sudan III. we have a valuable addition to our laboratory equipment, for it possesses distinct advantages over osmic acid. For rapid work osmic acid is often employed, but it results in a dirty black smeary specimen, which is very unsatisfactory, and even when Fleming's mixture is used, unless the portions of tissue are very small they are very unequally stained, and in laboratories the solution is rarely in good working order. Osmic acid also tinges other bodies besides fat. With Sudan III. the preparations are clean, clear and distinct, and the finer structure can be readily studied, very pretty pictures being produced. The tint is also readily distinguished from brown atrophy or other forms of pigmentation. Care should be observed that after staining all excess of stain is washed away, and also not to mistake in the finished tissues fat droplets which have been misplaced in the process of cutting for a local fat invasion. For gross work at post-mortems only moderate results can be expected, and in my hands it has has not proved satisfactory.

POST-MORTEM EXAMINATION IN TWO CASES OF CHOREA CHOREA INSANIENS AND CHOREA MAJOR IN INSANITY.

BY

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Cases of chorea are rarely fatal and when so, death is from exhaustion, by accident traumatic or pathological, or when complicated with other diseases or accompanied by mental symptoms of a maniacal character. Cases being so infrequent, I wish to place on record these two cases of chorea major; one of which would come under Osler's classification of chorea insaniens, the other, a case of chorea major occuring in an insane patient.

CASE I.—For the clinical notes of this case I am indebted to Dr. Croskery, late House Physician to the General Hospital, Kingston.

Alf. R., æt. 17, apprentice plumber.

Family history.—Patient comes of a "neurotic" family. (Epilepsy) Otherwise no family history of disease.

Personal History.—Patient has had no previous illness so far as can be learned from parents. On April 9th, 1897, on returning from work he developed a peculiar train of neurotic symptoms. irritability of temper, crying alternating with loud fits of laughter, and other hysterical manifestations. A physician was called who placed him on valerian and bromides. No movements were at this time detected. Two days later, however, decided muscular movements set in, the other symptoms persisting, but in milder form. The movements grew decidedly worse till on the 16th he was ordered into the hospital, but was not removed till the following day. He was admitted into the hospital at 4 p.m. on the 17th. When admitted muscular movements were incessant over entire body, least marked in face. He was incapable of any progression and could neither sit nor lie. His countenance was dull and stupid, and mouth wide open. Respiration 24. Pulse 88. He was given, with difficulty, pot. bromide 20 grs., choral 15 grs., by mouth, without the slightest effect. At 8.30 p.m., he seemed to have lost all knowledge of his surroundings. Shortly after he sprang from bed to floor and then jumped high into air, falling heavily on floor. Morphia $\frac{1}{4}$ grain was given hypodermically without effect; the movements continuing incessantly. At 9.15 he was placed under chloroform and then he slept till 11 p.m., occasional movements occurring. When he awoke movements were

exaggerated and patient was perfectly crazed, requiring several persons to prevent him from injuring himself; ¼ grain morphia was again given, and was followed by sleep till 4 a.m., quite peaceably. Then his breathing became loud and deep and mouth filled with mucus. He roused up occasionally and when he did, exaggerated movements came on. Toward 6 a.m., respiration became very slow, only three to five per minute. Artificial respiration was resorted to. The pupils were contracted, the right one particularly. Repeated hypodermics of strychnia and brandy were given and breathing became better. Stupor persisted till 12 m., when he revived under further stimulation, the movements again commencing. He died at 1 p.m., 21 hours after admission.

The autopsy was not made till April 26th, eight days after death, as permission was at first refused by parents. The body was kept in the meantime in a vault at a low temperature and was in an excellent state of preservation, better than I often find in bodies 30 hours dead in summer time.

External Examination. Early surface signs of putrefaction evident in greenish discoloration of the iliac regions and back and in the outlined surface veins. A few small bruises over arms and legs. Pupils contracted and equal.

Head. A small blood extravasation over occiput due to fall on floor. Skull cap and dura mater normal. Longitudinal sinus full of fluid blood. Veins of pia mater engorged. The arteries of the base of brain were carefully dissected out and were found normal except the right anterior cerebral, in which there was found a recent embolus, just anterior to the anterior communicating artery. The artery beyond contained recent clot.

The brain weighed 46 ozs. Cerebral substance quite soft. The ventricles were slightly dilated. No lesions found in any part of brain substance. No change could be detected in parts supplied by right anterior cerebral, an evidence of the recent nature of the embolism.

Thorax.—Pleuræ contained no fluid nor adhesions.

Lungs.—Show commencing gaseous putrefaction, but no pathological features.

Trachea and Bronchi.—Show putrefactive changes.

Pericardium.—Normal.

Heart.—Weighed 8½ ozs. Both sides of heart full of blood. Walls healthy.

Right auricle and ventricle and their valves normal.

Left auricle and ventricle present no particular features. Aortic valve competent.

On both cusps of mitral valve there are present on auricular aspects, just behind free edge, recent pin head to mustard seed sized vegetations. These were most marked on the aortic cusp where at one spot there was a mass of these forming a granular heap the size of a split pea. Microscopically no bacteria other than those found in blood (putrefactive bacilli) were detected in these vegetations.

Aorta and pulmonary arteries were normal.

Abdomen.—Putrefaction had advanced to a considerable extent in the abdominal organs. No pathological feature detected in any of the abdominal organs.

No cultures were made as putrefactive bacteria would mask any result of value.

Summary.—Acute simple endocarditis. Embolism of the right anterior cerebral artery.

It is quite possible that in this case there were also emboli in the minuter branches of the cerebral vessels. Microscopic sections of the basal ganglia were a failure owing to failure of hardening and fixing re-agents to act, due to age and consistence of brain.

CASE II.—This case was one occurring in Rockwood Asylum for Insane, which I am permitted by the courtesy of its medical officers to publish.

Mrs. M. L. T., æt. 50. Has five children. Was admitted to Rockwood Asylum, Sept. 2nd, 1896, for "dementia with occasional suicidal and homicidal propensities."

Her family history is practically nil, except that she has no insane nor choreic relations. On admission the woman was in fair health. Choreic movements were present chiefly in arms and face. Her history dates back 10 years and has been one of gradually increasing mental weakness, but no history as to period of onset of the muscular movements can be obtained.

On the dates given I find the following notes of her case: Oct. 6th 1896.—Patient walks with a shuffling gait, feet widely separated and toes turning in, and requires help. Both shoulders are jerked forward and upward, and there are marked twitchings of the fingers. The facial muscles are continually in motion. Speech is indistinct and jerky. Dizziness is constantly complained of. She sleeps well and spasms subside during sleep. The eyes are normal. The appetite is inordinate as a rule. Heart and lungs *nil*. Feb 2nd, 1897.—Walking is becoming very difficult, even when assisted. The patient is now a distressing feature from the constant movements, and has now to be fed.

May 1st.—The patient has been rapidly failing of late. There is

now not the slightest control over her movements and if not held she tumbles about, injuring herself. Ulcerated sores have appeared on scalp, sacrum and along spine. The patient has become very noisy.

She gradually weakened and died on May 13th. The autopsy was made by myself, 30 hours after death (May 14th).

The body was very much emaciated. There were numerous ulcers on dorsum of body, one large one over the sacrum, several small ones about an inch in diameter were present over dorsal spine and one over occiput an inch in diameter. This latter one was surrounded by an area of purulent infiltration of the scalp tissues. The others showed no marked inflammatory signs nor yet any evidences of healing. The pupils were partially dilated and equal.

Skull Cap.—Normal. Longitudinal sinus empty. On inner surface of dura mater, on both sides and extending to the base, was a fibrous membrane about $\frac{1}{16}$ inch in thickness, containing a small amount of serous fluid in its interstices, and presenting no signs of recent or old hæmorrhage. This fibrous membrane was separable with difficulty from the dura, but was readily separable from the pia-arachnoid and did not dip down at any part between the convolutions. I believe this fibrous membrane to have been the results of a hæmorrhagic pachymeningitis despite the absence of any traces of hæmorrhage. The membrane itself was very white and possessed a scanty blood supply

The brain weighed 42 ozs. Substance softer than usually met with. The cortex cerebri somewhat dark in color. Ventricles of normal size and filled with usual fluid. Nothing of import in other portions of brain.

Thorax.—Pericardium contained 2 drachms serous fluid.

Heart.—7½ ozs. Muscle pale. Connective tissue appreciably increased in heart wall.

Right ventricular wall hypertrophied slightly, the wall measuring ½ inch, and chamber slightly dilated. Tricuspid and pulmonary valves competent.

Left auricle about ⅓ larger than right.

One cusp of mitral valve was thickened by fibrous tissue formation and the edge slightly retracted, but proved competent by water test. Evidently it was not quite so, as witness the dilatation of the left auricle and the slight hypertrophy of the right side of the heart.

The aorta showed numerous areas of primary fatty degeneration and an occasional early atheromatous patch.

Œsophagus.—Normal.

Trachea—Normal but contained considerable mucus.

Pleura.—No fluid nor adhesions.

Lungs.—34 ozs. together, quite normal in all respects.

Abdomen.—Peritoneal cavity contains no fluid. Intestines moderately distended. Great omentum free from fat.

Small Intestine.—Contains a little bile stained fluid contents. Mucus membrane healthy.

Great Intestine.—Shows a few scattered scybala.

Stomach.—Normal.

Liver.—30 ozs. Normal. Gall bladder partially filled and the ducts patent.

Pancreas and Suprarenals.—Unchanged.

Kidneys.—Right 3½ ozs. Capsule slightly adherent. Cortex diminished in size and pale. Medulla dark red. No cysts in cortex. Microscopically there is found a slight increase of the interstitial connective tissue, most marked between and about the convoluted tubules.

Left Kidney.—3¼ ounces, same condition as right.

Bladder.—Empty.

Uterus, Tubes and Ovaries.—Are small and atrophied.

Summary.—Pachymeningitis interna fibrosa, (likely hæmorrhagic in origin). A thickened slightly retracted mitral valve cusp. Slight interstitial nephritis.

Case I, is of particular interest in the finding of the plugged anterior cerebral vessel, together with the acute endocarditis. It is to be regretted that permission to hold the autopsy was not sooner obtained so that a bacteriological investigation could have been made. It is a matter of question whether the death in this case was due to the embolism, a pathological accident. I am more ready to ascribe it to the cumulative action on the nerve centres of the toxic agent (whatsoever it may be) of the disease.

Clinical Reports.

SAW CUT THROUGH SKULL.

BY

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E. E., aged 26, French-Canadian speaking both languages, was admitted to Dr. Kirkpatrick's ward in the General Hospital, on the 18th of October, 1897.

The patient was employed in a saw mill, and while clearing away the sawdust beneath the circular saw his head came in contact with it and a severe wound was inflicted. He was taken to hospital and on admission was attended by Dr. Wainwright. On examination the patient was found to be in good condition, sat up in bed and did not seem to suffer from the injury. Nothing abnormal was observed about the face or body with the exception of the left eye, which the patient could not open as wide as the right. Patient was taken to the operating room and the cut on head examined under ether. It was found to be in the left side of the head, three-quarters of an inch from the median line at the anterior extremity, and three and three-quarter inches from the same line at the posterior extremity of the cut. The anterior extremity of the wound was three-quarters of an inch above the supra-orbital ridge and the cut extended backwards and outwards six and one-half inches The edges of the wound were gaping, showing the bone beneath, which had been cut through. The edges of the cut bone were cracked in places and many splinters found in the wound. A probe was passed between the edges of the bone, which were one-quarter of an inch apart, and it penetrated the wound (from its own weight) two inches at the centre and one and one-half inches at either extremity. There was free bleeding from the vessels of the scalp which were ligatured with catgut. The wound was irrigated with hydrargyrum bichloride solution (1-5000) and then with saline solution. The space between the edges of the bone was plugged with iodoform gauze, the ends of the gauze projecting at either end of the wound. The edges of the scalp wound were then brought together with silkworm gut sutures, dressings applied and patient sent to ward.

The following morning, Oct. 19th, the patient had no temperature and pulse 86. The right side of the face was paralyzed, the mouth

being drawn to the left side. The patient was not able to purse up his lips. Grasp of right hand was considerably less than that of left. Knee-jerks not increased. Sensation normal. Retention of urine.

Oct. 20th.—Patient showed evidence of motor aphasia. He was unable to give the name of an object but knew if it was called by its correct name. Is unable to write, although able to do so before the accident. Passed urine naturally.

Oct. 21st.—No change in condition of patient.

Oct. 22nd.—Slight rise in temperature, wound dressed. No change in the nervous symptoms.

Oct. 23rd.—Sensation was less acute over dorsum of right hand. Hand grasp slightly weaker than on the 19th. Patient would not speak unless spoken to, and then usually confined his answers to "yes" and "no."

Oct. 28th.—Sensation in hand appears normal. Facial paralysis slightly less marked. Hand grasp rather stronger.

Nov. 1st—Laryngoscopic examination made by Dr. Birket and nothing abnormal found. Firm union of scalp wound except at posterior end, where gauze had been kept inserted.

Nov. 4th.—Patient can now remember the names of articles (if told him) for a short time. Previous to this names were forgotten immediately after being told.

Nov. 5th.—Patient was able to give the name (in French) of a key and a knife, when shown him. Facial paralysis less marked.

Nov. 9th.—Patient had no difficulty in giving the names (in French) of articles shown him. Facial paralysis becoming less marked.

Nov. 14th.—Patient can now name (in French) any article shown him, and also fairly well in English. Wound almost completely healed.

Nov. 22nd.—Patient speaks perfectly in French and is rapidly recovering his English. Discharged. Cured.

"OBITER SCRIPTA" III.

(Casual notes from the Medical Clinic of the Royal Victoria Hospital.)

BY

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SOME INTERESTING CASES AFFECTING THE RESPIRATORY SYSTEM.

CASE I.

Serous membrane tuberculosis, involving pleura and peritoneum, with chronic non-tuberculous muco-purulent bronchitis. Terminal disseminated miliary tuberculosis.

Cases of this kind are always of very great interest and belong really to the more uncommon manifestations of tuberculosis. The victim of this disease was a young man, who, had according to his account been in good health up to the onset of his present illness.

History.—He entered the hospital on the last day of December, 1897, complaining of dyspnœa, cough, general malaise and an acute pain in the left side which had come on *suddenly* two weeks previously. All these symptoms had followed exposure to cold and wet and in a very short time copious expectoration and prostration supervened. There was a family history of tuberculosis.

On admission, his temperature was 100½°, his pulse 106, and the respirations 28 per minute. He was markedly anæmic and the skin was moist. Apart from some irregularity in the pulse, the circulatory system showed no other evidence of disease.

His *chest* was of a tuberculous conformation being long with an acute angle at the ensiform cartilage, widened intercostal spaces and generally flattened. Examination showed a left sided pleural effusion of moderate degree, while on auscultation a few moist râles were heard over the left apex; the breathing on the right side was harsh. The sputum was copious, muco-purulent in character, and repeated examination *failed to give evidence of either tubercle bacilli or elastic tissue.* The *digestive system* showed in the main, a full and tense abdomen with no spontaneous pain, and palpation revealed neither tenderness, tumour, nor evidences of fluid. The spleen and liver were of normal size. The urine gave no evidence of disease.

Course.—Throughout the course of the malady the temperature assumed the daily intermittent type; there was copious sweating and rapid emaciation, while the cough and expectoration persisted.

Puncture of the pleural cavity revealed the presence of slight hæmorrhagic effusion, which on microscopical examination showed mainly a few blood cells and very few leucocytes which had undergone marked fatty degeneration. Death followed in less than three months after the onset of symptoms.

Autopsy.—The autopsy showed *bilateral hæmorrhagic pleurisy*, more advanced on the left side; tuberculosis of the peribronchial glands; a subacute more or less *dry chronic tuberculous peritonitis* which was obviously of longer standing than the pleural affection The *mesenteric glands* were caseous and the *ileum* presented one small shallow ulcer evidently tuberculous in nature; the pericardium was free from disease. In the *lungs* there was a chronic simple muco-purulent bronchitis, but no evidences of chronic tuberculosis. The only other condition of interest at the autopsy was the generalised miliary tuberculosis which evidently had induced the lethal termination.

Remarks.—The special features of interest in this case are as follows:—A chronic peritonitis which had been completely masked through the acute symptoms in the pleural cavity; the course of the malady throughout; the presence of a simple muco-purulent expectoration with many râles in one lung, naturally arousing the suspicion of chronic pulmonary tuberculosis, though oft repeated examination for bacilli had been quite negative.

Infection had occurred here no doubt from the alimentary tract as seen by the condition of the ileum and mesenteric glands, the peritoneum being thereby secondarily involved. The pleura was infected through the diaphragm as is usual in cases of this kind where the peritoneum is the primary seat of disease. In many cases recorded by Vierordt, the pleura was first involved and the peritoneum secondarily, and not infrequently the pericardium was likewise secondarily affected. That authority has never seen a primary pericardial tuberculosis under such conditions.

Clinically, cases of serous membrane tuberculosis vary considerably, being often extremely insidious in the onset, at other times, as in our present case, very acute. It is unusual to find other organs of the body affected. Frequently a pleurisy, evidently tuberculous in nature becomes "healed" and then within some months after the pleural symptoms have disappeared, the peritoneum shows evidence of acute inflammation, and later on again the pleura becomes involved for the second time. This feature in the course of the disease is often of aid in the diagnosis. It would appear that, from various observations made, fever is not a necessary accompaniment of the disease, though usually present. Dropsy is often a very marked symptom, and then

the differential diagnosis between cirrhosis of the liver and serous membrane tuberculosis becomes extremely difficult, more especially where fever is absent. The difficulties are all the more striking when the spleen is palpable, for in many cases of this form of tuberculosis, that organ is distinctly enlarged. Indeed, observations have shown that not uncommonly cirrhosis of the liver occurs with serous membrane tuberculosis, usually as a result of an old standing peritoneal involvement and capsular fibrosis. When the double affection occurs the actual condition must present great difficulties of diagnosis though doubtless *one* of the two processes would readily be assumed. The *treatment* is on the whole unsatisfactory, puncture of the pleural cavity being recommended for effusions in that region, and laparotomy for the peritoneal affection.

CASE II.

Latent pyo-pneumothorax.—Signs of extreme pleural effusion; normal temperature, pulse and respirations.

(The notes of this case are in part abstracted from the careful report of Dr. McCallum, one of the Resident Physicians).

A young woman who had cough, expectoration and dyspnœa, entered the medical clinic last April under Dr. James Stewart. She had been ill for nearly a year from influenza, so she stated, upon which a pneumonia had supervened. She was confined to her bed almost constantly from July to October with cough and frothy expectoration and slight intermittent attacks of dyspnœa. She never had had any hæmoptysis, nor were there sweatings, chills nor other evidences of pulmonary tuberculosis. The dyspnœa had at no time been very marked till two weeks before admission, which in this connection is a point of distinct interest, all the more so, inasmuch as she stated, that in December, of the past year she noticed splashing sounds in the chest on any rapid movement; this only persisted for a few weeks, and the onset had never been attended with any acute symptoms or pain. Ever since the splashing had been observed however, she had also noticed palpitation of the heart on the *right* side of the chest.

There could be no doubt from the history given that the patient had been suffering from a pneumothorax contracted in the course of a more or less chronic pulmonary disturbance. In the absence of further history however, it was impossible to state more definitely the actual course of the malady.

Her condition on admission was very briefly as follows:—

She was distinctly anæmic, preferred the left lateral decubitus, other positions causing marked dyspnœa and distress. The temperature was 98°, the pulse 96 and the respirations 20 per minute. Examination of the chest showed a condition typical of that induced by

extreme left pleural effusion; viz., fulness of the left side and obliteration of the intercostal spaces; diminished expansion; absence of vocal fremitus; a flat note on percussion from the extreme apex to the base; absence of breath sounds and of vocal resonance on auscultation. There was dulness too on the right side in front, close to the sternum, up to the 2nd rib, and at the level of the 3rd rib this dulness extended outwards for three inches and was continuous below with the hepatic dulness. In the 4th interspace three inches to the right of the sternum, was seen the diffuse apex beat of the heart and the dulness extended slightly beyond. Both basal sounds were markedly accentuated, but there were no murmurs. The spleen was distinctly palpable, being pushed down evidently by the superjacent fluid. The urine was normal. The sputum contained no bacilli of tuberculosis, and there were no signs at this time of pneumothorax.

On the day after admission 30 oz. of creamy pus was removed, though without altering to any marked degree the physical signs in the chest. Cultures made of the fluid remained sterile. Three days later she was again aspirated, and 15 ozs., removed. This time the heart receded slightly towards the normal position; there was a tympanitic note over the upper third of the left lung, and the Hippocratic sucussion was readily obtained, and quite audible to those standing some distance from the bed. Beyond this feature, however, there were no definite evidences of pneumothorax, the coin sound and metallic tinkling not being elicited. Operation was urged but the patient refused, and nine days later she was aspirated again, this time 40 ozs., of creamy pus and lymph being removed. Great relief followed and the physical signs altered to a marked degree. There was a tympanitic note on percussion as far as the 4th rib, and from there down to the base the note was flat, the sucussion splash, coin sounds and metallic tinkling being all readily obtained. Respirations were performed with greater ease, and the patient felt in every way so comfortable that she insisted on leaving the hospital the same day.

Throughout the course of her stay, with one day's exception, her temperature remained normal. The tuberculin test was not employed.

The case is of particular interest as illustrating how insidiously pneumothorax may sometimes develop and quite in the absence of the usual acute symptoms which call for urgent treatment.

Cases of this kind have been recorded by others, particularly by S. West of London, who mentions instances where in the so called apparently healthy, pneumothorax has been found from time to time, and in many instances associated with strain. Probably in 90 per cent. of cases tuberculosis is the main etiological factor though numerous instances exist showing other causes to be at work, and in not a few the antecedent condition has been, as in our own case, quite obscure.

RETROSPECT

OF

CURRENT LITERATURE.

Surgery.

UNDER THE CHARGE OF GEORGE E. ARMSTRONG.

Surgery of the Kidney.

MORRIS. "The Hunterian lectures on the surgery of the kidney."—*British Medical Journal*, March 26th, 1898, and subsequent numbers.

These lectures are really of very great value. They deal with an important subject in which great advances have been made during the past twenty years. Mr. Morris can speak with authority as he has had exceptional opportunities for observation, and an unusually large experience in operative work upon the kidney.

In the first lecture the history of kidney surgery is briefly given, showing that although the practicability of nephrotomy was discussed during the sixteenth, seventeenth and eighteenth centuries, yet it was not established as a feasible operation until the latter half of the nineteenth.

The dawn of renal surgery admittedly dates from August, 1869, when Gustav Simon of Heidelburg, removed by the lumbar method the kidney of a woman who had a ureteral fistula opening upon the abdominal wall over the pubes, and following an ovariotomy done one year and a half before. Simon performed his operation in the face of the pronouncement long before made by Rayer that it was "madness to dream of extirpating the kidney in the human subject" For himself, he compared the result of fifteen hysterectomies with fifteen nephrectomies, practiced on animals, and satisfied himself that there was no more danger from a surgical point of view, in the one operation than in the other.

A most important advance has been made in the excision of a diseased part of a kidney instead of the whole. Cysts and tuberculous breaking-down masses have been successfully removed, without undue hæmorrhage, and the results have been satisfactory. Mr.

Morris thinks that partial resections are capable of a wider application than has yet been given to them, especially in the case of tumours taking origin from the capsule of the kidney itself.

Operations upon the ureter are an advance of the last few years and are destined to have an important influence upon renal surgery generally. Ureterotomy has been employed in the treatment of hydronephrosis, calculus and calculous anuria. Ureteral anastomosis and other plastic operations on the ureter will save many kidneys which would formerly have been nephrectomised on account of hydronephrosis, fistulæ, and surgical injuries to the ureter. The removal of calculi lodged in and obstructing the ureter is now an established operation. They have been successfully removed from the renal and vesical ends. Every abdominal surgeon should now be familiar with the different methods of dealing with accidentally made wounds of the ureter in its course and be able to unite the severed end to the pelvis of the kidney above or into the bladder below.

Mr. Morris in his second lecture deals with renal calculus; the difficulties and errors in diagnosis in their relation to exploration of the kidney, and unsuspected, quiescent and migratory calculi. He puts them in the very forefront of surgical affections of the kidney for the following reasons:

1. They are the most frequent and most painful of the surgical diseases of the kidney. Probably no disease except acute tetanus is capable of causing worse suffering.

2. Renal calculus, whilst slowly destroying the kidney, often physically disables its victims by its unrelenting irritation, and its unyielding resistance to every form of dietetic and medical treatment.

3. No disease gives rise to such a variety of morbid changes in the kidney as calculus; and none is more certainly fatal when allowed to progress without surgical interference.

4. Few operations are so successful as nephrolithotomy, by which a calculus is removed from a kidney not disorganized by the calculus or otherwise. No great operation is followed by a smaller mortality. Nephrolithotomy gives absolute cure, saving the kidney from progressive destruction and the patient from what at any moment may prove to be an imminent danger to life.

5. Renal surgery will grow in confidence and in favour with the profession and the public, as nephrolithotomy anticipates and displaces nephrotomy and nephrectomy.

6. Another reason why calculus disorders take so prominent a lead amongst the surgical affections of the kidney is because of the difficulties and errors attending their diagnosis.

These difficulties arise from four causes:

1. Several other renal and ureteral affections give rise to the same symptoms as stone.

2. Several diseased conditions of other organs cause symptoms which similate those of renal calculus.

3. Symptoms caused by renal calculus may be transferred to other organs; or may be of a psychical order, with or without high temperature, and not referred at all to the kidney itself.

4. Calculi may be for an indefinite time masked, giving rise to no symptoms whatever, yet causing the while progressive destruction of the kidney.

The symptoms of renal calculus are well-marked attacks of renal colic, with crystals of uric acid or calcium oxalate frequently occurring in the urine, and when the urine is intermittently mixed with a good deal of blood, or persistently contains a microscopical quantity. If the patient has already passed a stone, there is all the more likelihood of another. Mr. Morris' remarks on the different diagnostic symptoms are very clear and instructive, and are of great value because of his very large experience in kidney surgery. Mr. Morris lays great stress on pain and tenderness, and makes it a rule always to operate on the painful side. To illustrate I may briefly mention the following case of calculous anuria. Calculi could be felt through the abdominal wall in the right kidney, but the attacks of pain associated with the anuria were referred to the left kidney. Mr. Morris operated upon the left kidney, found a small stone in a large hypertropied kidney, and the paroxysms of pain and anuria disappeared entirely. In this connection it is pointed out that the nerve supply is such that pain is much more likely to be reflected up or down on the side of irritation, than across to the opposite side.

In this way is explained a large group of cases of *unsuspected calculi with symptoms transferred to other organs.* It is sometimes the same with the ovary or the bladder.

A third group include *unsuspected calculi with high temperature and great psychical disturbance,*—unfortunately not common. This group is illustrated by the following: The patient having been desperately ill for two or three months with symptoms of a typhoid type, attended by acute but vague pains over the whole body and with great mental prostration, the illness terminates after the discharge of a few ounces of pus and some renal calculi, presumably from the left kidney. Two or three other cases are reported, having a somewhat similar history. The difficulties of diagnosis in this group are very great. It is in such cases that catheterization of the ureter and the passing of a

sound along the ureter to the pelvis of the kidney, might give very valuable information. The character of the disturbance in these cases is one of depression, melacholia, stupor and nervous apprehension of danger. Mr. Morris does not think it due to uræmic poisoning. It occurs when there is no suppression of urine and is absent in calculous anuria when no urine is excreted for six or eight days in succession. The writer, in one instance, observed these symptoms present in an extreme degree, while from 40 to 60 ounces were secreted daily, containing 10 grains of urea to the ounce. The patient had both kidneys filled with calculi. In such cases culture experiments should be made with the urine from the diseased kidney.

The possibility of the presence of *quiescent* or *migratory* calculi should always be borne in mind. On this question Mr. Morris speaks as follows: "It is difficult to shake oneself free from the influence of tradition, and thus for years I made it a practice, in cases in which the symptoms were slight or of only recent occurrence, to treat the patients for a time upon the expectant plan. I possess several calculi which have been passed naturally; but I now regard them as the emblems of what ought to be very largely an obsolete treatment, and from which I have had one or two rude awakenings" This is a strong position to take, and such cases offer an opportunity for the exercise of good sound judgment on the part of the surgeon.

In his third lecture, Mr. Morris deals with Fistula caused by Renal Calculus; Obstructive Anuria caused by Renal Calculus and the technique of the Exploration of the Kidney and Ureter for Calculus.

Fistulæ are not of common occurrence, and are usually found to follow only when perinephritis or perinephritic abscess was present at the time of operation. It is, as a rule, unsafe to explore the kidney when the tissues around are in an acutely suppurating condition. The safe course is to drain the abscess and perform a second exploratory operation after the abscess is healed.

Mr. Morris recommends the oblique incision, which can, if necessary, be prolonged downward over the course of the ureter, and he does not fear much being unable to find the stone be it ever so small.

Another point emphasised in these lectures is the frequency with which complete relief of symptoms follows exploratory operations upon the kidney where no stone is found. This is sometimes due to the breaking up of adhesions, and in other cases the way improvement follows is unknown.

George E. Armstrong.

Ophthalmology.

UNDER THE CHARGE OF J. W. STIRLING.

High Myopia and its Surgical Treatment.

FUKALA. *Archiv. für Augenheilkunde*, XXV.. 1897.

D. MAGEN. *Weiner Klin. Woch.*, No. 1—2, 1898.

Fukala here gives an exhaustive account of his results in the extraction of the lens in cases of high myopia. The results are certainly highly gratifying, and, if time shows that the results gained are permanent, then surely abstention from operative interference in cases of high myopia will be very culpable. Fukala operates with a Bowman stop needle—performing discission about the centre of the capsule, and if glaucoma threatens, he paracenteses the anterior chamber.

Magen adds nothing new. He holds old age is no contraindication to the operation.

The Unprotected Treatment of Wounds of the Eyeball.

T. HJORT, Christiania. *Centralblatt für Prakt. Auyenheilkunde*, November, 1897.

Hjort's statement as to his method of treating wounds of the eye is certainly novel, if not startling. He allows the eye to go absolutely uncovered after an operation, and he claims that the results of his operations, especially on cataracts, are so favourable that they justify his procedure. His theory is that the natural secretions of the eye, as long as the lachrymal passages are in order and the lids work properly, perform through the flowing away of the tears a physiological toilet of the eye and are actual antiseptics. Anything that interferes with this lachrymal flow and drainage favours the development of micro-organisms, and is dangerous. Bandages act this way, and also render the eye succulent and congested, as does also the use of irritating antiseptic lotions. Epilation of the lashes must always be done, as this eliminates a markedly infecting surface.

Interstitial Keratitis following Influenza.

E. HILBERT, Sensburg. *Die Ophthal. Klinik.*, February, 1898.

Cases of this eye disease following influenza were reported after the epidemic of 1891. Hilbert now brings forward another case which

occurred during convalescence from influenza, and under treatment ran a short course of six weeks, passing through all the typical stages of infiltration, vascularization and clearing up.

Hard Chancre of the Eyelids.

GAGZOW. *Deut. Med. Woch.*, February 10, 1898.

This is the case of a child of fifteen months, in whom the inner canthus was infected from mucus patches on the father's tongue. Mercurial inunctions and local iodoform dressing affected a cure. There was but little induration.

Cortical and Psychical Blindness.

M. A. LUNZ. *Deut. Med. Woch.*, No. 38, 1897.

Lunz describes two cases with these symptoms, of which one died. The post-mortem on this case revealed arteriosclerosis of the posterior cerebral artery, and spots of softening in both occipital lobes.

Antisyphilitic treatment of the second case resulted in the partial recovery of the optical memory pictures, as well as the ability to identify new optical impressions with former ones. Here there was entire loss of colour vision and alexia which improved.

Difficulties in Localizing Cerebral Tumours.

L. BRUNS. *Wein. Klin. Rundschau.*, No. 46, 1897.

This paper is well worth careful perusal. Bruns points out many difficulties, and gives some valuable hints to assist one in localizing cerebral tumours. Tumours of the frontal lobes and of the cerebellum both cause an upset of equilibrium. Here, however, a careful investigation of the general and local symptoms clear the diagnosis, as they are very different when the tumour is in either of these two situations.

Bruns considers homonymous hemianopsia as of little value in the typical diagnosis of cerebral tumours. Right homonymous hemianopsia, associated from the outstart with alexia and word blindness, points to a tumour in the white matter of the left occipital lobe. Bruns rather lauds the method of percussion advanced by MacEwen. The anomalies of cranial percussion, tenderness, tympanites and cracked pot sound are of great importance, when they are clearly marked and exhaustive, for general diagnosis and for local when they are distinctly circumscribed. Especially is this so when they coincide with the conjectural seat of the brain symptoms. Marked tenderness and tympanites can only exist when the tumour is in the cortex.

J. W. Stirling.

Pathology.

UNDER THE CHARGE OF J. G. ADAMI.

Antistreptococcic Serum.

LOUIS COBBETT, M.B., F.R.C.S. "Antistreptococcic Serum"—*The Lancet*, April 9th, 1898.

So much has been written of late upon anti-streptococcic serum, both from the experimental side and in reference to the results of the employment of the same, and those results have been so contradictory and so uncertain that without a very full and careful study of the literature, it is impossible to arrive at any very satisfactory conclusion as to the value of the medium; and so scattered is that literature that only he who has entrance to a library singularly rich in foreign periodicals and society transactions, is able to master it. Thus Mr. Cobbett has performed a most valuable service in bringing together the literature upon the subject. His own studies upon diphtheria serum and upon streptococcus disease renders him peculiarly fitted to undertake a review of the matter, and he has in this article contributed to the *Lancet*, treated the whole in so judicial a spirit and so fully, that little more is necessary than to summarise the more important points here on which he lays stress. Indeed the present article does not pretend to be more than an epitome of Mr. Cobbett's paper.

It is perhaps going over old ground to point out that the first to investigate the anti-bacterial and protective properties of sera, was H. Roger, who in 1890 found that the streptococcus of erysipelas grow as freely in the serum of the immunised as in that of the normal rabbit. But while it grew thus freely, the coccus had been affected, for such cultures, previously virulent, made in the serum of immunised animals were now found to be perfectly harmless, and Roger further pointed out that "if one injects the serum (of an immunised rabbit) into the veins of a rabbit, and at the same time inoculates it with a virulent culture, one can see that the animal survives, but it is on condition that it has received a large quantity (5 to 6 cc.) of the serum." As Cobbett points out, these observations were probably erroneous, for Bordet has recently shown that streptococci growing in the Marmorek's serum, lost none of their virulence; and again, if the cocci be washed free from the serum in which they have

been grown, they are virulent, thus indicating that the harmlessness of injections of streptococci grown in serum of immunised animals, is largely due to the simultaneous inoculation of the serum.

This, however, was the practical beginning of studies upon the sera inoculated with streptococci. In 1893, Mironoff employed the streptococcus culture which he had received from Roger, immunised rabbits by various procedures, and found that the serum was capable of conferring immunity on other rabbits, and also of favourably influencing the course of septicæmia already progressing. In 1895, therewere several investigators working in different laboratories: Petruschky in Berlin, Borneman in Vienna, Denys and Leclef in Louvain, and in February, in Paris, Marmorek, Charrin and Roger simultaneously published their results. Marmorek had been able to so intensify the virulence of the streptococcus that a dose of 0.000,000,0001 c cm, killed rabbits, and produced a toxin of intense power. With the living cultures of this and the filtered toxins he was able to immunise animals and from them to obtain a serum which had very active and preventive powers. In a later publication he pointed out that the best serum which he had been able to obtain was of sufficient strength that 0.2 c cm. from 12 to 18 hours later, would protect a rabbit of from 1500 to 1800 grms. weight, against ten times the minimal fatal dose of virulent streptococci injection given. Charrin and Roger had immunised an animal with cultures concentrated and heated to 115° C., but they merely stated that the serum was curative. In the same year and almost simultaneously appeared Gromakowsky's paper in which he recorded that he immunised rabbits with the streptococcus of erysipelas (Marmorek's streptococcus had been obtained from a case of tonsilitis) and had obtained a serum of which 2. cc. would protect rabbits from peritoneal inoculations of quantities of culture slightly larger than those which killed the control animals. Denys and Leclef in Belgium, also obtained a serum which could protect against feeble infection. Bulloch in London in the same year obtained from a horse inoculated with streptococci from erysipelas a very strong serum of which 0.02 c cm., protected against ten times the lethal dose of the culture. But Marmorek's results we need scarcely remark, were those which were of the most importance and attracted the greatest attention.

When other bacteriologists began to test the serum, there was a remarkable want of agreement. Petruschky tested it against the streptococcus of a case of human septicæmia and found that it had absolutely no protective power against this, and furthermore when he obtained from Marmorek, who gave him every help, cultures of his

own streptococcus, he got equally unsatisfactory results. Nor again when he employed it upon patients who had been inoculated with erysipelas in order to cure inoperable cancers did he get any result. Van de Velde obtained like unsatisfactory results. On the other hand Merieux and Niemann confirmed Marmorek's original observations.

How are these contradictory results to be explained? All the observers are men of recognised ability, and it would be absurd to impute to them any bias. Yet there must have been some flaw somewhere. One possibility is suggested by Cobbett, namely, that much depends upon the dose injected. He points out that streptococci differ from other bacteria, in that from their growth in chains they tend to clump together, and thus an injection of 1 c cm. of a 1,000,000 dilution of streptococcus culture into a series of animals will result in some having relatively large doses, while others receive no streptococci at all, Petruschky's tables show that Marmorek's serum was not wholly without results. Thus animals which received a dose of serum insufficient to save their lives, lived ten or eleven days while the controls died in 30 hours.

Another important matter was brought forward by Aronson in 1896, who utilising the streptococcus obtained from the pus of a case of phlegmon, the virulence of which had been raised until it was intensely pathogenic, found that whereas the serum gained from a horse inoculated with this, had a certain protective power, Marmorek's serum employed against the same microbe, was absolutely worthless. Later Merieux and Niemann tested their own serum together with Marmorek's and a serum made by Charrin and Rogers, against several streptococci including that used to produce their serum and that used by Marmorek. They found that Charrin and Roger's serum proved worthless throughout and the other sera were found to be successful, but at the same time their own serum (Serum Vaise) proved eight times as strong against their own streptococcus as did Marmorek's serum, and contrariwise, against the streptococcus of Marmorek Marmorek's serum was twice as strong as was the serum Vaise.

It is possible, therefore, that there is not one form of streptococcus, but several, and that the serum obtained from an animal immunised against one streptococcus is unaffected, or has but slight power when utilised against another. Everything points to this being the case. This matter of the existence of different species of streptococcus, is one that has been debated for long years. Everyone knows that for a long period, the streptococcus of erysipelas was held to be a distinct species from the streptococcus of ordinary suppuration; we now no longer draw this distinction, for no clear demarcation can be found between the two.

Later Lingelsheim studying a large number of streptococci obtained from various conditions, classified them into long and short streptococci (those forming long or short chains.) But we now know that it is easy to transform the long chain into the short chain form, and *vice versa*, thus the view has become almost generally accepted that in man there is only one pathogenic streptococcus, and that all the various streptococcal diseases are caused by a single species which may present modifications in virulence.

Now, however, this work upon sera is showing us that there may be several distinct varieties, and that with regard to the streptococcus we seem to be in the very same position that we were a few years ago with regard to the typhoid bacillus and its allied forms. Several facts may be mentioned in support of this contention. Thus, Van de Velde selected two streptococci, A and P, which both when introduced into the rabbits ear produced erysipelas without as a rule any septicæmic conditions or presence of streptococcus in any of the internal organs. With each of these he immunised a horse, obtained two seras, A and P, each of which he tested against these two microbes, and against the streptococcus of Marmorek and that of Belfanti. Serum A protected against streptococcus A, but had a very much slighter action against streptococcus P, while serum P protected against streptococcus P, but was absolutely useless against streptococcus A. Serum A had no action against streptococcus Marmorek, and possibly a feeble action against the Belfanti streptococcus. Serum P. on the other hand, had a considerable power against streptococcus Belfanti.

Schenk with another streptococcus, obtained the same results. Mérie, found that Marmorek's serum did not protect against a streptococcus from a case of scarlet fever, yet it protected against one given by Marmorek. Courmont tested a serum obtained from Marmorek, together with serum obtained by himself from animals inoculated with Marmorek's streptococcus, against seven different races of streptococci, and he found that this serum protected against Marmorek's streptococcus, but altogether failed to protect against the others, if indeed it did not render them more susceptible to the action of this. Five of these races came originally from a case of erysipelas in man, the others from an abscess.

Lemoine did not get such a large proportion of failures with races of streptococci, nevertheless he isolated a considerable number against which the serum was inactive. Thus it is evident that the anti-streptococcic serum obtained from animals which have been immunised against one race or variety of streptococcus, is incapable of protecting against every streptococcus which is pathogenic in man.

This must be regarded as definitely proved. If it be so, can we recommend the use of antistreptococcic serum, for the treatment of human disease? In attempting to answer this question we have to proceed very cautiously. In the first place, we must I think, admit that in some cases, the serum has been of use. The altered complexion that the disease has taken on almost immediately after the inoculation, has in several cases been so remarkable that the improvement in condition can only be ascribed to the use of the serum, and if there be such cases, then it would be cruel and altogether unscientific to recommend the discontinuance of its employment. On the other hand we have to acknowledge that a very large number of cases do not appear to react against the serum, and this being so, if it could be proved that the injections aggravate the disease, it would be our duty to urge the discontinuance of the treatment.

At the present time I do not think that there is very adequate evidence pointing in this direction; the inoculations in man if they produce no good, at least produce no evil result. At the most it has seemed to me that in one or two cases which more especially have come into my notice, in which the injections were at first attended with remarkable amelioration, there later developed a curious lingering condition, and while the disease was lessened in its intensity, it assumed a more chronic or lingering subacute condition, as though, while the organism had been rendered capable of neutralising the lethal properties of the streptococcus, its power of destroying the microbes had been if anything, lessened. On the whole, therefore, if the serum while incapable of having a beneficial effect upon many cases, can be shown to have no baneful influences, and if in a definite proportion of cases it is of distinct use, we must regard it as being still to be recommended, and must hope that further research will help to give us a medium of greater use in this connection.

Van de Velde makes a very useful suggestion, he recommends that the horse used to supply the anti-streptococcic serum should be inoculated with a succession of cultures of streptococci gained from a number of different cases; in this way, the serum obtained should be active against a large variety of streptococci. This suggestion seems evidently worth carrying into practice.

Cobbett himself is more cautious in his recommendations, he concludes, "We can therefore hope to gain but little evidence of any value from the experimental use of the serum in cases of human disease. The value of the remedy must be first clearly determined by experiments on animals. Until it has been shown beyond the possibility of doubt, that the serum will protect them from, and cure

them of, artificial streptococcal infections; until the opinion of experimental observers is unanimous in its favour, it is not yet ready for the physician. The opinion of experimental observers is by no means unanimous. Much therefore, remains to be done before the serum can be recommended to be used in cases of human disease.

"The possibility of an anti-streptococcic serum has been demonstrated, but the remedy is yet in its infancy. For its infancy the laboratory is the proper nursery.

"The anti-streptococcic serum cannot, then, be recommended in cases of human disease. In the meantime, the physician may reasonably hope to obtain some day a specific remedy for these diseases, justified by the measure of success which the serum has already obtained in the laboratory."

From these remarks we rather take it for granted, that Mr. Cobbett has not seen the serum in action upon a favourable case. Anyone who has, and who has observed the patient, whose life seemed to be a question of but a few hours, suddenly pick up strength, and appear to be saved from the jaws of death, could scarcely give such advice.

I cannot therefore but say in conclusion that the serum is to be used at the present time with the distinct understanding that it is an uncertain agent, and is to be used when other means have failed, as a last resort, and as affording a possibility that life may be saved.

On the Treatment of Malignant Growths by Injections of Mixed Toxins.

MANSELL MOULLIN, M.D., F.R.C.S. "The Treatment of Sarcoma and Carcinoma by Injections of Mixed Toxins."—*John Bale, Sons & Danielsson*, 1898.

Another employment of the streptococcus has caused much interest of late years, especially on this continent, from the observations of Dr. Coley of New York. We have referred to this in previous numbers of the JOURNAL, and will not here give any full account of the results obtained. As with the employment of the serum in septic cases, so with the employment of the streptococcus toxins in cases of inoperable malignant growths, while there have been some brilliant successes, there have been numerous cases in which the treatment by the injections of the living streptococcus or again by the filtered toxins of this microbe, or again by the mixed toxins of the streptococcus of erysipelas and the bacillus prodigiosus, have failed to effect any amelioration of the condition.

To those interested in this treatment, we would recommend the above pamphlet, which is an enlargement of a paper read before the Harvian Society of London.

Dr. Mansell Moullin's conclusions are as follows:—

1. It cannot be denied that there is a considerable number of cases in which sarcomata, that had been given up as hopeless, often after repeated operations, have absolutely and entirely disappeared under this method of treatment. There is no other method of treatment (except inoculation with the streptococcus of erysipelas itself) of which this can be said.

2. Some of these cases have remained free from recurrence for upwards of three years, the period which, in the case of excision of the breast for scirrhus, is regarded by many operators as justifying the use of the term cured.

3. Several of the cases in which sarcomata have disappeared after an attack of erysipelas, have remained free from recurrence for seven years and upwards.

4. The fact that there may be a few—a very few—cases recorded in which sarcomata have disappeared, either spontaneously or after such diseases as acute specific fevers, has nothing to do with these conclusions. (The statement that sarcomata do occasionally disappear has been made but well authenticated cases in which this has taken place, verified in the way in which Dr. Coley's has been verified, are very difficult to find.)

5. Nor are the conclusions in any way invalidated by the fact that injections of the mixed toxins are sometimes followed by the disappearance of other growths, such as lupus, keloid, syphilitic deposits, carcinomata, etc. It may make the disappearance of sarcomata more difficult to understand, but in no way disproves it.

6. The proportion of cases of sarcomata that are cured by the injection of the mixed toxins, depends among other things on the histological character of the growths. Spindle-celled sarcomata are by far the most successful. This suggests the conclusion that the mixed toxins have a selective action, even if it is not specific.

7. The disappearance of sarcomata is not due to inflammation but to an intensely rapid form of fatty degeneration, comparable only to that which affects the hepatic cells in acute yellow atrophy of the liver. Inflammation and sloughing when they do occur, are septic complications.

8. Degeneration and absorption may occur, whether the toxins are injected directly into the tumours or into some distant part of the body. In the former case, however, the constitutional symptoms are more severe and the effect more rapid.

9. The method is attended by a considerable degree of danger. It should therefore only be adopted in those cases in which there is no other remedy. The chief risk appears to be from collapse and pyæmia. There must always be danger of the latter if there is a suppurating or a sloughing sore. It may be argued that patients whose lives are immediately threatened by a malignant growth will never be cured by any remedy that does not involve some degree of risk.

10. The toxins are of no use unless the cultures are taken from a virulent case of erysipelas or are made virulent by passing the streptococcus through rabbits.

11. The bacillus prodigiosus in spite of theoretical objections, has the effect of immensely increasing the reaction.

12. The effect is most striking in the case of rapidly growing sarcomata. Slowly growing ones appear to have much more resistance. Probably this merely means that masses of embryonic cells with little stroma give way to injurious influences more readily than those that are more closely knit together.

13. Patients often gain in weight and strength while under treatment.

14. Treatment should be continued till the whole growth has vanished or has become so small that it can be removed.

15. If there is a recrudescence of the disease, it does not follow that the toxins will be as efficacious the second time as they were the first. Whether this is the result of tolerance being established cannot be said.

16. Growths of a similar character may spring up in other parts of the body after many years.

17. The severity of the reaction is very variable. Probably this depends upon the rapidity with which the injection is absorbed, rather than upon any cumulative action it may possess.

The disappearance of the growths is not the result of the high temperature. High temperature due to other causes, such as specific fevers, is not followed by this result.

Coley suggests that injections of the mixed toxins may be useful in preventing recurrences after sarcomata have been removed by operation.

Incidently it may be mentioned that injections of the streptococcus of erysipelas apparently never cause suppuration. If, therefore the streptococcus of erysipelas is identical with the streptococcus pyogenes, the name of the latter had better be changed.

The only objection—and it is a most serious one—which we have to offer to Moullin's own work, is that his diagnosis is purely clinical, unsupported by microscopical examinations.

To us, a very equally valuable portion of the pamphlet is the appendix, in which Dr. Mansell Moullin has collected together with great thoroughness all the cases recorded in literature.

1. In which, in cases of sarcoma and carcinoma, erysipelas has broken out accidentally. (21 cases.)

2. In which erysipelas has been caused intentionally (17).

3. In which the toxins of the streptococcus, alone, or in conjunction with those of the bacillus prodigiosus, have been employed. This last list includes cases from American, French, German, Italian, and English literature.

J. G. Adami.

Canadian Medical Literature.

UNDER THE CHARGE OF KENNETH CAMERON.

[The editors will be glad to receive any reprints, monographs, etc., by Canadian writers, on medical or allied subjects (including Canadian work published in other countries) for notice in this department of the JOURNAL. Such reprints should preferably be addressed to Dr. Kenneth Cameron, 913 Dorchester street, Montreal.]

The Canadian Practitioner.

February, 1898.

1. The Prognosis in Cardiac Disease. J. E. GRAHAM.
2. Spontaneous Rupture of a Fatty Heart. W. J. GREIG.
3. A Note on Death from Chloroform. R. D. RUDOLF.
4. The Selection of an Anæsthetic. H. D. SCADDING.

March, 1898.

5. The Great Omentum. J. G. ADAMI.
6. Suppurative Cholangitis. H. PARSONS.
7. Two Cases of Tubercular Pleurisy with Effusion. H. H. OLDRIGHT.

April, 1898.

8. Disease of the Coronary Arteries and its Effects. G. SILVERTHORN.
9. Dicephalous Monster. W. H. PEPLAR.
10. Anæsthesia and Analgesia. H. H. OLDRIGHT.
11. The Surgical Treatment of Insanity. E. HALL.

1. GRAHAM discusses, at length, the prognosis of heart disease. In early life, valvular lesions are the result of endocarditis, and the prognosis during the attack depends largely upon the nature of the infective agent. If due to rheumatism, the immediate prognosis is favourable, but there is a liability to its recurrence. When as a result of scarlatina, it is not likely to recur, and hence the prognosis is more favourable than when due to rheumatism. Murmurs occurring during an attack of rheumatism frequently disappear, so the attending physician should not alarm the friends by predicting serious heart disease. When, however, the murmur lasts from the beginning to the end of the attack it will never disappear in after life. The question often arises: How long will complete compensation last after it has once been completely established? To determine this, it is necessary to find out, if possible, the extent of the lesion, the length of time during which it has existed, the changes which have taken place in the heart itself as a result of the lesion, the condition of the other organs of the body, the temperament, mode of life, habits and calling of the patient, hereditary tendencies and presence or absence of other

disease. After a full discussion of the physical signs of the various lesions, the comparative gravity is stated in general terms to be, commencing with the gravest, tricuspid insufficiency, aortic insufficiency, mitral stenosis, aortic stenosis, and mitral insufficiency. The social condition of the patient is an important factor in prognosis. If he is of regular habits, does not use tobacco, is either a total abstainer or a very moderate user of alcohol, possesses an even temper, is one whose calling requires a moderate amount of regular exercise, who lives among healthy surroundings and who has freedom from worry, he stands the best chance of a long life. Valvular lesions which originate after the middle period of life are usually the result of various forms of degeneration, and are often the direct result of an infection. When systolic or diastolic aortic murmurs occur in an elderly individual, and are accompanied by fever, a very guarded prognosis should be given as to the immediate results. There is a class of cases in which there is serious disease of the myocardium, when no valvular murmurs can be heard, and may be due to local or general fatty degeneration. The diagnosis of such conditions is extremely difficult, and on that account a prognosis is not usually made. One of the most frequent questions asked by a patient and his friends is with regard to the possibility of sudden death. If the patient can be assured that in his particular form of disease sudden death is not probable, it will have a wonderful influence in relieving his mind. In all cases of advanced disease there is a possibility of sudden death, but it may be safely stated that there is no danger from sudden death, in the sense of falling down dead, in any of the valvular lesions except aortic incompetence. In all the class of cardiac neuroses, palpitation, brachycardia, tachycardia, the most careful examination should be made to exclude organic cardiac disease before a prognosis is given. It will then depend upon whether the cause can be removed or not. Arythmia is more grave than palpitation.

2. GREIG exhibited before the Toronto Pathological Society, the heart of a woman who had died suddenly while he was in the act of passing a soft rubber stomach tube. There was advanced fatty changes with rupture of the wall. The appearance of the woman was not such as to lead one to suspect degenerative changes. She was not very stout, and there was no disease except the gastritis which she was being treated for at the time of her death.

3. RUDOLF presented before the Toronto Pathological Society, frozen sections of a dog which had died during the early administration of chloroform. There was enormous distention of the right side of the heart, both auricle and ventricle. The dog had been chloroformed in a thin sack, had struggled violently at first, breathing hard,

but suddenly the breathing became shallow and ceased. He was removed, but efforts at resuscitation were without avail. The heart could be felt beating for several minutes after natural breathing had ceased. The writer had found that some dogs were much more susceptible to the drug than others, and he suggests that the very contrary results obtained by very trustworthy observers in different quarters of the world might be explained by the use of dogs differing widely in their natures, and adds that possibly the different races of mankind and the different individuals of the same race might vary in a like manner.

4. SCADDING, writing of general anæsthetics says, that we should use the anæsthetic which is the safest, providing the nature of the operation and its method of operating offer no contrary indication. Nitrous oxide, is admittedly the safest of all general anæsthetics, but is inapplicable for prolonged administration. Next to it, no one in the face of our present knowledge, will deny that ether is safer than chloroform. He follows closely Fredk. Hewitt's comparison between the two agents and classification, and is convinced of the superiority of the method of anæsthetizing with gas followed by ether in a closed inhaler.

5. ADAMI remarks upon the little attention that has been paid to the great omentum; there is a scanty literature upon its pathological condition, mainly upon cysts and tumours found in it. After reviewing the theories of the older writers he discusses what is now known concerning it and its function or functions. There is one feature about the great omentum which is the all-important feature histologically and physiologically, and that is the main characteristic of its structure. It is a double membrane folded upon itself, and fused together, consisting of endothelial and connective tissue elements. However delicate and however loaded with fat, its main characteristic is its great vascularity. The vascular supply is altogether in excess of the needs of the membrane itself. The great omentum may be regarded as a mechanism for supporting and keeping in position a rich aborization of delicate vessels separated by as slight a cell-layer as possible from the peritoneal cavity. The viscus may be endowed with powers of locomotion and so soon as any localized injury and inflammation manifests itself, forthwith some portion of its border, makes its way to the affected area, and within a little time becomes adherent over it and thus helps to prevent the spread of inflammation. This protective power does not seem to be present in all cases, for in typhoid fever we find that a serous rather than a leucocytic and fibrinous exudation obtains, and then no adhesions take place even though the omentum be lying over the area of injury.

The records of 150 consecutive and unselected cases are given, and illustrate the fact that the omentum may gain attachment to every viscus lying in the abdominal cavity, and does so very frequently.

6. PARSONS reports the case of a woman, aged 45, in whom there was found suppuration in the upper bile passages, and extension of the process into the surrounding tissues, with abscess formation and general peritonitis.

7. OLDRIGHT describes two cases of pleurisy with effusion, with interesting peculiarities. In one the vocal fremitus, bronchial breathing and vocal resonance could be felt and heard through the effusion which extended from the level of the right nipple to the base. This may be accounted for by the compression of the lung as far as the bronchus and the density of the fibrous effusion, which clotted on being withdrawn. In the second case the peculiarity was in the paralysis of the right serratus magnus muscle, which came on some weeks after thelast aspiration.

8. SILVERTHORN discusses the diseases of the coronary arteries, and describes a case of rupture of the left ventricle in a man of sixty years of age. The progress of events in the heart could be easily surmised. There had been atheroma of the left coronary artery, and which was most marked in its right branch. A thrombosis of the vessel occurred as a result of the rough calcareous plates found in the artery and the partial occlusion of the vessel. There resulted an anæmic infarct of practically the full depth of the muscular tissues on the anterior surface of the left ventricle. The blood pressure was at first sufficient to tear asunder near the apex some softened or necrotic fibres of the wall, including some columnae carnae, and four days later complete rupture occurred.

9. PEPLER describes an incomplete dicephalous monster. It had two perfectly formed faces placed side by side on a single head, one face looking to the front and right, the other to the front and left, the head was slightly broader than that of a fœtus at term. The body and limbs were well developed and normal.

10. OLDRIGHT discusses that subject of never-failing interest to the profession, anæsthesia. He covers the ground very fully, but does not add anything to what is already well recognized.

11. HALL reports the brief history of a woman who had spent two years and eight months in an asylum, "a hopeless case of insanity." Under an anæsthetic an examination revealed extensive pelvic disease. The appendages were removed and the uterus curetted. Convalescence was rapid, and thirty-five days after the operation she returned to her home with all the reason and energy of her former self. Arguing from the results of this case, and from the work of Hobbs of London,

and Rohé of Baltimore, Hall strongly advocates the careful examination by men experienced in those classes of cases, of all the female inmates of the various asylums.

Canada Medical Record.

February, 1999.

1. Report of Two Cases in which a Fibroid Tumour was Expelled from the Uterus after Electrical Treatment. A. LAPTHORN SMITH.

March, 1898.

2. Artificial Anus Closed Five Months after Colostomy. F. R. ENGLAND.
3. Aneurism of the Ascending Portion of the Aorta. J. B. MCCONNELL.

April, 1897.

4. Valedictory Address to the Graduating Class, 1898, at the 26th Annual Convocation of Bishop's College, Faculty of Medicine. J. W. STIRLING.
5. Valedictory of the Graduating Class, 1898, University of Bishop's College, Faculty of Medicine. MACD. FORD.

1. These cases have already been reported in the April number of this JOURNAL (page 304) in the proceedings of the Montreal Medico-Chirurgical Society.

2. ENGLAND describes a case that came under his care in the Western Hospital. The patient, a boy of fifteen, had, five months before, symptoms of appendicitis for which he had been operated upon. The cæcum and appendix were found normal, but a tumour of considerable size was discovered blocking the rectum, and situated about four inches from the anus. The mass was looked upon as tuberculous. A left iliac colostomy was performed. The symptoms were relieved. Since the operation, the bowels had acted two or three times a day by the artificial anus. The boy wished to have the normal relations of the bowels restored.

After thorough examination to determine if the lower bowel was pervious, the fistula was separated and the bowel freed from the parietes. The omentum was found closely adherent to the abdominal wall and to the intestines. These adhesions were freed and the opening closed transversely after the method of Heinecke. No tumor or thickening could be discovered in the sigmoid or rectum. The boy made an uninterrupted recovery, and his bowels move freely twice daily without medicine.

3. MCCONNELL relates the history of a woman, aged 32, who presented all the typical signs and symptoms of aneurism. At autopsy the site of the aneurism was found to be the ascending arch, commencing just above the aortic valves and reaching nearly to the origin of the innominate artery. The tumour itself measured six inches in diameter.

Kenneth Cameron.

Reviews and Notices of Books.

Brief Essays on Orthopædic Surgery. Including its Relation to General Surgery, its Future Demands, and its Operative as well as the Mechanical Aspects, with Remarks on Specialism. By Newton M. Shaffer, M.D., Surgeon-in-Chief to the New York Orthopædic Dispensary and Hospital; Clinical Professor of Orthopædic Surgery, University of New York City (Medical Department); Consulting Orthopædic Surgeon to St. Luke's and the Presbyterian Hospitals, New York; Consulting Surgeon, New York Infirmary for Women and Children; Member New York Orthopædic Association, New York Academy of Medicine, New York Neurological Society, etc. New York: D. Appleton and Company. 1898.

A very neat little book of 81 pages, comprising essays which have appeared at various intervals during the past fourteen years.

Dr. Shaffer first gives, briefly, a very interesting history of the development of this department of surgery into a specialty, showing at the same time the very great activity of American surgeons in this interesting field. Orthopædic surgery is defined as "that department of surgery which includes the prevention, the mechanical treatment, and the operative treatment of chronic or progressive deformities, for the proper treatment of which special forms of apparatus or special mechanical dressings are necessary." A sound warning is given against the allurements of general surgery, and it is clearly pointed out that it requires an exceptional man with a decidedly mechanical bent to succeed in orthopædic work. The author recommends that after graduation and a term of service as *interne* in a hospital, a course of study of at least five years should be demanded of those who expect to become orthopædic surgeons.

The scope, present needs and future demands of orthopædic surgery are discussed in an entertaining and instructive style. G. E. A.

Inflammation of the Bladder and Urinary Fever. By C. Mansell Moullin, M.D., Oxon., F.R.C.S., Surgeon and Lecturer on Surgery at the London Hospital; Examiner in Surgery at the University of Oxford; late Radcliffe's Travelling Fellow and Fellow of Pembroke College, Oxford, and Hunterian Professor at the Royal College of Surgeons. London: H. K. Lewis, 136 Gower Street, W.C. 1898.

A small book of 153 pages written with the avowed object of proving or rather emphasizing the fact already proved, that inflammation of the bladder is always due to micro-organisms, which in the vast majority of

cases have invaded it from without; and that what is commonly called urinary fever is nothing but septic intoxication or septic infection occurring under special conditions.

A few anatomical and physiological conditions in connection with inflammation of the bladder first receives attention. It is pointed out that there are no glands in the mucous membrane lining the bladder, and that the mucus seen in normal urine is derived from the urethra and its accessories; that the thick layer of so-called mucus or muco-pus which adheres to the bottom of the vessel in certain cases is composed of pus altered by an alkali together with micro-organisms, epithelium and debris and has no mucin in its composition.

The description of the different forms of cystitis is based on their clinical features and is instructive and well written. A very interesting fact, very clearly brought out is that while, unlike the urethra, the healthy bladder does not absorb drugs or ptomaines, yet when diseased both are rapidly absorbed into the system.

The chapter on urinary fever embodies the facts of this interesting and often very grave condition, and the directions given for catheterisation of the distended bladder and the introduction of catheteric life, in prostatic retention are such as should be more fully appreciated than is sometimes the case. While it is true that this book contains little that is new, it is full of facts that need emphasizing, and it is to be highly recommended as a most useful and helpful work. G. E. A.

Yellow Fever in the West Indies. By IZETT ANDERSON, M.D. London: H. K. Lewis, 1898. Small 8vo. 106 pages.

This little work is of interest in that in a very modest manner it gives the author's observations and conclusions upon an experience of Yellow Fever in the West Indies, extending over more than 30 years. It does not pretend to contain anything very novel, the main object of the writer being throughout to be of service to young practitioners and others settling in the West Indies, or being called upon to diagnose and treat the disease without much previous experience of its course and characters.

Dr. Anderson gives a very clear account of the stages of this curious fever, and dwells much upon the importance of recognising its first or febrile stage, and the mitigation or alleviation of symptoms which usher in a second stage, the fall in temperature, diminution of the headache and pains of back and limbs, the diminished thirst, the quieter pulse, and the more cheerful condition of the patient. He dwells with special importance on the character of the vomit, the white or acid vomit at the beginning of the second stage followed by the development of the black vomit and development of hæmorrhages in other parts of the body. Most particularly is he of opinion that the study of the urine is the mos important, in prognosis and in the recognition of the gravity of the case.

According to him albuminuria is the one constant feature ; it may be found as early as the first day and most commonly appears on the second or third day of the disease, although he acknowledges that in four out of 31 cases it was only manifested on the 5th day, and he recommends the daily examination to determine the amount of albumin as giving information as to the condition of the patient. He advises that the urine of one day, that has been treated by boiling and the addition of nitric acid, be kept until the next, to give a rapid means of comparison of the progress of the albuminuria.

The treatment which he has found most successful and which he has pursued for many years, has been the steady administration of carbolic acid and potassium bicarbonate in effervescents with lime juice throughout the whole course of the disease and until convalescence is established. It is interesting to note that Sternberg strongly recommends a somewhat similar mixture in which the perchloride of mercury takes the place of the carbolic acid as the antiseptic employed. J. G. A.

THE

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ON MILK AS A DIET IN TYPHOID FEVER.

Thoughtful physicians in all ages have recognized the influence which a judiciously arranged dietary has in modifying diseased processes. Even in the early days of the healing art, the great Hippocrates in his teachings constantly emphasized the importance of a carefully regulated diet for the sick, and in more modern times the illustrious Graves considered that his chief claim on a grateful posterity lay in the fact that he had taught the profession to feed fevers. Physicians of the present day with their clearer insight into the physiology and chemistry of the tissues, are in a better position to appreciate the importance of a diet which, while supplying in an assimilable form sufficient nutriment to sustain, shall yet not place additional burdens upon overtaxed excretory organs, or by the formation of toxic products within the intestinal tract lead to still further intoxication of the system.

In no form of illness does the character of the nourishment supplied, and the amount appropriated, influence the course and outcome of the disease to a greater extent than in typhoid fever. For some years past milk administered in small quantities and frequently, has formed the routine feeding of typhoid fever almost all over the civilized world.

Very recently, however, this indiscriminate routine feeding of all patients suffering from typhoid, on milk, has had several vigorous opponents. Amongst others, Dr. Burney Yeo, of London, Dr. Shattuck, of Boston, and Dr. Barr, of Liverpool, have stongly protested against this rigid routine. They have questioned whether the large amount of solid fæcal matter frequently found as a result of this continued milk diet, is not a cause of some of the perforations, which occur in the later weeks of the disease, and whether with such

a large amount of residual matter a diet exclusiuely of milk, affords a sufficient amount of nutriment to the tissues. They have, therefore, insisted that the fæces should be carefully watched for indications of imperfect digestion, and that occasionally a more generous dietary better sustains the nutrition and strength of the patient.

We have much pleasure, therefore, in publishing in this number a paper read before the McGill Undergraduates Medical Society, in which the propriety of always adhering to this diet in typhoid fever is discussed. While by no means agreeing with all the statements made, we feel convinced that an exclusive milk diet in this disease, by no means fulfils all the requirements of the case, and that a thoughtful discussion of the matter may do good.

In any continued fever we have undoubtedly to deal with an impaired secretion from all the secreting glands of the alimentary tract, the salivary, the gastric, the pancreatic, the duodenal, and the intestinal. According to Sir Wm. Roberts, gastric digestion is, however, distinctly more impaired than is intestinal digestion. The sympathy of the stomach with the general condition of the system is much more active and close than that of the intestines; the former organ approximates more nearly to the animal life of the body, the latter to the vegetative life. The stomach ceases to be the principal organ of digestion and becomes more or less of a conduit to pass on liquid foods to the duodenum. We have thus a reversion to a condition more or less infantile in character. At the same time the appetite fails for solid food, which if forced upon the stomach, quickly undergoes fermental changes, and may give rise not only to irritation and discomfort, but, by the formation of toxins, may still further depress the system. With this disturbance in the digestive functions of the patient, we also know that all febrile states are accompanied by an increased waste of the tissues of the body; a waste that appears to effect chiefly the muscular tissues, and only in a minor degree the fatty.

This waste appears to be due not merely to a deficiency in the amount of nutriment supplied, but also to an impaired power in the tissues to assimilate nutriment during the higher grades of pyrexia. Although, therefore, we may effect, by the administration of suitable albuminous food in sufficient quantities, a distinct saving in the waste of tissue, yet, owing to this defective power of assimilation at high temperatures it would appear to be impossible to maintain a nitrogenous equilibrum in patients with continuous high fever. Failure in nutrition is, however, much less marked in patients whose fever is of a markedly remittent or intermittent type, for as Bauer writes,

"the cellular elements recover during the periods of remission the power of adding to their substance and of metabolizing an excess of nutrient material."

While, therefore, it is extremly important to supply our fevered patients with albuminous food which can be utilized in checking waste of tissue, such food must be supplied in a form which can be readily absorbed and assimilated, and an effort should be made to utilize the periods of remission for giving nutriment in increased quantities, as it can at these periods be better digested and appropriated. During all pyrexial conditions an abundance of water favours the processes of absorption and elimination.

We are still of the opinion that for a majority of our patients, no food will so satisfactorily fulfil the above conditions as will fresh uncontaminated milk. It supplies all the elements needed for the nutrition of the tissues. It is cheap, and always readily obtainable. The slight deficiency in the carbohydrates is easily filled by the addition of a thin farinaceous gruel. In many instances the lack is made up by the administration with it of alcohol. Milk should, however, in all severe cases be freely diluted. with water, or a thin gruel, or a weak tea or coffee; it should be rendered alkaline by the addition of lime water or sodium bicarbonate (ten or twenty grains or more to the pint), and should be given in small quantities at intervals of an hour or two hours. While a patient is on a milk diet a watch should be kept for symptoms of indigestion, as indicated by nausea, flatulence, and in some cases constipation. The motions should be carefully examined. Milk when given carelessly is liable to form in many stomachs a solid curd, indigestible either in the stomach or small intestines. Such curds in their passage through the intestines must be a source of much irritation and call for the more muscular effort on the part of the intestinal wall. Such a condition is especially to be avoided in typhoid fever, when either unnecessary irritation or unnecessary muscular effort in the neighbourhood of the inflamed and ulcerated patches may lead to serious results.

In cases where milk is distasteful, we may supplement it by, or substitute for it, any of the simple meat broths. The small amount of nutrition which they possess may be increased by the addition of vegetable juices, a point emphasized many years ago by Sir William Jenner. Such soups may be variously flavoured with aromatic herbs rendering them more grateful to the patient and more easy of assimilation. The various preparations of gelatine give us readily assimilated albuminous foods which prevent tissue waste. Some of the

starchy foods may also be employed with care, especially if they are combined with one of the malt extracts.

In the later stages of the disease, when the fever remits for many hours at a time and appetite returns, many good authorities strongly recommend the administration of stronger food. We, too, would echo this plea and would extend the dietary at this period to finely minced meat, eggs, soft puddings, and stale bread or toast for those who, with a clean tongue, and a keen desire for food, show no special signs of intestinal irritation.

HOSPITAL ABUSE.

An article read before the Montreal Medico-Chirurgical Society by Dr. Armstrong, will be found on another page of the present number. It introduces for the first time in this JOURNAL a subject which has been claiming the attention of physicians wherever the hospital system is found, and which in many respects seems to be as far from a solution as ever. How to discriminate between those, who, we feel, are deserving of the gratuitous services of our profession, and those who, while not entitled to them, can, by the present constitution of hospital regulations, demand such services, is perhaps one of the most difficult points to be solved. Dr. Armstrong's suggestion of a central hospital board would seem to meet this difficulty and should be given a trial. The discussion on the paper was considered to be of such importance that it was deferred until the autumn, when a more representative gathering of medical men could be obtained than during the present month. We shall be glad to publish the views of our readers on the matter and invite correspondence from those interested in the subject.

THE FORTHCOMING ELECTION AT THE COLLEGE OF PHYSICIANS AND SURGEONS OF THE PROVINCE OF QUEBEC.

The temper of the profession at large in this province in respect to the conduct of the business of the College of Physicians and Surgeons under the present regime has been so clearly shown during the last three months, that it is not necessary for us once again to point out the urgent need of reform. The profession at large has determined that the present thoroughly unsatisfactory and discreditable state of affairs must cease, and we now know that the majority is in favour of reform. *As proxies can be used solely for the election of governors and the vote of those present at the meeting decides all other questions* those who have given proxies must not individually relax their efforts

or think that it will be safe to absent themselves on July 13th. We must be present without fail for the opening of the meeting at 9 a.m. prepared to give up the entire day, and not saunter in at mid-day when the college has already been in session for some hours. We have to deal with those who are fighting for life and for position, with all that such position implies; and we have already had experience during the course of the present contest, that in order to gain votes and retain their positions sundry of the officials of the present Board are prepared to resort to practices which are not to be commended.

It must not be forgotten that under the present method of voting it is the easiest thing in the world for those politically minded to completely control the election. If the Board be united it is a comparatively simple matter for each of the 30 or so elective members to obtain personal proxies from ten or twenty professional friends, the proxies being obtained primarily in order to assure the return of the individual member, and when obtained, being thrown into the common pool, so that the united Board in this way can without difficulty control some four to six hundred votes, a number which is ample to insure the election of any board as now constituted, under ordinary conditions.

Fortunately in this present election the conditions are not ordinary, and fortunately very many of the more influential members of the Board are so disgusted with the conduct of affairs that they have cut themselves adrift from what until now has been the dominant party. Nevertheless it must not be thought that Dr. Beausoleil and his friends will submit meekly to losing the control over the affairs of the college that they have exercised during all these years, because a majority desires it.

This consideration of how easy it is for a "combine" to gain possession of medical affairs in the province, is in itself sufficient to prove the urgent necessity for a radical change in the mode of election of the Board of Governors. Under the present method if practitioners in any district are dissatisfied with the action of a member of the Board hailing from that district, they are wholly powerless to obtain proper representation. A member has no need to depend upon district support, provided that he is working hand-in-hand with the "combine"; he depends upon the proxies obtained from the other members of the same; while if the district puts forward a candidate as its own nominee, that nominee, if unknown outside his district, may receive but a rare vote from other parts of the province and so in all likelihood will be hopelessly beaten, although he is the choice of his district and receives the overwhelming majority of the votes cast in the same. This has repeatedly occurred in the past.

It may be retorted, and it has been urged that the Electoral Committees, French and English, which are working against the present officials, are themselves a combine and are working for selfish ends. It has been stated by Dr. Beausoleil and his friends, that the Electoral Committees are the result of a plot on the part of the Universities to gain their own ends. We can only say that if this be so, they have gone to work in a very peculiar way, for not a single candidate recommended by those committees is connected with a University staff. The Electoral Committees simply desire to cleanse the Board and to improve its methods, and to render it thoroughly representative and respected—there is no question of the Universities as opposed to the practitioner.

Out of 1,403 licensed physicians the Reform Committee has already received proxies from 830, having a majority in every separate district as well as this general plurality in the whole province. The candidates have been selected by the districts themselves and not by the committee, and it is obvious that if the majority in each district vote with the majority in the other districts success is inevitable unless improper tactics are tolerated.

Before our next issue the meeting will have come and gone. So now for the last time we urge that every member of the profession who has the good of the profession at heart, will be present either in person or by proxy at Laval University, Montreal, at 9 a.m. on the morning of July 13th, and will do his utmost to put an end to our present discredit.

THE INTERNATIONAL ASSOCIATION OF RAILWAY SURGEONS.

Officers for 1897-8: President, Surg. Geo. Ross, Richmond Va.; Vice-Presidents, Surg. J. Alex. Hutchison, Montreal, Can.; Surg. A. L. Fulton, Kansas City, Mo.; Surg. De Saussure Ford, Augusta, Ga.; Surg. John J. Buchanan, Pittsburg, Pa.; Surg. H. L. Getz, Marshalltown, Ia.; Surg. R. R. Lawrence, Hartford, Mich.; Surg. W. Q. Marsh, Sierra Mojada, Mexico. Secretary, Surg. Louis J. Mitchell, Chicago, Ill. Treasurer, Surg. Eugene R. Lewis, Kansas City, Mo. Executive Board, Surg. A. I. Bouffleur, Chairman, Chicago, Ill. Committee on Transportation, Surg. W. B. Outten, Chairman, St. Louis, Mo. Local Committee of Arrangements, Surg. B. L. Riordan, Chairman, Toronto, Ont.

Above are the names of Officers of International Association of Railway Surgeons for this year.

The meeting will be held in Toronto on Wednesday, Thursday and Friday, July 6th, 7th and 8th, 1898. These dates have been decided on so as not to interfere with other important medical meetings in the United States and Canada; and also because the weather will be very warm in the country to the south of us about that time, so that a large number of members may be expected to come north and enjoy the cool breezes of Lake Ontario.

This—the eleventh annual meeting—will be the first one held outside the borders of the United States.

The purpose of the Association is stated in Article 1, Sect. 2, of the Constitution, viz.:

"The object of the Association shall be to promote acquaintance and fraternal relations among railway surgeons, to secure interchange of ideas and the adoption of the best methods of development and improvement of railway surgery, and to establish it as a special branch of the chirurgical art."

At present there are about six hundred members paid up for this year, but, as many join at each meeting, a much larger number is expected. Canadian Railway Surgeons have not been very numerous at the meetings of the Association heretofore, about forty being the largest number during any year; but a much larger representation is looked for this year.

Free transportation will be granted to members of the Association connected with railways to and from Toronto upon application through the proper officers of the Company employing such surgeons, C. M. Hays, Esq., General Manager of the Grand Trunk R'y. System has kindly signified his desire to give an excursion to the members attending the Association meetings from Toronto to the Muskoka Lake district. This excursion will probably take place on Saturday, the day following the closing of the meeting. Other entertainments of a social nature are being arranged for.

The Chairman of the Committee of Arrangements would be pleased to hear from any Canadian Railway Surgeon who would favor the meeting with a paper.

Application blanks for membership will be forwarded by the Treasurer of the Association, E. R. Lewis, Kansas City, Mo.

The President has suggested the subject of "Shock" for the special consideration of the coming meeting. Different aspects of the subject will be presented by members already chosen by the President. A daily journal, "*The Railway Surgeon*" will be published during the meetings.

An exhibition of surgical appliances and physicians supplies, gener-

ally will be a feature; at Chicago last year thirty-nine manufacturers had exhibits, some firms having four or five representatives present to show their goods. The duty being now removed from surgical instruments should ensure a large show of these goods by foreign manufacturers.

GESTA MEDICORUM.

"QUICQUID AGUNT MEDICI NOSTRI FARRAGO LIBELLI."

The Maritime Medical Society also meets at Halifax during the same week. Dr. D. A. Campbell is the President-elect.

The Medical Society of Nova Scotia meets this year at Halifax, on Wednesday, July 6th. Dr. William Tobin of that city is the President-elect.

The triennial meeting of the College of Physicians and Surgeons for the election of the Board of Governors, will be held on Wednesday, the 13th of July next, at Laval University, St. Denis street, Montreal, at 9 o'clock a.m. A. G. Belleau, M.D., Quebec, and A. T. Brosseau, M.D., Montreal, Secretaries.

A new Society has been added to the many already existing. It is called the Medico-Psychological Society of Quebec. It is composed of 20 members who are attached to the various Insane Asylums in the province, and it will be devoted to the study of mental disorders.

Dr. Arthur Vallée, of the Quebec Insane Asylum, is the President; Dr. T. J. W. Burgess, of the Protestant Hospital for Insane, is Vice-President; Secretary, Dr. E. P. Chagnon, of Longue Pointe.

An Association of Medical Librarians was organised at a meeting of a number of representatives of medical libraries held in Philadelphia on May 2nd. The officers elected were: President, Dr. George M. Gould, Editor of the *Philadelphia Medical Journal*; Vice-President, Dr. J. L. Rothrock, of St. Paul. Minn.; Secretary, Miss M. R. Charlton, of McGill University, Montreal; Treasurer, Dr. William Browning, of Brooklyn, N.Y.

There seems to be a revival of literary activity among medical men. One or two doctors recently have produced very readable novels, if indeed they should not be classed in the ranks with Goldsmith, Smollet, Lever, Holmes and Conan Doyle.

Dr. S. Weir-Mitchell's "Hugh Wynne" is one of the hits of the season. The latest addition to the list is Dr. M. Greenwood's "John Armstrong" which is highly spoken of.

According to the *Universitats-Kalender* recently published, the number of medical students in the universities of Germany and the German speaking universities of Austria and Switzerland are as follows:—Vienna 1,508, Munich 1,396, Berlin 1,360, Würtzburg 742, Leipzig 724, Graz 521, Erlangen 434, Freiburg 392, Zurich 353, Breslau 345, Strassburg 329, Griefswald 293, Geneva 289, Halle 265, Bonn 264, Kiel 263, Tübingen 255, Marburg 249, Göttingen 236, Königsberg 235, Giessen 224, Bern 208, Heidelberg 203, Jena 198, Lausanne 157, Basel 149, Rostock 106.

Honors are coming thick and fast upon McGill men. We are pleased to learn that Prof. J. G. Adami has been elected a Fellow of the Royal Society of Edinburgh.

Dr. D. J. Evans, Demonstrator of Obstetrics, was, in January last, elected a Fellow of the Royal Obstetrical Society of London.

To Dr. Osler the bulk of the honors have fallen, however. He has been recently elected a Fellow of the Royal Society, and is also to receive the degrees of D.C.L. from Trinity University, Toronto, and LL.D., from Aberdeen.

The annual meeting of the Canadian Medical Association will be held in Quebec this year on August 16, 17 and 18. Medical men throughout Canada are looking forward with much interest to the event, and it is expected to be very largely attended. The membership in Canada is something like six hundred, and all the prominent physicians in the Dominion are numbered among them.

The present officers of the Association for the province are as follows : President, Dr. J. M. Beausoleil, Montreal ; Vice-President, Dr. C. F. Park, Quebec ; and Secretary, Dr. Marois. The General Treasurer is Dr. Small, of Ottawa, and the General Secretary, Dr. Starr, of Toronto.

One of the most successful of recent conventions was the meeting of the Ontario Medical Association in Toronto on June 1st and 2nd.

The address of the President, Dr. Wm. Britton, was a thoughtful effort dealing with matters of general medical interest. He expressed the wish that the scheme for intra-provincial registration would become an accomplished fact.

An important paper was that of Dr. R. Ferguson of London, on "The Injurious Effects of Over-wrought School System on the Health of Public and High School Pupils."

The Association passed a series of resolutions calling the attention of the Government to the fact that pupils were expected to overtake too much work and had too much home-work.

Dr. A. T. Rice of Woodstock, reported a remarkable case of "Vicarious Urination." The patient was hysterical and for a period of three weeks, presented suppression of urine. During this time there exuded three times a day from the skin of the legs below the knees a fluid having a urinous appearance, which underwent ammoniacal fermentation and contained urea. One pint to a quart was excreted each time. On Oct. 30th, 1897, a gallon was excreted in the same way in half an hour. It necessitated the patient surrounding the limbs with wrappings. The author also referred to a few other cases in the literature, but his was the most characteristic.

Dr. W. McKeown of Toronto, read a paper on "The Application of the Principles of Osmosis to the Treatment of Toxæmia." His method was to give large subcutaneous injections of normal saline and at the same time rectal enemata of magnesium sulphate. He believed that this increased the elimination of toxins by the bowel, and had treated a case of septicæmia in this way with good results.

Dr. A. McPhedran of Toronto, reported 20 cases of sporadic cretinism occurring in Ontario, with lantern views.

Prof. J. G. Adami of Montreal, read a paper upon the "Hepatic Complications of Syphilis."

The president entertained the members on June 1st, to a smoking concert, and the Royal St. Lawrence Yacht Club to luncheon.

About 180 members were present.

NEW BOOKS, ETC., RECEIVED AND NOTED.

Manual of Operative Surgery. By H. J. Waring, M.S., M.B., B.Sc., F.R.C.S. Edinburgh and London, Young J. Pentland; New York, The MacMillen Company, 1898.

A Manual of Instruction in the Principles of Prompt Aid to the Injured including a Chapter on Hygiene and the Drill Regulations for the Hospital Corps, U.S.A. By Alvah H. Doty, M.D., second edition. New York, D. Appleton & Company, 1898.

A Treatise on Aphasia and other Speech Defects. By H. Charlton Bastian, M.A., M.D., Lond., F.R.S. London. H. K. Lewis, 1898.

A Manual of Hygiene and Sanitation. By Seneca Egbert, A.M., M.D. Lea Brothers & Co., Philadelphia and New York, 1898.

Clinical Report of the Rotunda Hospitals for One Year, Nov. 1st, 1896, to Oct. 31st, 1897. By R. Dancer Purefoy, M.D. Master; and T. Henry Wilson, Henry Jellett. R. P. R. Lyle, Assistant Masters. Dublin, John Falconer, 1898.

The Pathology of Ante-Natal Life—An Address delivered before the Glasgow Obstetrical and Gynæcological Society. By J. W. Ballantyne, M.D. Reprinted from the Glasgow Medical Journal, April, 1898.

A Clinical Study of Kryofine. By Sydney V. Haas, M.D., and J. Bennett Morrison, M.D. Reprinted from the New York Medical Journal, March 25th, 1898.

Injuries from "Live" Electric Light and Trolley Wires. By J. J. B. Brownson, M.D. Reprinted from the Tri-State Medical Journal and Practitioner, April, 1898.